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Commentary

Genomic Organization of Disease and Insect Resistance Genes in Maize

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The development of molecular marker techniques has enhanced our ability to map loci involved in disease and insect resistance in maize (*Zea mays* L.) and other plant species (Prince and Tanksley 1992). Prior to the advent of molecular marker analysis, mapping resistance genes in maize typically required the use of endosperm marker-linked translocations and chromosome arm tester stocks (Burnham 1982; Louie et al. 1991). These specialized stocks provided only partial coverage of the genome and, in many cases, were present in inappropriate genetic backgrounds for mapping of disease and insect resistance traits. Restriction fragment length polymorphism (RFLP) analysis alleviates the need to use cytological and morphological markers to map genes, and, in maize, virtually any combination of parental lines can be used for RFLP analysis (Coe et al. 1988; Hoisington and Coe 1989). Over the past 5 years, there has been a large increase in the number of qualitative genes and quantitative trait loci (QTL) mapped in maize for plant response to pests and pathogens. It is becoming apparent, as more genes for disease response traits are placed on the maize linkage map, that loci for disease and insect resistance are not randomly distributed over the maize genome.

Our laboratory has been developing a high resolution genetic map in the region of the maize dwarf mosaic virus (MDMV) resistance gene, *mdm1*, on the short arm of chromosome 6 (Simcox et al. 1995). We discovered that there were two additional resistance genes that map near *mdm1*: (i) *wsm1*, which confers dominant resistance to a related potyvirus, wheat streak mosaic virus (WSMV) (McMullen and Louie 1991; McMullen et al. 1994); and (ii) a recessive gene, *rhm1*, which confers resistance to the fungal pathogen *Cochliobolus heterostrophus* (Drechs.) Drechs. race O (Zaitlin et al. 1993). A search of the literature was undertaken to determine to what extent genes for disease and insect response traits were linked or "clustered" in the maize genome.

Descriptions of various disease and insect traits are listed in Table 1. Table 2 summarizes the map locations of disease and insect resistance genes and QTL reported in the literature. Map positions are reported in the form of "chromosomal bin" locations taken from the 1995 UMC maize RFLP linkage map, generated by the USDA-ARS Plant Genetics Research Unit and the Plant Science Unit at the University of Missouri. For the bin map, each chromosome is divided into approximately equal segments of about 20 cM. The boundaries of the bins are fixed by "core" markers (Gardiner et al. 1993). Map positions given for QTL correspond to the most significant RFLP marker associated with that trait. All information presented in this article can be accessed through the USDA maize genome database, MaizeDB. Information for accessing MaizeDB can be obtained by contacting the MaizeDB curator, Dr. Mary Polacco, at the University of Missouri (maryp@teosinte.agron.missouri.edu).

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Clustering of disease and insect resistance genes.

All 10 of the maize linkage groups contain either disease/insect resistance genes or QTL (Table 2). The majority of the resistance loci and QTL are located in chromosomal bins containing other disease or insect resistance factors. A chi-square test of independence of gene distribution within bins was performed after pooling adjacent bins to increase the expected class numbers to greater than 1.0 (Snedecor and Cochran 1967). The probability the observed distribution could be obtained by chance was $P < 0.001$. The distribution obtained did not fit a random model. These clusters of resistance genes occurred on each linkage group with the exception of chromosomes 7 and 9. The cM distance between loci within a particular cluster varied. In some instances, such as the gene clusters present in bins 3.04 and 6.01, loci were tightly linked, whereas resistance genes in other clusters were distributed over 20- to 40-cM regions. This variation in the size of the clusters may reflect the actual position of the loci, or be due to differences in experimental procedures used in the different mapping studies.

Do resistance genes cluster to a greater extent than other genes? The distribution of expressed sequences in the maize genome has not been adequately addressed. Certainly clusters of gene families such as the zeins, rDNA genes, and P-450 monoxgenases have been identified. Recently, Khavkin and Coe (1995a, 1995b) have reported that single genes and QTL for morphological traits and growth regulation are clustered in the maize genome.

Table 1. Description of pathogens and pests

Disease/insect	Causal organism	Symptom[a]	Vector	Reference
Maize dwarf mosaic virus	Potyvirus	S, Mo	Many aphid species	Simcox et al. 1995
Wheat streak mosaic virus	Potyvirus	S, Mo	*Eriophyes tulipae* (Keifer) mite	McMullen et al. 1994
Maize mosaic virus	Rhabdovirus	S, Mo	*Peregrinus maidis* (Ashmead) leafhopper	Ming et al. 1995b
Maize streak virus		S	*Cicadulina mbila* Naude leafhopper	Kyetere et al. 1995
Stewart's wilt	*Erwinia stewartii*	F, V, Ne	*Chaetocnema pulicaia* corn flea beetle	Ming et al. 1995a
Carbonum leaf spot	*Cochliobolus carbonum* Nelson	F, M, Ne		Coe et al. 1988
Gray leaf spot	*Cercospora zeae-maydis* Theon and Daniels	F, M, Ne		Bubeck et al. 1993
Southern corn leaf blight	*Cochliobolus heterostrophus* (Drechs.) Drechs.	F, M, Ne		Zaitlin et al. 1993
Northern corn leaf blight	*Setosphaeria turcica* (Luttrell) K. J. Leonard & E. G. Suggs	F, V, Ne		Simcox and Bennetzen 1993 Zaitlin et al. 1992 Bentolila et al. 1991 Hoisington and Coe 1989
Common rust	*Puccinia sorghi* (Schwein.)	F		Coe et al. 1988
Southern rust	*Puccinia polysora* Underw.	F		Coe et al. 1988
Anthracnose stalk rot	*Collectotrichum graminicola* (Ces.) Wils.	N		Jung et al. 1994
Fusarium stalk rot	*Gibberella zeae* (Schwein.) Petch	N		Pè et al. 1993
European corn borer	*Ostrinia nubilalis* Hübner	F, N		Schön et al. 1993
Corn earworm	*Helicoverpa zea* (Boddie)	E		Byrne et al. 1995

[a] E = mature ear, F = foliar, M = mesophyll cell layer, Mo = mosaic symptoms, N = stalk internode invasion, Ne = necrosis, R = root infection, S = systemic infection, V = vascular wilt

Table 2. Chromosomal bin locations of disease and insect resistance genes and quantitative trait loci (QTL)[a]

		Chromosome									
Disease/insect trait	**Locus**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Northern corn leaf blight	*ht1*		2.08[b]								
	ht2								8.06		
	htn1								8.06		
	QTL	1.01/2		3.07/8	4.02/3	5.01/2 5.06		7.03	8.03/4		
DIMBOA	*bx1*				4.02/3						
European corn borer	QTL	1.01 1.07	2.03 2.08/9	3.04/5	4.02/3			7.04			10.04/5
Corn earworm	QTL	1.03								9.01/2	10.06
Gray leaf spot	QTL	1.04	2.04/5		4.02 4.04 4.08				8.05		10.05
Anthracnose stalk rot	QTL				4.08						
Maize streak	*msv1*	1.04									
Stewart's wilt	QTL	1.05									
Carbonum leaf spot	*hm1*	1.07									
	hm2									9.04/5	
Fusarium stalk rot	QTL	1.07	2.04	3.04/5	4.04	5.02 5.04					10.06
Common rust	*rp3*			3.04							
	rp4				4.02/3						
	rp1										10.01
	rp5										10.01
	rp1-G										10.01
Southern rust	*rpp9*										10.01
Maize mosaic	*mv1*			3.04							
Wheat streak mosaic	*wsm1*						6.01				
	wsm2			3.04							
	wsm3										10.05
Maize dwarf mosaic	*mdm1*						6.01				
Southern corn leaf blight	*rhm1*						6.01				

[a] Map positions and scores of restriction fragment length polymorphism loci can be accessed through the maize genome database, MaizeDB. Information concerning access to MaizeDB can be obtained by contacting the MaizeDB curator, Dr. Mary Polacco, at the University of Missouri (maryp@teosinte.agron.missouri.edu).

[b] Bin locations are designated by an X.Y code, where X is the linkage group containing the bin and Y is the location of the bin within the linkage group.

An interesting aspect of these clusters is the spectrum of pathogens/pests and host-pathogen/pest interactions represented (Table 1). For example, near the centromere on chromosome 3 is a tight cluster of resistance genes and QTL, located within bins 3.04 and 3.05 (Table 2). This cluster contains three dominant disease resistance genes mapping within 5 cM of *umc102*: (i) the *rp3* locus, which confers resistance to *Puccina sorghi* (Schwein.) (Sanz-Alferez et al. 1995); (ii) the *wsm2* locus for resistance to WSMV (McMullen et al. 1994); and (iii) *mv1* for resistance to maize mosaic virus (MMV) (Ming et al. 1995b). Also mapping in bin 3.04 were QTL for resistance to *Fusarium* stalk rot (FSR), caused by *Gibberella zeae* (Schwein.) Petch, and the European corn borer (ECB), *Ostrinia nubilalis* Hübner (Pè et al. 1993; Schön et al. 1993). The *rp3* locus appears to be involved in the pathogen recognition pathway; infection by an avirulent race of *P. sorghi* will result in a localized hypersensitive response (HR) (Bennetzen et al. 1988). Both *wsm2* and *mv1* confer resistance to viral pathogens. While the mechanisms of resistance to these unrelated viruses are currently unknown, neither resistance is characterized by an HR similar to *rp3*. The QTL for ECB resistance in bin 3.04 is associated with degree of tunnel length caused by second generation larvae (Schön et al. 1993), and the FSR QTL is associated with percent stalk internodes infected (Pè et al. 1993). The association between resistance to ECB tunneling and FSR resistance is interesting since ECB tunneling provides entry points for stalk-rotting pathogens. However, the association between QTL for resistance to ECB and FSR is not universal, since only three of the seven ECB QTL identified by Schön and co-workers mapped to the same region as a QTL for FSR identified by Pè and co-workers. Although *O. nubilalis* and *G. zeae* share part of their life cycle within the stalk, the interactions between maize and these organisms are very different. Each interaction between maize and a pathogen or pest will have aspects unique to the invading organism.

Functional relationships.

The question of whether resistance genes within a cluster are functionally related is unclear, particularly because resistance to unrelated organisms is often found within a cluster. Except for a few cases, the biochemical and physiological bases of resistance to pathogens and pests have yet to be elucidated in maize. The only example of a disease resistance gene having been cloned in maize is the *hm1* locus, which confers resistance to carbonum leaf spot caused by *Cochliobolus carbonum* Nelson race 1 (Johal and Briggs 1992). The *Hm1* allele encodes a functional NADP reductase that detoxifies the HC-toxin, a fungal pathotoxin required by *C. carbonum* race 1 for disease development.

Another example of a known maize defense compound is the involvement of the flavone glycoside, maysin, in silk-feeding resistance to the corn earworm, *Helicoverpa zea* (Boddie) (Byrne et al. 1995). Resistance to corn earworm is a quantitative trait, with maysin synthesized via the flavonoid metabolic pathway (Coe et al. 1988). This pathway is well characterized in maize and efforts are currently underway in our laboratory to equate QTL for corn earworm resistance to specific steps in the pathway. The major QTL for maysin concentration corresponds to the transcription regulator gene *p1* located in bin 1.03 near the gene for resistance to maize streak virus, *msv1*, and an FSR QTL (Table 2).

An additional preformed antimicrobial compound—2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA)—has been identified in maize and is associated with resistance to feeding by the first-generation ECB and correlated with northern corn leaf blight (NCLB) lesion expansion, caused by *Setosphaeria turcica* (Luttrell) K. J. Leonard & E. G. Suggs (Coulture et al. 1971; Guthrie et al. 1986). The accumulation of DIMBOA is regulated by the action of the *bx1* locus, which is located in bin 4.02 or 4.03 on the short arm of chromosome 4 (Table 2; Simcox and Weber 1985). This region also contains a dominant resistance gene to *P. sorghi*, *rp4*, and QTL for resistance to first brood larvae of ECB, gray leaf spot (GLS) caused by *Cercosprora zeae-maydis* Theon and Daniels, and NCLB (Schön et al. 1993; Bubeck et al. 1993; Freymark et al. 1993). It would seem unlikely that a preformed antimicrobial compound, such as DIMBOA, would be a primary factor in an *rp4*-mediated HR. However, the presence of a phytotoxic and antimicrobial compound might affect QTL for lesion size and sporulation, which are quantitative parameters for resistance to NCLB and GLS.

Genome duplications in the region of disease resistance loci.

The genome of maize is highly duplicated, with as many as 40% of the genomic RFLP and cDNA probes detecting more than one unlinked locus under high strigency hybridization conditions (Helentjaris 1995; Helentjaris et al. 1988). Furthermore, the existence of duplicate arrays of colinear loci, identified by the same RFLP probe, indicates whole segments of chromosomes are likely duplicated. Examination of the chromosomal position of disease resistance genes and QTL within duplication regions reveals that many resistance loci may be related through duplication. An example is the region around *hm1*, bins 1.06 to 1.07, which is duplicated on the long arm of chromosome 9, near 9.04 to 9.05. The chromosome 9 region contains the *hm2* locus that is involved in adult plant resistance to *C. carbonum* race 1 (Coe et al. 1988; Johal and Briggs 1992). The gene product of the *hm2* locus has yet to be determined, but the *Hm2* allele confers resistance against the same HC-toxin-producing *C. carbonum* isolates as *Hm1*.

On the long arm of chromosome 8, within bin 8.06, are two dominant NCLB resistance genes, *ht2* and *htn1* (Table 1; Simcox and Bennetzen 1993; Zaitlin et al. 1992). The *Ht2* allele confers a chlorotic-necrotic lesion response to infection by an avirulent race of *S. turcica*, analogous to an HR, which inhibits or limits sporulation. On the other hand, the *Htn1* allele results in delay of symptom expression when challenged with an avirulent race. Reduction of sporulation and inhibition of lesion development are two of the parameters that are used to measure quantitative resistance to NCLB (Freymark et al. 1993, 1994). It is interesting to note that when chromosomal regions containing homoeology to bin 8.05 (Helentjaris 1995) are examined, NCLB QTL are also present. RFLP probes mapping to bin 8.05 also detected loci mapping to chromosome 3, bin 3.07 to 3.08, and chromosome 5, bin 5.06 to 5.07. Both of these chromosome regions contain NCLB QTL (Freymark et al. 1993). It therefore appeared that these three genes may represent duplications of the same gene or represent genes derived from an ancient cluster (Helentjaris 1993).

Origin and implication of resistance gene clusters.

Recent reports describing cloning of disease resistance genes conferring HR resistance from *Arabidopsis thaliana*, tobacco, and tomato suggest that resistance genes involved in the recognition of an invading organism represent steps in the signal transduction pathway (Bent et al. 1994; Jones et al. 1994; Martin et al. 1993; Mindrinos et al. 1994; Whitman et al. 1994). There are often linked, duplicated sequences related to the resistance genes (Martin et al. 1994; Sanz-Alferez et al. 1995; Whitman et al. 1994). If these duplicated sequences also function in resistance to other pathotypes of the same pathogen, or other pathogens, a basis for resistance gene clustering for HR genes can be envisioned. The *rp1* complex on the short arm of chromosome 10 consists of duplicated genetic elements that encode alleles for resistance to *P. sorghi* (Bennetzen and Hulbert 1993; Sudapak et al. 1993). Unequal exchange and gene conversion events have been implicated in the instability of resistance at this complex, but also in the evolution of new alleles with altered race specificites (Hu and Hulbert 1994; Scot Hulbert, personal communication). In the case of resistance to rapidly evolving pathogens, the ability of a resistance gene cluster to evolve new specificities might be influenced by the chromosomal location. Distinct regions of the genome might evolve at a pace sufficient to coevolve with changing pathogen/pest challenges.

However, the diversity of organisms and resistance responses of loci mapping within a particular cluster probably rule out the possibility that all genes in clusters are involved in signal transduction. The *bx1* locus controlling the synthesis of DIMBOA and the *p1* locus controlling the synthesis of maysin may be clues to a second class of genes that may lead to gene clustering through duplication. Regulation of the anthocyanin pathway is controlled by a set of *myb*-like (*c1*, *p1*, and *pl1*) and *myc*-like (*b1* and *r1*) transcription regulators (Grotewold et al. 1994). Duplicated elements are often associated with these loci. The involvment of the *p1* locus in the synthesis of maysin has already been discussed, but the action of the *bx1* gene is also consistent with this locus acting as a transcription regulator for genes in the DIMBOA biosynthetic pathway. If other QTL or single genes for resistance are transcription regulators then the resistance genes could be structurally related, such as *myb*-like or *myc*-like, but control biochemical pathways for compounds effective against very diverse pathogens or pests.

Applications in gene mapping and cloning.

Clustering of disease resistance genes suggests several applications to enhance the efficiency of cloning disease and pest-resistance genes in maize. Maize contains several active transposable element systems that have been used successfully to transposon tag and clone genes (Johal and Briggs 1992). One useful characteristic of these transposable element systems is propensity of active elements to transpose more frequently to linked sites than to more distant chromosome regions (Dellaporta and Moreno 1994). This property of maize transposable elements has recently been successfully used to clone disease resistance genes in both tobacco and tomato using the maize *Activator* transposable element (Jones et al. 1994; Whitman et al. 1994). Chromosomal regions containing clusters of resistance genes in maize could be more efficiently targeted for transposon tagging by preselecting active transposable element populations that contain transposable elements linked to the target region (Chang and Peterson 1994).

A second application of the phenomena of resistance gene clustering is the construction of high resolution genetic maps in the region of disease resistance gene clusters, which can then be used in positional cloning of a number of disease resistance traits (Martin et al. 1993; Simcox et al. 1995). Positional cloning in maize is at best a difficult undertaking, due mainly to high amounts of repetitive DNA (Springer et al. 1994). However, the realization that the genomes of maize and related grasses are colinear, despite the occurrence of translocations and inversions, has led to novel approaches for positional cloning (Ahn and Tanksley 1993; Hulbert et al. 1990). This colinearity of grass genomes has led to the premise of the cereals as a single experimental system allowing the exchange of tools and techniques of genome analysis between grass species (Bennetzen and Freeling 1993; Helentjaris 1993). Difficulties in positional cloning in maize could be overcome by utilizing high molecular weight libraries from smaller genomes that are somewhat colinear, such as rice and sorghum (Bennetzen and Freeling 1993). Establishment of colinear relationships of resistance genes across species will greatly aid gene isolation and understanding the actions of genes for resistance.

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LITERATURE CITED

Ahn, S., and Tanksley, S. D. 1993. Comparative linkage maps of the rice and maize genomes. Proc. Natl. Acad. Sci. USA 90:7980-7984.

Bennetzen, J. L., Blevins, W. E., and Ellingboe, A. H. 1988. Cell-autonomous recognition of the rust pathogen determines *Rp1*-specified resistance in maize. Science 241:208-209.

Bennetzen, J. L., and Freeling, M. 1993. Grasses as a single genetic system: genome composition, collinearity and compatibility. Trends Genet. 9:259-261.

Bennetzen, J. L., and Hulbert, S. H. 1993. Organization, instability and evolution of plant disease resistance genes. Plant Mol. Biol. 20:575-578.

Bent, A. F., Kunkel, B. N., Dahlbeck, D., Brown, K. L., Schmidt, R., Giraudat, J., Leung, J., and Staskawicz, B. J. 1994. *RPS2* of *Arabidopsis thaliana*: A leucine-rich repeat class of plant disease resistance genes. Science 265:1856-1860.

Bentolila, S., Guitton, C., Bouvet, N., Sailland, A., Nykaza, S., and Freyssient, G. 1991. Identification of an RFLP marker tightly linked to the *Ht1* gene in maize. Theor. Appl. Genet. 82:393-398.

Bubeck, D. M., Goodman, M. M., Beavis, W. D., and Grant, D. 1993. Quantitative trait loci controlling resistance to gray leaf spot in maize. Crop Sci. 33:838-847.

Burnham, C. R. 1982. The location of genes to chromosome by the use of chromosomal interchanges. Pages 65-70 in: Maize for Biological Research. W. F. Sheridan, ed. Plant Molecular Biology Association, Charlottesville, VA.

Byrne, P. F., McMullen, M. D., Snook, M. E., Musket, T., Widstrom, N. W., and Wiseman, B. R. 1995. Quantitative genetic analysis of loci controlling synthesis of maysin, a corn earworm resistance factor, in maize silks. Maize Genet. Coop. Newsl. 69:53-54.

Chang, R.-Y., and Peterson, P. A. 1994. Chromosome labeling with

transposable elements in maize. Theor. Appl. Genet. 87:650-656.
Coe, E. H., Jr., Neuffer, M. G., and Hoisington, D. A. 1988. The Genetics of Corn. Pages 81-258 in: Corn and Corn Improvement. G. F. Sprague and J. W. Dudley, eds. Crop Science Society of America, Inc., Madison, WI.
Coulture, R. M., Routley, D. G., and Dunn, G. M. 1971. Role of cyclic hydroxamic acids in monogenic resistance of maize to *Helminthosporium turcicum*. Physiol. Plant Pathol. 1:515-521.
Dellaporta, S. L., and Moreno, M. 1994. Gene tagging with Ac-Ds elements in maize. Pages 219-233 in: The Maize Handbook. M. Freeling and V. Walbot, eds. Springer Verlag, New York.
Freymark, P. J., Lee, M., Martinson, C. A., and Woodman, W. L. 1994. Molecular-marker-facilitated investigation of host-plant response to *Exserohilum turcicum* in maize (*Zea mays* L.): Components of resistance. Theor. Appl. Genet. 88:305-313.
Freymark, P. J., Lee, M., Woodman, W. L., and Martinson, C. A. 1993. Quantitative and qualitative trait loci affecting host-plant response to *Exserohilum turcicum* in maize (*Zea mays* L.). Theor. Appl. Genet. 87:537-544.
Gardiner, J., Melia-Hancock, S., Hoisington, D. A., Chao, S., and Coe, E. H. 1993. Development of a core RFLP map in maize using an immortalized-F2 population. Genetics 134:917-930.
Grotewold, E., Drummond, B. J., Bowen, B., and Peterson, T. 1994. The *myb*-homologous *P* gene controls phlobaphene pigmentation in maize floral organs by directly activating a flavonoid biosynthetic gene subset. Cell 76:543-553.
Guthrie, W. D., Wilson, R. L., Coats, J. R., Robbins, J. C., Tseng, C. T., Jarvis, J. L., and Russell, W. A. 1986. European corn borer (*Lepidoptera*: *Pyralidae*) leaf-feeding resistance and DIMBOA content in inbred lines of dent maize grown under field versus greenhouse conditions. J. Econ. Entomol. 79:1492-1496.
Helentjaris, T. 1993. Implications for conserved genomic structure among plant species. Proc. Natl. Acad. Sci. USA 90:8308-8309.
Helentjaris, T. 1995. Atlas of duplicated sequences in maize. Maize Genet. Coop. Newsl. 69:67-81.
Helentjaris, T., Weber, D., and Wright, S. 1988. Identification of the genomic locations of duplicated nucleotide sequences in maize by analysis of restriction fragment length polymorphisms. Genetics 118:353-363.
Hoisington, D. A., and Coe, E. H., Jr. 1989. Methods for correlating RFLP maps with conventional genetic and physical maps in maize. Pages 19-24 in: Development and Application of Molecular Markers to Problems in Plant Genetics. T. Helentjaris and B. Burr, eds. Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.
Hu, G., and Hulbert, S. 1994. Evidence for the involvment of gene conversion in meiotic instability of the *Rp1* rust resistance genes of maize. Genome 37:742-746.
Hulbert, S. H., Richter, T. E., Axtell, J. D., and Bennetzen, J. L. 1990. Genetic mapping and characterization of sorghum and related crops by means of maize DNA probes. Proc. Natl. Acad. Sci. USA 87:4251-4255.
Johal, G. S., and Briggs, S. P. 1992. Reductase activity encoded by the *HM1* disease resistance gene in maize. Science 258:985-987.
Jones, D. A., Thomas, C. M., Hammond-Kosack, K. E., Balint-Kurti, P. J., and Jones, J. D. G. 1994. Isolation of the tomato *Cf-9* gene for resistance to *Cladosporium fulvum* by transposon tagging. Science 266:789-793.
Jung, M., Weldekidan, T., Schaff, D., Paterson, A., Tingey, S., and Hawk, J. 1994. Generation-means analysis and quantitative trait locus mapping of anthracnose stalk rot genes in maize. Theor. Appl. Genet. 89:413-418.
Khavkin, E. E., and Coe, E. H. 1995a. Organization of growth-regulating genes in maize: 1. The functional clusters of genes. Russ. J. Plant Physiol. 42:408-420.
Khavkin, E. E., and Coe, E. H. 1995b. Organization of growth-regulating genes in maize: 2. Quantitative trait loci. Russ. J. Plant Physiol. 42:558-574.
Kyetere, D., Ming, R., McMullen, M., Pratt, R., Brewbaker, J., Musket, T., Pixley, K., and Moon, H. 1995. Monogenic resistance to maize streak virus maps to the short arm of chromosome 1. Maize Genet. Coop. Newsl. 69:136-137.
Louie, R., Findley, W. R., Knoke, J. K., and McMullen, M. D. 1991. Genetic basis of resistance in maize to five maize dwarf mosaic virus strains. Crop Sci. 31:14-18.
Martin, G. B., Brommonschenkel, S. H., Chunwongse, J., Frary, A., Ganal, M. W., Spivey, R., Wu, T., Earle, E. D., and Tanksley, S. D. 1993. Map-based cloning of a protein kinase gene conferring disease resistance in tomato. Science 262:1432-1436.
Martin, G. B., Frary, A., Wu, T., Brommonschenkel, S., Chunwongse, J., Earle, E. D., and Tanksley, S. D. 1994. A member of the tomato *Pto* gene family confers sensitivity to fenthion resulting in rapid cell death. Plant Cell 6:1543-1552.
McMullen, M. D., Jones, M. W., Simcox, K. D., and Louie, R. 1994. Three genetic loci control resistance to wheat streak mosaic virus in the maize inbred Pa405. Mol. Plant-Microbe Interact. 7:708-712.
McMullen, M. D., and Louie, R. 1991. Identification of a gene for resistance to wheat streak mosaic virus in maize. Phytopathology 81:624-627.
Mindrinos, M., Katagiri, F., Yu, G.-L., and Ausubel, F. M. 1994. The *A. thaliana* disease resistance gene *RPS2* encodes a protein containing a nucelotide-binding site and leucine-rich repeats. Cell 78:1089-1099.
Ming, R., McMullen, M. D., Brewbaker J. L., Pratt, R. C., Moon, H. G., Musket, T., and Holley, R. 1995a. RFLP mapping of genes conferring resistance to *Erwinia stewartii*. Maize Genet. Coop. Newsl. 69:60.
Ming, R., McMullen, M. D., Brewbaker J. L., Pratt, R. C., Moon, H. G., Musket, T., and Kyetere, D. T. 1995b. RFLP mapping of maize mosaic virus resistance gene. Maize Genet. Coop. Newsl. 69:60-61.
Pè, M. E., Gianfranceschi, L., Taramino, G., Tarchini, R., Angelini, P., Dani, M., and Binelli, G. 1993. Mapping quantitatvie trait loci (QTL) for resistance to *Gibberella zeae* infection in maize. Mol. Gen. Genet. 241:11-16.
Prince, J. P., and Tanksley, S. D. 1992. Restriction fragment length polymorphism in plant breeding and genetics. Proc. Royal Soc. Edin. 99B:23-29.
Sanz-Alferez, S., Richter, T. E., Hulbert, S. H., and Bennetzen, J. L. 1995. The *Rp3* disease resistance gene of maize: mapping and characterization of introgressed alleles. Theor. Appl. Genet. 91:25-32.
Schön, C. C., Lee, M., Melchinger, A. E., Guthrie, W. D., and Woodman, W. L. 1993. Mapping and characterization of quantitative trait loci affecting resistance against second-generation European corn borer in maize with the aid of RFLPs. Heredity 70:648-659.
Simcox, K. D., and Bennetzen, J. L. 1993. The use of molecular markers to study *Setosphaeria turcica* resistance in maize. Phytopathology 83:1326-1330.
Simcox, K. D., McMullen, M. D., and Louie, R. 1995. Co-segregation of the maize dwarf mosaic virus resistance gene, *mdm1*, with the nucleolus organizer region in maize. Theor. Appl. Genet. 90:341-346.
Simcox, K. D., and Weber, D. F. 1985. Location of the benzoxazinless (*bx*) locus in maize by monosomic and B-A translocational analyses. Crop Sci. 25:827-830.
Snedecor, G. W., and Cochran, W. G. 1967. Statistical Methods. The Iowa State University Press, Ames. p. 235.
Springer, P., Edwards, K., and Bennetzen, J. L. 1994. DNA class organization on maize *Adh1* yeast artifical chromosomes. Proc. Natl. Acad. Sci. USA 91:863-867.
Sudupak, M. A., Bennetzen, J. L., and Hulbert, S. H. 1993. Unequal exchange and meiotic instability of disease-resistance genes in the *Rp1* region of maize. Genetics 133:119-125.
Whitman, S., Dinesh-Kumar, S. P., Choi, D., Hehl, R., Corr, C., and Baker, B. 1994. The product of the tobacco mosaic virus resistance gene *N*: similarity to toll and the interleukin-1 receptor. Cell 78:1101-1115.
Zaitlin, D., DeMars, S., and Gupta, M. 1992. Linkage of a second gene for NCLB resistance to molecular markers in maize. Maize Genet. Coop. Newsl. 66:69-70.
Zaitlin, D., DeMars, S., and Ma, Y. 1993. Linkage of *rhm*, a recessive gene for resistance to southern corn leaf blight, to RFLP marker loci in maize (*Zea mays*) seedlings. Genome 36:555-564.