Systemic Acquired Resistance

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SYSTEMIC ACQUIRED RESISTANCE

Diseases of plants often limit crop quality, quantity, and choice. We have been investigating manipulation of systemic acquired resistance (SAR)—one of a plant's natural defense systems—to relieve these problems. This natural phenomenon of resistance development in response to prior pathogen infection, initially called plant immunity, was recognized in the early 1900s. In 1933, Chester reviewed research on "acquired physiological immunity in plants" and suggested its general importance for "the preservation of plants in nature" (Chester, 1933). However, the first systematic study of SAR was published by Ross (1961b), who infected xanthi-nc tobacco (*Nicotiana tabacum* L.) (local lesion host) with tobacco mosaic virus (TMV) and found that subsequent infections were reduced in severity. He termed the resistance that developed in the distal, untreated portions of TMV-inoculated plants "systemic acquired resistance" (Ross, 1961b). Resistance in the same leaf that had been TMV-inoculated was called "local acquired resistance" (LAR) (Ross, 1961a). Treatments, such as wounding or applying phytohormones, had no significant effect on the development of SAR (Uknes et al., 1993a; unpublished data). Others, including Kuc (1982), extended work on SAR to nonviral pathogens and the field (for review, see Kessmann et al., 1994). In addition, several synthetic chemicals, such as 2,6-dichlorisonicotinic acid (INA), can activate the same spectrum of resistance in plants while exhibiting no direct antimicrobial activity (Kessmann et al., 1994).

SAR appears to be a ubiquitous phenomenon of higher plants and has been demonstrated, for example, in soybean (*Glycine max* L.), potato (*Solanum tuberosum* L.), tomato (*Lycopersicon esculentum* Mill.), pearl millet (*Pennisetum glaucum* L.), alfalfa (*Medicago sativa* L.), cucurbits (*Cucurbita* spp.), green bean (*Phaseolus vulgaris* L.), *Arabidopsis thaliana*, rice (*Oryza sativa* L.), and barley (*Hordeum vulgare* L.), as well as other plants (for review, see Kessmann et al., 1994).

SAR IS CORRELATED WITH SPECIFIC GENE EXPRESSION

Many of the so-called "defense responses" that are induced by pathogen infection (e.g., expression of certain genes, phytoalexin accumulation, cell wall cross-linking) are only induced locally, near the pathogen (Lamb et al., 1989). Therefore, these responses are not associated with the resistant state found in the noninoculated portion of the infected plant. Van Loon and Antoniw (1982) demonstrated that a subset of the pathogenesis-related proteins (PR proteins), the acidic-extracellular forms in tobacco, accumulate during the onset of resistance. More recently, steady-state mRNA levels from at least nine families of genes were coordinately induced in uninfected, resistant leaves of inoculated plants; these gene families are now known as "SAR (systemic acquired resistance) genes" (Ward et al., 1991). Several of the SAR genes encode proteins that are similar to proteins, with direct antimicrobial activity. These activities include β -1,3-glucanases, chitinases, and cysteine-rich proteins related to thaumatin (for review, see Kessmann et al., 1994). Direct evidence for SAR gene involvement in resistance comes from experiments showing that constitutive high-level expression of tobacco PR-1a in transgenic tobacco (*Nicotiana tabacum* cv. Xanthi nc) results in increased tolerance to two oomycete pathogens, *Peronospora tabacina* Adam (causal agent of blue mold disease) and *Phytophthora parasitica* (black shank

disease) (Alexander et al., 1993). These two pathogens are effectively controlled by SAR, and PR-1 is the most abundant protein associated with the resistant state.

Support for the involvement of SAR gene homologs in resistance comes from experiments with transgenic tobacco and *Brassica* seedlings that express a chitinase from bean; these plants are significantly protected against damping-off caused by *Rhizoctonia* (Broglie et al., 1991). We have observed similar resistance to *Rhizoctonia* in tobacco transformed with acidic and basic chitinases from tobacco and cucumber (*Cucumis sativus* L.) (Uknes et al., unpublished data).

Although several SAR genes apparently are common among plants, each species seems to have evolved a distinct subset of SAR genes. For example, PR-1 is the most abundant SAR gene in tobacco, but an acidic class III chitinase is most abundant in cucumber (Métraux et al., 1988; Ward et al., 1991). Although not proven, such differences may reflect differences in evolutionary or breeding constraints that have selected for the most effective SAR response to the particular pathogen spectrum to which the host is subject. Therefore, plants apparently have developed an inducible pyramid of defenses, a likely evolutionarily stable form of resistance.

Microscopic studies have shown that plants exhibiting SAR appear to mimic the type of resistance observed with dominant genetic resistance. This response is demonstrated as increased deposition of papilla-like material and a hypersensitive-like response, indicated by rapid plant cell death at the attempted site of penetration (Dean and Kuc, 1988; Stein et al., 1993; Uknes et al., 1992, 1993b).

SALICYLIC ACID AS A SIGNAL FOR SAR

In 1979, White showed that the exogenous application of salicylic acid (SA) and certain other benzoic acid derivatives could induce resistance to TMV and the accumulation of PR-proteins (White, 1979). Recently, the involvement of SA in SAR signaling has been clearly demonstrated. Transgenic tobacco that expresses the salicylate hydroxylase gene (*nahG*) from *Pseudomonas putida* (Trevisan) does not accumulate SA in response to pathogen infection, and it shows no SAR gene or resistance expression in the distal portions of the plant (Gaffney et al., 1993). Salicylate hydroxylase catalyzes the conversion of SA to catechol, which has no SAR-inducing activity. Therefore, SA appears to be required for SAR development. More recently, Vernooij et al. (1994) performed grafting experiments using wild-type and salicylate-hydroxylase-expressing plants to test whether SA was a systemic signal. NahG transgenic rootstocks, when inoculated with TMV, could induce SAR in wild-type scion tissue equally as well as wild-type rootstocks inoculated with TMV. In addition, scions that expressed salicylate hydroxylase were unable to become resistant, regardless of the rootstock to which they were grafted. The simplest conclusion is that SA is not the primary long-distance signal required for SAR, but SA must be present for the signal to be transduced into gene expression and resistance.

To determine the general role that SA plays in plant health, we assayed bacterial and fungal disease progression in NahG and control tobacco and *Arabidopsis thaliana* L. (Delaney et al., 1994). *nahG* tobacco plants showed more severe disease symptoms than controls when inoculated with TMV, *Pseudomonas syringae* pv. *tabaci* (Wolf and Forster), *Cercospora nicotianae* (Ell. and Ev.), and *Phytophthora parasitica* (Dastur). NahG *Arabidopsis* showed greater disease symptoms compared to the control when inoculated with *Pseudomonas syringae* pv. *tomato* (DC 3000) (Okabe) and *Peronospora parasitica* (Pers.:Fr.). Surprisingly, disease symptoms and pathogen growth occurred on NahG *Arabidopsis* even when avirulent bacteria or races of the fungus to which *Arabidopsis* is normally resistant were used.

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These results underscore the importance that SA and SA-dependent responses play in plant health.

GENETIC MODIFICATION OF SAR CAN LEAD TO RESISTANT PLANTS

To genetically dissect the signal transduction mechanism leading to SAR, we isolated mutants of *Arabidopsis* with altered SAR gene expression (Lawton et al., 1993; Uknes et al., 1993c). Plants that cannot induce SAR gene expression and resistance in response to pathogens or chemical treatment are called “nims” (no immunity). Plants that have high constitutive SAR gene expression and resistance are called “cims” (constitutive immunity). *cim Arabidopsis* fall into two categories: plants with lesions that simulate disease (*Isd*) (Dietrich et al., 1994) and plants with no obvious macroscopic phenotype. *Isd* mutants are similar to other so-called lesion mimics of important crop species such as maize (*Zea mays* L.) and wheat (*Triticum aestivum* L.) (Walbot et al., 1983). Such mutants may help us understand the processes involved in disease resistance.

CONCLUSION

SAR appears to be a ubiquitous higher-plant defense response. SAR is tightly correlated with the expression of specific genes that, when expressed constitutively via strong promoters or genetic alteration (*cim*), confer resistance to disease. Furthermore, when SAR is eliminated, due to the obliteration of an essential signal (SA), generalized severe disease susceptibility occurs. Therefore, SAR appears to be an essential component of the defenses that ensure plant health in nature.

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