Multiple Disease Resistance in Maize

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Introduction

Different pathogenic fungi use different strategies to cause disease on maize. Some pathogens, ***necrotrophs***, derive their nutrition from dead cells, while others, ***biotrophs***, feed on living cells. Also within these two broad classes there are differences in the ways the pathogens are dispersed and enter and grow within the leaf . Having said this, there are often also many similarities in pathogenesis strategies between plant pathogenic fungi. For instance, in **Figure 1** below, the fungi causing southern leaf blight (SLB) and gray leaf spot (GLS) both enter the leaf primarily through the stomata, grow between host cells outside the vascular bundle and eventually kill them.

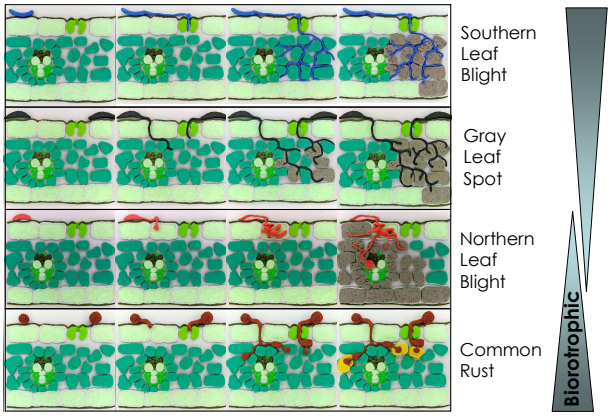


Figure 1. Claymations showing the progression of leaf infection of four maize foliar fungal diseases

If different pathogens share aspects of their pathogenesis strategies, it seems likely that host resistance genes that target these shared aspects may confer ***multiple disease resistance (MDR)***. We have looked for evidence for multiple disease resistance in maize in several ways.

Genetic correlations in the association mapping population provides evidence for genes conditioning MDR

The maize association mapping population consists of 302 lines comprising a great deal of maize genetic diversity (Flint-Garcia et al Plant J. 44 :1054-1064). Linkage disequilibrium within this population is generally very low. Therefore, significant genetic correlations among different traits in this population would suggest that either the same genes or very closely linked genes underlie the co-varying trait variation.

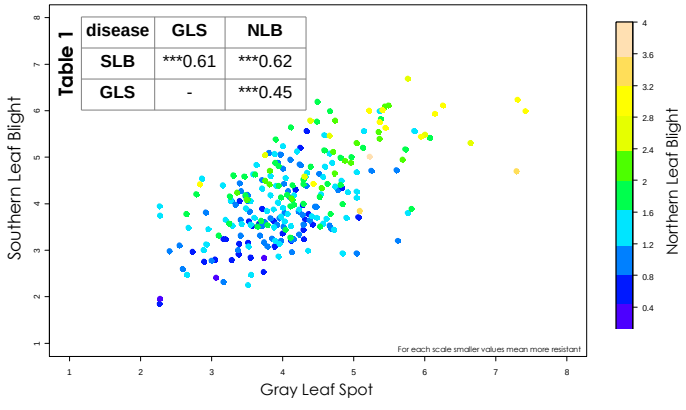
In replicated trials over several environments, we assessed the resistance of 274 of the lines within this population to three foliar diseases of maize: SLB (5 environments), GLS (3 environments), and NLB (3 environments). Breeding values for area under disease progress curve (AUDPC) were calculated for each disease for each line. Breeding values were derived using a model that accounted for controllable experimental effects, flowering time (a significant confounding factor of disease resistance), population structure, and kinship.

Figure 2 shows the breeding values (of each line) for AUDPC of SLB plotted against GLS. For NLB, the breeding values are indicated on a color scale. The appearance of a strong relationship between resistance to these three diseases is quantified in **Table 1**, which reports the (genetic) correlations of the breeding values for each disease.

Figure 2

Table 1

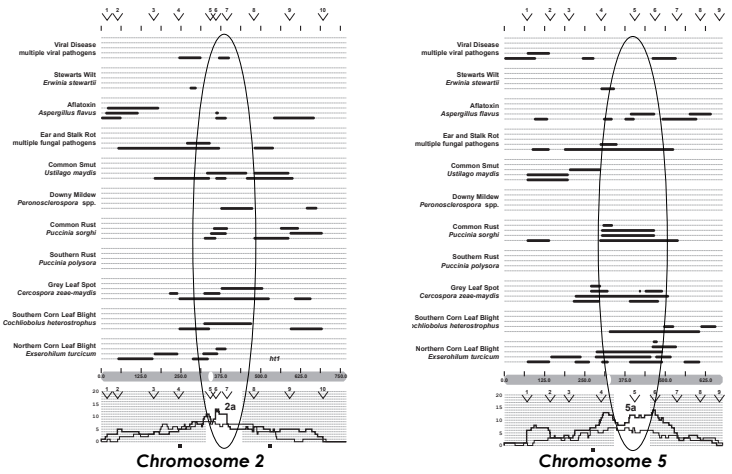
disease	GLS	NLB
SLB	***0.61	***0.62
GLS	-	***0.45



Literature Survey

We synthesized disease resistance QTL reported in the literature (Wisser et al. 2006 Phytopathology 96:120-129) and found statistical evidence that QTL were non-randomly distributed with several genomic regions carrying QTL for resistance to several different diseases. In **Figure 3**, all reported disease resistance QTL on chromosomes 2 and 5 are represented and genomic regions associated with abundant QTL for resistance to multiple diseases are circled.

Figure 3



Chromosome 2 *Chromosome 5*

Mapping multiple disease resistances in the IBM population

We have evaluated the high resolution IBM mapping population (Lee et al.2002. Plant Mol. Biol. 48:453-461) over several environments in replicated field trials for resistance to SLB, GLS, and NLB. Genetic correlations between AUDPCs are shown in **Table 2.** The correlations are highly significant though modest. **Table 3** shows the bin locations and chromosomal regions on the IBM2 map where QTL for SLB, GLS and NLB resistance have been localized. Co-localizing QTL are highlighted in yellow. **Figure 4** shows examples of co-localizing and non-co-localizing QTL: **(A)** A major SLB resistance QTL (bin 1.10) has no co-localizing QTL for resistance to the other diseases; **(B)** In bin 2.04 SLB and GLS resistance QTL co-localized, and **(C)** in bin 2.07 an NLB QTL co-localized with a GLS effect (i.e. a putative QTL that did not surpass the significance threshold); **(D)** Resistance effects for all three diseases co-localized on chromosome 4, though the SLB effect was non-significant.

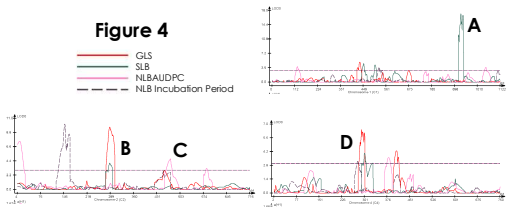
Table 2

disease	GLS	NLB
SLB	***0.42	**0.16
GLS	-	***0.26

Table 3

Bin	GLS	SLB	NLB
1.03		237-257	
1.05	412-417	400-411	
1.06		568-583	
1.06		601-607	
1.1		928-941	
2.00-2.01			0-22
2.02			148-153
2.04	284-294	285-303	
2.06-2.08			391-522
3.04		164-166	
3.04		217-258	
4.02/03		123-137	
4.05	288-292		277-283
4.08			449-457
6.02		124-128	
6.05			299-326
6.07			481-503
7.03		340-356	
8.02/03		165-175	
8.05			375-379
8.07			464-501
9.03	243-249		
9.05	349-362		

Figure 4



Conclusions

1. The highly significant genetic correlations between resistances to three different fungal foliar diseases detected in the association and IBM mapping populations strongly suggest that **functional variation in genes for multiple disease resistance exists in maize.**
2. Breeding value correlations estimated from the association population data also suggest that **selection for improved MDR to SLB, GLS and NLB is attainable.**
3. Our QTL mapping results from the IBM populations (and other populations [data not shown]) further suggest that **MDR QTL tend to be of low to moderate effect and larger effect QTL tend to be disease-specific.**

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