The Analysis of Quantitative Resistance to Foliar Diseases of Maize

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Introduction

We are interested in the analysis of quantitative resistance - the dominant form of resistance utilized in cultivated maize. Very little is known about the molecular genetic or physiological basis of this type of resistance in maize or any other crop. Working with the foliar diseases gray leaf spot (GLS), southern leaf blight (SLB) and northern leaf blight (NBL), we are identifying and mapping new sources of resistance from diverse germplasm.

Use of a Disease Lesion Mimic Gene to Identify Components of the Defense Response

Rp1-D21 is an obovate major disease resistance gene, which triggers the hypersensitive resistance response (HR) in the absence of pathogen recognition, leading to constitutive necrotic spotting, called a disease lesion mimic phenotype. We have shown that the strength and quality of the phenotype conferred by Rp1-D21 is variable, depending on the genetic background. Table 1 below shows the Diversity of the Rp1-D21-mediated HR in maize. In this case the mutant tester - H95 heterozygous for Rp1-D21(called H95:Rp1-D21) - was crossed to each of the lines indicated. The height ratio is the average height of the mutant F1 segregants divided by the average height of the wild type F1 segregants

<table>
<thead>
<tr>
<th>Cross</th>
<th>Height ratio (M/WT)</th>
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<tbody>
<tr>
<td>H95:Rp1-D21 x B73</td>
<td>0.71</td>
</tr>
<tr>
<td>H95:Rp1-D21 x NC330</td>
<td>0.67</td>
</tr>
<tr>
<td>H95:Rp1-D21 x NC338</td>
<td>0.67</td>
</tr>
<tr>
<td>H95:Rp1-D21 x DH4</td>
<td>0.75</td>
</tr>
<tr>
<td>H95:Rp1-D21 x P37</td>
<td>0.79</td>
</tr>
<tr>
<td>H95:Rp1-D21 x TX303</td>
<td>0.84</td>
</tr>
<tr>
<td>H95:Rp1-D21 x Zb1</td>
<td>0.81</td>
</tr>
<tr>
<td>H95:Rp1-D21 x Msd2W</td>
<td>0.92</td>
</tr>
</tbody>
</table>

We are now using this system to investigate the genetic architecture of the defense response induced by Rp1. Since the Rp1-D21 phenotype is much stronger in an H95/Mo17 background than in an H95/B73 background, we were able to use the Mo17 x B73 -derived IBM population to map genomic regions that are responsible for this differential. Each of the IBM lines was crossed to H95:Rp1-D21. The resulting F1 seed segregated 1:1 for the presence of Rp1-D21 (since H95:Rp1-D21 is heterozygous for the gene). Each F1 population was scored in the field for several traits: for lesion severity on a 1-10 scale, average difference in height between wt and mt segregates and the average difference in flowering time between wt and mt segregants. The population was scored in replicated field trials in North Carolina and Indiana and in the greenhouse. The same major QTL mapping on chromosome 10 and designated Hml1, for HR modulating locus 1, was identified in each environment for each of these traits.

Using Image Analysis to Accurately Score Disease

One of the main challenges in the analysis of quantitative disease resistance is simply how to score the resistance trait accurately and efficiently. With a foliar blight disease, the basic trait scored is necrotic leaf area but it is challenging to accurately assess this quickly. We use a semi-qualitative scale (Fig. 6) which makes for relatively rapid scoring but is likely non-linear. We are investigating image analysis to more accurately score disease phenotypes (Fig. 7). A segregating population derived from a B73 x De811 cross was assessed using the normal scoring system and using image analysis. Comparison of QTL mapping analyses based on these two data sets indicate that different sets of QTL are identified (Fig. 8).

Dissection of Components of Quantitative Disease Resistance

We have mapped resistance QTL for SLB, GLS and NLB in several populations, including the IBM and NAM populations (see papers 215, 216, 220 and refs 1-5). In general we use a trait known as “Area under disease progress curve” - AUDPC as a measure of disease resistance. This is a measure of disease over time.

In general, there are three ways to decrease AUDPC (and increase resistance): 1. Initial resistance can increase but rate of disease progression remains same (Fig. 4A). 2. Initial resistance stays the same but rate of progression decreases (Fig. 4B). Some mixture of the above. With this in mind we reanalyzed our SLB data collected on the IBM population, using slope (~disease progression) as a trait. Figure 5 shows the three major QTL for SLB AUDPC on chromosomes 1 (Fig. 5A), 3 (Fig. SB) and 8 (Fig. SC). The chm 1 SLB QTL colocalizes with the main QTL for SLB slope while there is no effect on slope at the other two SLB AUDPC QTL. From this it can be inferred that the chm1 and chm3 SLB AUDPC QTL increase the initial level of resistance while the chm1 SLB AUDPC QTL may function by decreasing the disease progression rate.


Footnote: USDA-AMS, CGMIP Challenge program, USDA-AMS.