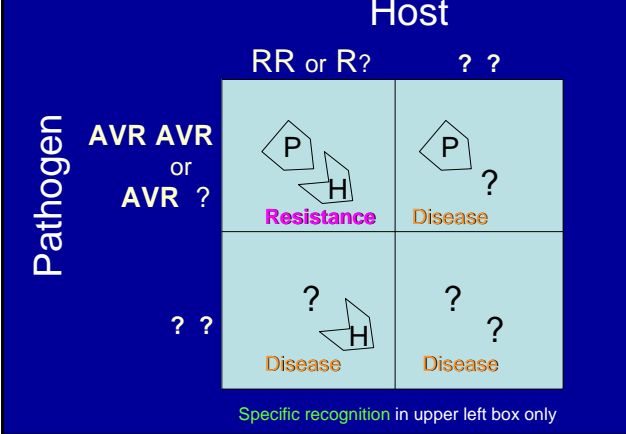


Gene-for-Gene Hypothesis



How many races?

Three cultivars, each with a different R gene yields 8 races (2^3)

Ten cultivars, each with a different R gene could discern a possible 1024 races (2^{10}) !!

The set of cultivars used to characterize races is called a 'differential set'

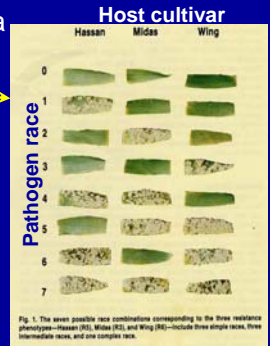


Fig. 1. The seven possible race combinations corresponding to the three resistance phenotypes—Hassan (H), Mides (M), and Wing (W)—include three simple races, three intermediate races, and one complex race.

A common test question: 😊

For each of the boxes, indicate whether or not the races of a rust pathogen will succeed in infecting the host cultivars

Avirulence (A) or lack-of-avirulence (? or 'a') genes possessed

Resistance (R) or lack-of-resistance (? or 'r') genes in each cultivar	Race: $A_1A_2A_3A_4$	$A_1a_2A_3A_4$	$a_1A_2A_3A_4$	$a_1a_2A_3A_4$	$a_1a_2a_3a_4$
	$r_1r_2f_3f_4$				
	$R_1r_2f_3f_4$				
	$r_1R_2f_3f_4$				
	$R_1R_2f_3f_4$				
	$R_1R_2f_3R_4$				

'R', 'A' = active product present '?' , 'a', 'r' = active product absent

A common test question: 😊

For each of the boxes, indicate whether or not the races of a rust pathogen will succeed in infecting the host cultivars

Avirulence (A) or lack-of-avirulence (? or 'a') genes possessed

Resistance (R) or lack-of-resistance (? or 'r') genes in each cultivar	Race: $A_1A_2A_3A_4$	$A_1a_2A_3A_4$	$a_1A_2A_3A_4$	$a_1a_2A_3A_4$	$a_1a_2a_3a_4$
	$r_1r_2f_3f_4$	yes	yes	yes	yes
	$R_1r_2f_3f_4$	no	no	yes	yes
	$r_1R_2f_3f_4$	no	yes	no	yes
	$R_1R_2f_3f_4$	no	no	no	yes
	$R_1R_2f_3R_4$	no	no	no	no

'R', 'A' = active product present '?' , 'a', 'r' = active product absent

Where do we see R-gene resistance used?

Obligate biotrophs

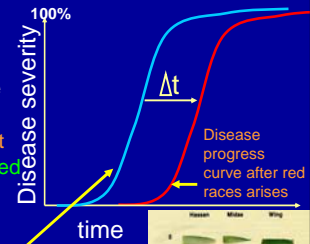
rusts (cereals, pine, flax, coffee, corn)
 smuts (cereals, corn)
 eastern filbert blight (Oregon hazelnuts)
 downy mildews (sunflower, lettuce, crucifers)
 powdery mildews (cereals, cucurbits)
 viruses (many crops)
 nematodes: sedentary endoparasites (tomato, cereals)

Facultative saprophytes

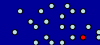
apple scab
 bacterial leaf spots (legumes, cotton, arabidopsis)
 ascomycete leaf spots (rice, tomato, bean)
 vascular wilts (crucifers, tomato, banana)
 oomycetes - root rot (soybean), late blight (potato)

Host resistance & Disease dynamics

Qualitative, race-specific resistance
 Reduces initial inoculum because only a subset of pathogen genotypes (in the beginning, most if not all) are specifically recognized

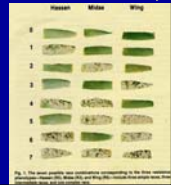


Pathogen population



After introduction of the R-gene, the 'Blue' race is recognized but red race is not. Frequently, the red race arises later via mutation

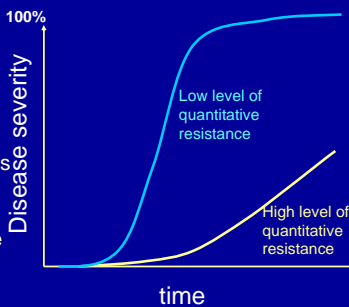
Disease progress curve prior to Introduction of blue race R-gene



Host resistance & Disease dynamics

Quantitative, non-race specific resistance

Reduces the infection rate
 Slows how rapidly disease develops
 Is effective against all isolates of the pathogen
 Generally, it lasts a long time



Major gene (R-gene) resistance:

Upside: simply inherited and extremely effective 😊

Downside: ☹️
 it breaks down when pathogen loses its *elicitor* (i.e., stops making the AVR gene product)

Rice blast



Facultative saprophyte
 Polycyclic disease cycle
 High mutation rate in genome

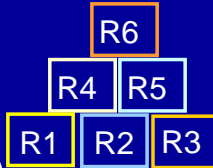


Release and breakdown of varietal resistance to blast in Korea
 = period of time R-gene resistance was effective

Smart deployment of Race specific resistance:

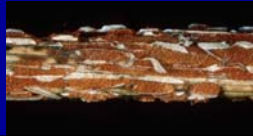
R-Gene pyramids

(i.e., use of multiple, effective R genes simultaneously)



Theory: If a mutation from AVR1 (recognized) to avr1 (i.e., no *elicitor*) occurs once every 10^8 to 10^{10} asexual spores, then a simultaneous mutation from AVR₁, AVR₂, ..., AVR₆ to avr₁, avr₂, ..., avr₆ will be a very, very rare event.

R-Gene pyramids have been highly effective for control of cereal rusts



Smart deployment of race specific resistance:

What is this man doing?



Hints:

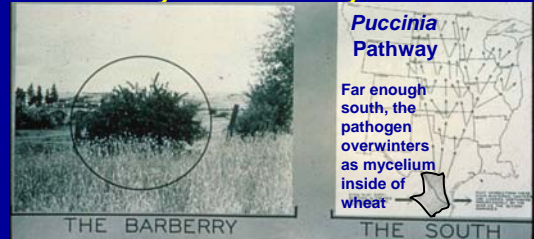
- He works for the government
- The white stuff is NaCl
- The bush is *Berberis vulgaris*



Between 1918 and 1975, under the direction of the federal government, more than 100 million bushes of *Berberis*, *Mahonia* and *Mahoberis* spp. were killed in 16 Northern wheat-growing states. The program was stopped in 1975.

Recently (2003), common barberry has begun a resurgence.

Stem rust of wheat can turn its primary cycle in two ways:



Why was/is eradication of barberry so important?

Elimination of meiosis greatly(!) slows genesis of new races

Take home message:

Everything else being equal, asexual pathogens are better candidates for suppression by R-genes than those frequently recombining their genomes

R-Gene Deployment - Cultivar Mixtures

Mechanisms of suppression:
 Dilution of inoculum
 Resistant plants are dispersal barriers
 Avirulent spores induce SAR

Works best with polycyclic, foliar diseases of 'skinny' plants (rusts or powdery mildew of cereals)

Lecture: Considerations in breeding for and managing host resistance

Avoiding loss of R-gene resistance

- pathogen's life cycle
polycyclic vs monocyclic ☹️ 😊
- pathogen's genetics
meiosis, high mutation rates ☹️ 😊
- Longevity of host plant
↓ 😊 ↑ ☹️
- Ease of deploying multiple R-genes
gene pyramids 😊
cultivar mixtures 😊

Maintaining effective minor gene resistance

- host exposed to maximum pathogen diversity? 😊
- yield/quality evaluated and maintained under conditions most favorable for disease? 😊
- continued diligent, field-based screening and marker-assisted selection during breeding efforts 😊

Breeding for Minor gene (polygenic) resistance

It is important to expose the potential cultivar to the extremes of pathogen diversity for a significant period of time

Oat crown rust nursery

Oat lines are grown in a 'pathogen brothel'. Those **completely** resistant or extremely susceptible are thrown away. Those that yield well with a bit of disease stay with the program. 😊

