Genetics of Plant-Pathogen Interactions (Plant Immunity)

Topics on
Systemic Acquired Resistance (SAR)
Host vs. Nonhost
Basic fact: In nature, most plants are healthy most of the time. If disease occurs, it is actually restricted to a few plants and affects only small amount of tissue.

*Most plants are resistant to most pathogens, so disease is the exception.*

Plants that cannot be successfully attacked by a given pathogen are called **nonhosts** for that pathogen.
Major types of plant pathogens:

- Bacteria
- Fungi (>80% loss)
- Viruses
- Viroid

**Pathogenesis**: Process of infection, colonization, and reproduction

**Pathogenecity**: Ability to cause disease

**Virulence**: Severity of disease

**Plants’ defense systems**:
- Preformed (nonhost)
- Activated or induced
Disease symptoms

Signs of disease
Botrytis cinerea, the gray mold fungus, sporulating on grapes.

noble rot
sweet dessert wines

Sauternes
Château d'Yquem
From J. Kronstad

Corn smut

Huitlacoche in Mexico (Mexican truffle)

From J. Kronstad
Activated plant defense systems

- Local responses
- Systemic responses (SAR)
Local responses:

hypersensitive reaction (HR)

HR is a pattern of localized cell death within the plant tissue at the site of infection.

This local cell death blocks pathogen growth by depriving biotrophic pathogens of nutrition, and by creating a highly oxidizing environment that damages proteins and cell structures.
Systemic acquired resistance (SAR)

Local HR

Systemic resistance

Necrosis

??

??
Systemic acquired resistance (SAR)

Later

No previous virus infection

Early in growth season

Blue mold of tobacco
Immediate responses of invaded cells
- Generation of reactive oxygen species
- Nitric oxide synthesis
- Opening of ion channels
- Protein phosphorylation/dephosphorylation
- Cytoskeletal rearrangements
- Hypersensitive cell death (HR)
- Gene induction

Local responses and gene activation
- Alterations in secondary metabolic pathways
- Cessation of cell cycle
- Synthesis of pathogenesis-related (PR) proteins
- Accumulation of benzoic and salicylic acid
- Production of ethylene and jasmonic acid
- Fortification of cell walls (lignin, PGIPs, HRGPs)

Systemic responses and gene activation
- (1→3)β-Glucanases
- Chitinases
- Peroxidases
- Synthesis of other PR proteins
Detailed analysis of TMV necrotic lesions forming on N gene—expressing resistant tobacco leaves reveals that total salicylic acid (SA) contents are greatest in the necrotic lesion center and rapidly diminish with distance from the center.
Is SA a signal molecule for SAR?

How would you test it?
Is SA a signal molecule for SAR?

Test for sufficiency: Is SA sufficient to induce SAR?

Test for necessity: Is SA required for SAR?
Effects of 200 μM salicylic acid (SA) treatments and inoculation of *Fusarium oxysporum* f. sp. *lycopersici* (*Fol*) on hydroponically grown tomato. (a) Vascular browning; (b) Leaf yellowing wilting.
NahG: salicylate hydroxylase from *Pseudomonas fluorescens*
Will SA save the day?
Chemical structures of three systemic acquired resistance (SAR)—inducing compounds.

Salicylic acid (SA)

Dichloroisonicotinic acid (INA)

Benzo-(1,2,3)-thiodiazole-7-carbothionic acid S-methylester (BTH)

Actiguard™

Ryals’ group, Novartis
Systemic acquired resistance (SAR)

Local infection

Systemic resistance → SA↑

Necrosis → SA↑

? → Systemic resistance
Immediate responses of invaded cells
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Major PR proteins

PR-1: cysteine-rich proteins, with antifungal activity

PR-2 (β-glucanses, BGL2): have (1,3)-endoglucanase activity in vitro

PR-3 (chitinases)

PR-4 (chitin-binding) proteins: have potent antifungal activity

PR-5 (TL): thaumatin like proteins, sweet tasting, stable structure, resistant to protease degradation, may affect membrane permeability
Can we enhance general disease resistance to pathogens by overexpressing PR genes?
What is the mobile signal for SAR? Is SA the mobile signal?

Local infection

Systemic resistance

PR gene expression

Necrosis

SA

?
Is SA the mobile signal for SAR?

Control grafts

Xanthi/Xanthi

nahG/nahG

Reciprocal grafts

nahG/Xanthi

Xanthi/nahG

CMV

TMV
References:

Summary on what we learned about SAR

1. SAR is a pathogen-induced defense response against broad-spectrum of pathogens.

2. PR genes are highly induced during SAR. They are used as molecular markers for defense responses.

3. SA level increases locally and systemically during SAR.

4. SA is required for SAR.

5. SA and its analogs are sufficient to induce SAR.

6. SA is most likely not the mobile signal for SAR.

7. Overexpression of a single PR gene is not sufficient to establish SAR.
Local infection

Systemic resistance

PR gene expression

Necrosis

SA

??
Proposing experiments to address questions on SAR

Hint: For each step in a signaling pathway, there are (almost always) not only positive regulators, but also negative regulators. Think about what kinds of phenotypes you would expect for l-o-f mutants of those regulators before you design a genetic screen.
# Proposed experiments to study SAR

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<tr>
<th>Question or hypothesis</th>
<th>Proposed experiments</th>
<th>Expectation</th>
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<tbody>
<tr>
<td>1. Arabidopsis can be used to study SAR</td>
<td>Test SAR response in Arabidopsis</td>
<td>Yes, SAR on Arabidopsis</td>
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<td>2. What is leading to lesion formation?</td>
<td>Mutant screen: surviving HR constitutive HR</td>
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<td>3. What are contributing to SA synthesis?</td>
<td>Measure Sa to look for Sa deficient mutants</td>
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<td>4. What are the + regulators of SAR?</td>
<td>Screen for SAR deficient mutants</td>
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<td>5. What are the - regulators of SAR?</td>
<td>Screen for high SA mutants</td>
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<td>6. What + regulator is downstream of SA?</td>
<td>Spray Sa, and look for susceptible mutants</td>
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