



# Analysis of Quantitative Resistance to Foliar Diseases of Maize and Evidence for Multiple Disease Resistance Loci.

P. Balint-Kurti<sup>1</sup>, G. Johal<sup>2</sup>, J. Kolkman<sup>3</sup>, R. Nelson<sup>3</sup>, R. Wisser<sup>1</sup>, J. Zwonitzer<sup>1</sup> and D. Rhyne<sup>1</sup>

<sup>1</sup>USDA-ARS, Dept of Plant Pathology, North Carolina State University, Raleigh, NC 27695-7616. <sup>2</sup>Dept. of Botany and Plant Pathology, Purdue University, West Lafayette, IN. <sup>3</sup>Dept. Of Plant Pathology, Cornell University, Ithaca, NY.

## Introduction

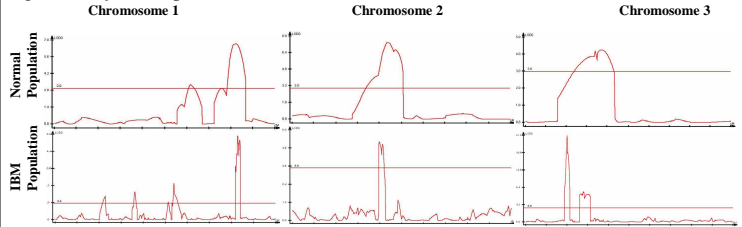
Most disease resistance used in maize is *quantitative* in nature, i.e. incomplete but sufficient to protect yield. Quantitative resistance is generally extremely durable, but *very little is known about its molecular or physiological basis*.

We are characterizing genomic regions conferring quantitative resistance (also known as quantitative trait loci or QTL) to three fungal diseases of maize with significant importance in North Carolina – **gray leaf spot (GLS)**, **southern leaf blight (SLB)** and **northern leaf blight (NLB)**.



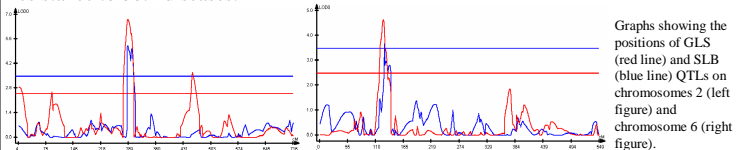
## Fine Mapping of Resistance QTL

Using a maize advanced intercross RIL population derived from a B73 by Mo17 cross (the IBM population), we precisely defined SLB QTL on chromosomes 1, 2 and 3, often to within 1-2 cM. We also used a 'normal' B73/Mo17 RIL population to map the same QTL. As expected, the same QTL were identified but with significantly lower precision (Balint-Kurti et al 2007).



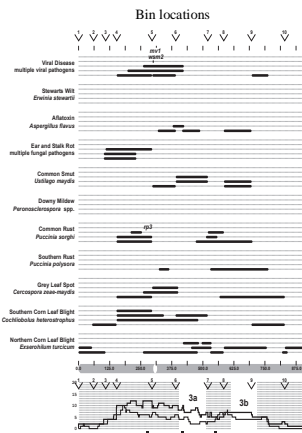
## Identification of QTL for Multiple Disease Resistance

By also mapping GLS QTL using the IBM population, we were able to precisely define at least two genomic regions, on chromosomes 2 and 6 conferring resistance to both diseases.



## A Summary of Maize Disease Resistance Studies

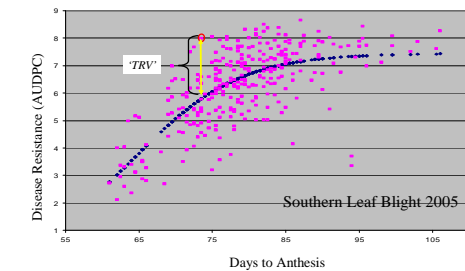
Fifty publications reporting the locations of 437 QTL for disease resistance (or dQTL) in maize were summarized (Wisser et al.). Statistical tests indicated the **presence of clusters of dQTL for multiple diseases**. Evidence was also found for the **association of dQTL with maturity-related QTL**. Bins 3.04 and 6.01 are hotspots for both SLB and viral QTL. Bin 1.05 is a hotspot for GLS resistance QTL.



Consensus map of resistance loci in maize. Chromosome 3 is shown here for illustration purposes. At the bottom of the diagram is a histogram summarizing the QTL frequency per centiMorgan. The thicker line shows the frequency of dQTL and the thinner line maturity QTL. dQTL hotspots (taking into account gene density) are indicated as white areas in the histogram. On chromosome 3 there are two such hotspots, 3a and 3b.

## Correlations Between Resistance to Different Diseases in a Population of Diverse Lines

We screened the diverse 302-line association mapping population for SLB, GLS and NLB resistance over several years. Disease resistance is strongly correlated with maturity for all diseases. We corrected for maturity to get the 'True Resistance Value' (TRV) for each line.



Disease resistance (measured on a 1-9 scale with 9 being completely resistant and 1 being dead), plotted against time to pollen shed for 302 diverse lines making up the maize association-mapping population. 'True resistance value' measurements were made by determining the distance of each point from the line of logistical regression.

**Significant pairwise correlations between the TRV values for SLB, GLS and NLB were found** (see values highlighted in blue below). This suggests that there may be a *significant number of QTL with pleiotropic effects, i.e. conferring resistance to more than one disease*.

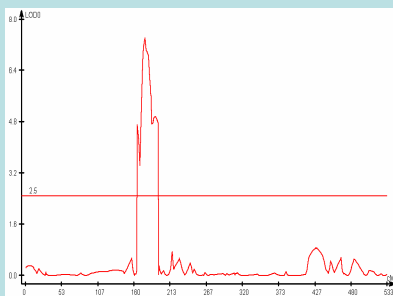
	SLB2004	SLB2005	SLBT/2006	SLB2006	GLS2004	GLS2005	GLS2006	NLB2006
SLB 2003	0.72799 +0.001 287	0.73181 +0.001 288	0.70602 +0.001 276	0.78116 +0.001 285	0.19669 0.0014 281	0.34456 +0.001 289	0.32246 +0.001 273	0.38589 +0.001 240
SLB2004		0.77347 +0.001 294	0.68165 +0.001 291	0.80236 +0.001 290	0.22052 0.0003 288	0.32243 +0.001 277	0.33104 +0.001 276	0.36154 +0.001 246
SLB2005			0.71202 +0.001 290	0.86049 +0.001 299	0.30116 +0.001 288	0.42417 +0.001 277	0.33026 +0.001 284	0.35109 +0.001 249
SLBT/2006				0.72884 +0.001 290	0.15974 0.0106 284	0.29527 +0.001 286	0.32295 +0.001 275	0.34084 +0.001 238
SLB2006					0.30974 +0.001 284	0.41363 +0.001 275	0.34063 +0.001 283	0.36808 +0.001 247
GLS2004						0.60932 +0.001 266	0.55695 +0.001 263	0.20616 +0.001 225
GLS2005							0.58512 +0.001 265	0.1954 0.0027 234
GLS2006								0.27399 +0.001 236

Table showing the correlation between TRV scores in the association mapping population for SLB ratings in five different environments, GLS ratings in three and NLB rating in one. The top number is the Pearson correlation coefficient, the middle number is the P-value associated with the coefficient and the lower number is the number of informative data points. Values highlighted in blue indicate 'between-disease' correlations with a P-value of <0.0001. Values highlighted in yellow are 'within-disease' correlations.

## Use of a Disease Lesion Mimic Gene to Identify Components of the Defense Response: Rp1-D21 is an aberrant major disease resistance gene, which triggers the defense response in the absence of pathogen recognition, leading to constitutive necrotic spotting, called a disease lesion mimic phenotype. We have shown that the strength of the phenotype conferred by Rp1-D21 is variable, depending on the genetic background. The Rp1-D21 phenotype in several different genetic backgrounds in seven week old plants is shown below:



We are mapping regions responsible for this phenotypic variation, which likely carry genes for important components of the defense response. The figure to the right shows a locus on chromosome 10 which controls variation in this response in a B73/Mo17 RIL population



## Acknowledgements

Primary funding for this work is from the USDA Agricultural Research Service (ARS). Other funding is provided by the CGIAR Generation Challenge program and the Corn Growers association of North Carolina.



Many thanks to the following: Donna Stephens, Major Goodman, Jim Holland, Steve Szalma, Richard Pratt, Marty Carson, Dan Gorman, Mike Jines, Mike Lee, Dave Marshall, Peg Redinbaugh, Enrico PC, Charlotte Bronson, Christina Cowger, Scott Walker, Dave Bubeck, Erik Stromberg.

## References

- Balint-Kurti, P. J., and M. L. Carson. 2006. Analysis of Quantitative Trait Loci for Resistance to Southern Leaf Blight in Juvenile Maize. *Phytopathology* 96 (3):221-225.
- Balint-Kurti, P. J., J. C. Zwonitzer, R.J. Wisser, M.L. Carson, M. Oropeza-Rosas, J. B. Holland, and S. J. Szalma. 2006. Precise mapping of Quantitative Trait Loci for Resistance to Southern Leaf Blight, caused by *Cochliobolus heterostrophus* race O, and Flowering Time using Advanced Intercross Maize Lines. *Genetics (In Press)*.
- Balint-Kurti, P. J., M. D. Krakowsky, M. P. Jines, L. A. Robertson, T. L. Molnar, M. M. Goodman, and J. B. Holland. 2006. Identification of Quantitative Trait Loci for Resistance to Southern Leaf Blight and Days to Anthesis in a Maize Recombinant Inbred Line Population. *Phytopathology* 96:1067-1071.
- Wisser, R.J., P.J. Balint-Kurti, and R.J. Nelson. 2006. The Genetic Architecture of Disease Resistance in Maize: A Synthesis of Published Studies. *Phytopathology* 96:120-129.