Mapping Components of Partial Resistance to Northern Leaf Blight of Maize Using Reciprocal Translocations

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Accepted for publication 3 September 1991 (submitted for electronic processing).

ABSTRACT

Brewster, V. A., Carson, M. L., and Wicks, Z. W., III. 1992. Mapping components of partial resistance to northern leaf blight of maize using reciprocal translocation. Phytopathology 82:225-229.

Maize F_2 populations were constructed by crossing a series of northern leaf blight (NLB, caused by *Exserohilum turcicum*) susceptible reciprocal translocation stocks with the partially resistant inbred line Mo17, followed by one generation of selfing. Translocation breakpoints were marked with the waxy (*wx*) recessive gene and F_2 seed were classified as waxy or nonwaxy before planting. F_2 plants inoculated with *E. turcicum* were evaluated in the field and greenhouse for infection efficiency, incubation period, lesion length, lesion expansion rate, and sporulation. F_2 plants in field experiments were also evaluated for disease severity on adult plants. Ratings of waxy and nonwaxy F_2 plants were compared using Student's *t* tests to determine if one or both of the chromosome arms involved in the reciprocal translocation carried a gene or genes for NLB resistance. Partial resistance (as expressed by disease severity on adult plants in the field) in Mo17 was consistently associated with factors on chromosome 3, the short arm of chromosome 4, and the long arm of chromosome 6. Measures of components of partial resistance on greenhouse-grown seedlings were rarely significant and were not in agreement with ratings of disease severity on adult plants in the field. Based on measurements on older but still vegetative plants in the field, the gene or genes on the short arm of chromosome 4 and the long arm of chromosome 6 appeared to be related to a reduction in lesion numbers and an increase in incubation period, both of which were correlated with disease severity. Lesion length, lesion expansion rate, and sporulation intensity were not associated with partial resistance in Mo17. The lack of consistency in expression of components of partial resistance in seedling plants may be due to environmental and developmental regulation of genes for resistance.

Additional keywords: corn, *Helminthosporium turcicum*, *Zea mays*.

Northern leaf blight (NLB), caused by *Exserohilum turcicum* (Pass.) Leonard & Suggs (syn = *Helminthosporium turcicum* Pass.), occurs in all maize-growing regions of the world where temperatures are moderate and heavy dews are frequent (25). In the northern and central corn belt of the United States, NLB can become a major disease problem. Depending on the time of disease onset and the presence of adequate moisture, yield losses as high as 68% can occur (8,10,23,28,29). If infection begins before anthesis and grain fill, losses will be more severe than if infection begins after grain fill is underway (3,8,22,28,29).

Resistance to NLB can be expressed in two different ways (9). Hooker described a chlorotic-lesion resistance that is monogenically inherited (6,18,27). This type of lesion is small with a pronounced area of chlorosis but little or no necrotic tissue. Sporulation from these lesions is negligible or completely suppressed (6). Three genes, *Ht*1, *Ht*2, and *Ht*3, that result in this type of resistance have been identified (10). An additional gene that increases incubation and latent periods has been designated as *HtN* (17,22).

Partial resistance to NLB that is under polygenic control has been demonstrated by several workers (3, 12–15). Working with crosses involving three susceptible and five resistance inbred lines, Jenkins and Robert concluded that resistance is expressed as fewer and smaller lesions, and the degree of expression can vary depending on the genetic content of the particular line (14).

Partial resistance can consist of several measurable components (5,20): infection efficiency (IE) = number of infection points resulting from a given dose of inoculum; incubation period (ICP) = number of days from inoculation to the first appearance of symptoms; latent period (LP) = number of days from primary inoculation to the production of secondary inoculum; lesion size (LS) = length or area of individual lesions; lesion expansion rate (LR) = average daily increase in lesion length; lesion number (LN or infection frequency) = number of lesions produced with a given inoculum dose; and sporulation (SP) = number of spores produced per unit area of a lesion. Generally, a low IE, long ICP, long LP, small LS, slow LR, low LN, and low SP would all contribute to lower disease severity or partial resistance.

The association of these components with each other and their relation to partial resistance varies substantially among different pathosystems. In the wheat:*Septoria nodorum* system, LP was of little use for screening cultivars, but a resistance index based on ICP and SP gave satisfactory results in identifying cultivars with partial resistance (5). LS did not appear to contribute to resistance in the barley:*Puccinia hordei* and wheat:*S. nodorum* systems (5,21). In the barley:*P. hordei* system, there was no correlation between spore production and resistance (20), and all of these components taken together may not adequately explain the observed resistance of some cultivars. The growth stage of the plant must also be considered when interpreting data on these components, because seedlings frequently produce disease reactions dissimilar to those produced in an adult plant (7,20,21). Genetic control of LR by the host in the maize:*E. turcicum* pathosystem is evident but expressed to a greater extent in adult plants than in seedlings (7,16,26).

Resistance genes, or blocks of genes, can be mapped to points or areas on individual chromosomes. In the case of partial resistance where single gene action is not evident and multiple genes are suspected of contributing to the resistance phenotype, reciprocal translocation stocks have been used to locate areas of the chromosome arms that condition resistance (2,14,15). In the maize:*E. turcicum* system, five resistant lines were studied (14). Four of these lines received their resistance from the same source, and the fifth line was not closely related to the others. In all five lines, the short arm of chromosome 7 and the long arm of chromosome 5 were associated with resistance, but all of the lines varied as to additional numbers and location of chromosome arms associated with resistance.

In this study, we have attempted to map components of partial resistance to NLB to specific chromosome arms using a series of reciprocal translocation stocks. The components of interest

are IE, ICP, LR, LN, LS, SP, and the total expression of these components, disease severity (DS).

MATERIALS AND METHODS

A series of reciprocal translocation stocks, marked with the recessive gene for waxy endosperm (*wx*), were obtained from the Maize Genetics Cooperative, Urbana, IL. All had been backcrossed four to nine times to the susceptible inbred W23 and then selfed two to seven times to obtain a series of near-isogenic lines differing mainly in the location of translocation breakpoints. The translocations are listed, along with their breakpoints, in Table 1. These waxy, NLB-susceptible translocation lines were then crossed with the partially resistant inbred Mo17 (22). F_1 plants were selfed, and the F_2 seeds were segregated in a 3:1 ratio for normal and waxy endosperm. If resistance in Mo17 is linked to the translocation breakpoint, it will segregate with the normal endosperm (*Wx*) seeds and susceptibility with the waxy endosperm (*wxwx*) seeds. In this case, disease ratings for plants grown from normal seeds would be expected to be significantly lower than ratings from susceptible plants grown from waxy seeds, indicating that one or both chromosome arms involved in the reciprocal translocation carry gene(s) for resistance.

TABLE 1. Maize reciprocal translocation stocks and location of breakpoints on translocated chromosomes

Translocation	Breakpoints[y]	
1-9c	1S.48	9L.22
$1\text{-}9_{4995}$	1L.19	9S.20
$1\text{-}9_{8389}$	1L.74	9L.13
2-9b	2S.18	9L.22
3-9c[z]	3L.09	9L.12
	(3S.15	9L.20)
4-9b	4L.90	9L.29
4-9g	4S.27	9L.27
$4\text{-}9_{5657}$	4L.33	9S.25
5-9a	5L.69	9S.17
5-9c	5S.07	9L.10
6-9a	6S.79	9L.40
6-9b	6L.10	9S.37
7-9a	7L.63	9S.07
$7\text{-}9_{4363}$	7 ctr.	9 ctr.
8-9d	8L.09	9S.16
$8\text{-}9_{6673}$	8L.35	9S.31

[y] Breakpoint locations are indicated as a proportion of the total distance of the chromosome arm from the centromere to the distal end.
[z] Both the breakpoints are listed in (20).

In field experiments in 1987 and 1988, each translocation × Mo17 F_2 population was treated as a separate experiment. Before planting, seeds were separated by endosperm phenotype. Waxy (*wxwx*) and nonwaxy (*Wx*) seeds were then planted in a completely randomized design with plants as experimental units. Experiments consisted of two to 18 rows, depending on seed availability, of 20 plants each and one row of each parent. Rows were 6.1 m long with 91 cm between rows. In 1987, planting took place on 15 May with the first inoculation on 11 June and the second 1 July. In 1988, planting was done on 13 May with inoculations on 17 and 23 of June. In both years, plants at the three- to five-leaf stage were first inoculated by placing 20.9 ml of inoculum into the leaf whorl. Inoculum consisted of finely ground, dried leaf tissue that was infected with race 0 of *E. turcicum* and had been collected from the field the previous year. Inoculum was measured so that ICP and LN would be based on the first infection cycle initiated by equal amounts of inoculum. Ground leaf tissue was also used for the second inoculation, but was not measured because the purpose at this point was to initiate a substantial epidemic.

Total lesions on each plant were counted six times over 8 days from 19 June to 26 June in 1987 (8–15 days after inoculation, six- to eight-leaf stage) and five times over 10 days from 5 July to 14 July in 1988 (19–28 days after inoculation, late whorl growth stage). In 1987, ICP was measured as the day at which 50% of the lesions were visible, which was determined by linear regression of the probit transformation of the lesion number over time (4,24). In both years, ICP was also measured as the number of days from the first inoculation until the first lesion was visible. LN in both years was the total number of lesions on each plant counted on the last measurement day.

In 1988, one random lesion on each plant was marked, and the length was measured every day that lesions were counted for a total of five measurements for each lesion. LR was defined as the slope from the linear regression of lesion length over time.

TABLE 2. Differences between means of six components of partial resistance of maize plants grown from *Wx* seeds and plants grown from *wxwx* seeds in translocation × Mo17 F_2 populations[r]

	1987					1988					
Translocation	ICP[s]	LN[t]	LS[u]	SP[v]	DS[w]	ICP	LR[x]	LN	LS	SP	DS
1-9c	0.43	0.164	2.18**[y]	24	0.00	0.71	−0.032	−0.73	−0.14	−49	−0.21
$1\text{-}9_{4995}$	0.05	−0.414	1.74*	80	−0.33	1.03*	0.050	−1.03*	−0.03	224**	−0.14
$1\text{-}9_{8389}$	−0.57*	0.673*	1.67*	110	0.08	0.05	0.057	−0.98	1.19	283**	0.10
2-9b	0.17	−0.560*	−0.59	−149*	0.43**	0.18	0.029	−0.51	0.18	126*	−0.40*
3-9c	0.04	0.133	1.26*	−18	−0.36**	0.89*	0.067*	−1.35**	0.43	38	−0.47**
4-9b	0.34	−0.550	2.33**	0	−0.28	0.54	0.008	−0.81	−0.29	106*	−0.17
$4\text{-}9_{5657}$	0.04	−0.242	0.55	132	−0.12	0.71	0.065*	−1.40**	1.25	−11	−0.23
4-9g	1.20**	−0.864**	0.02	34	−0.6**	0.31	−0.020	−0.69*	1.11	227**	−0.44**
5-9a	0.33	−0.087	−0.35	147	0.29	0.25	−0.022	0.06	0.78	−76	−0.16
5-9c	−0.26	0.500*	0.38	140*	−0.12	0.12	0.006	−1.19**	0.08	−65	−0.17
6-9a	0.42	−0.208	0.26	54	−1.04**	...[z]	...	...	...	...	...
6-9b	0.34	−0.575	−0.03	4	−0.87**	1.27**	−0.026	−3.05**	0.46	−73	−0.27*
7-9a	0.82**	−1.056**	−0.88	136	−0.38	0.09	−0.016	−0.80	0.71	−100*	−0.16
$7\text{-}9b_{4363}$	−1.49**	0.114	1.33	87	0.64**	...	...	...	...	...	...
8-9d	0.43	0.680	1.82	109	0.22	0.17	0.005	0.21	−0.06	36	0.13
$8\text{-}9_{6673}$	−0.38	0.797**	1.54**	25	0.69**	0.21	0.072	0.87	1.48	−114	0.17

[r] Plants were inoculated with *Exserohilum turcicum* in field trials in summers of 1987 and 1988 at Brookings, SD.
[s] ICP = Incubation period. The number of days between inoculation and the appearance of the first lesion.
[t] LN = Lesion number. Number of lesions 15 days after inoculation.
[u] LS = Lesion size. Length of lesions measured in centimeters.
[v] SP = Sporulation. Number of conidia per square millimeter from a sporulating area of the lesion.
[w] DS = Disease severity as estimated according to the Horsfall-Barratt scale. Ratings were taken during the week of August 20.
[x] LR = Lesion expansion rate measured in centimeters per day. This was not measured in 1987.
[y] * = $P \leq 0.05$.** = $P \leq 0.01$.1
[z] These populations were not evaluated in 1988.

DS ratings were recorded weekly for each plant beginning at anthesis (30 July in 1987 and 28 July in 1988), for 6 and 5 wk, respectively. Ratings were based on the Horsfall-Barratt scale (11).

After the final disease rating was taken, one randomly selected leaf with one distinct lesion was harvested from each plant. Leaves harvested were usually one to four leaves below the top ear. These leaves were pressed and dried and LS (length) was measured in centimeters. Dried lesions were placed in individual moist chambers and allowed to sporulate for 3 days. After incubation, four disks were punched out of each lesion with a No. 6 cork borer (11 mm diameter), and spores were washed off in 1 ml of water. Spores were then counted with a hemacytometer, and numbers were converted to spores per millimeter squared of sporulating lesion area.

Greenhouse experiments were done in February and March of 1988. Seeds of each translocation × Mo17 F_2 population were separated as for the field experiments, and 20 waxy and 20 normal seeds from each population (seed supply permitting) were planted in 38 × 44 cm flats in a pasteurized soil mix (2:1:1, loam:peat:sand, v:v:v) on 3 February. On 22 February, plants at the two- to four-leaf stage were inoculated by placing 4,000 conidia, obtained from sporulated leaf tissue and suspended in 50 μl of water, into the whorl of the plant. Infection points, visible as very small, chlorotic flecks, were counted 5–7 days after inoculation as a measurement of IE. Lesions were counted every day from 29 February to 9 March. ICP was calculated two different ways as described for field studies.

On 10 March, all leaves with infection points and lesions were cut off the plants (six- to eight-leaf stage), and the experiment was repeated by reinoculating the same plants on 11 March using the same technique but reducing the inoculum from 4,000 to 1,000 spores per 50-μl drop. IE, ICP, and LN were measured as for the February experiment with counts taken every day from 20 March to 31 March. LR was also measured in this experiment by marking one lesion per plant and measuring its length once a day when lesion counts were taken. LR was determined as it was for the 1988 field experiment.

For field and greenhouse experiments, the means of plants grown from *Wx* and *wxwx* seeds from each translocation × Mo17 F_2 population were compared for each parameter measured. The test criterion used was the two-tailed Student's *t* test. A significant difference in a given population would indicate that a gene (or genes) for resistance is located on the chromosome arm involved in that translocation.

RESULTS

Mean differences in IE, ICP, LR, LN, LS, SP, and DS between plants grown from normal endosperm (*Wx*) seeds and plants grown from waxy endosperm (*wxwx*) seeds, from translocation × Mo17 F_2 populations are shown in Tables 2 and 3. If the resistance gene or genes from Mo17 are linked to the translocation breakpoint, a significant difference in the means should be detectable. Linkage of resistance genes from Mo17 with the translocation breakpoint would be expressed as significant differences that would be negative for IE, positive for ICP, and negative for LR, LN, LS, SP, and DS.

In the 1987 field experiments, ICP was initially analyzed using probit transformations (4,24). There were no significant differences in ICP using the probit transformation (data not shown), but there were four significant differences when ICP was measured by counting the number of days between inoculation and the appearance of the first lesion. ICPs from probit analyses of the greenhouse data were inconsistent between the two experiments (Table 3). In the February experiment, two of the translocation × Mo17 F_2 populations, *wx*Δ4-9g and *wx*Δ6-9b, had significant differences between plants grown from *Wx* seed and plants grown from *wxwx* seeds. In the March experiment, three different F_2 populations, *wx*Δ1-9_{8389}, *wx*Δ7-9_{4363}, and *wx*Δ8-9_{6673}, had significant differences. Using the alternate method of measuring ICP, the *wx*Δ6-9b F_2 population in both experiments had significant differences. The probit analysis was not used in the 1988 field experiments.

In the greenhouse experiments, IE, LR, and LN were measured in addition to the ICP discussed above. LR was only measured in the March experiment, and no significant differences were found. IE was measured in both experiments. Two translocation × Mo17 F_2 populations, *wx*Δ2-9b in the February experiment and *wx*Δ1-9_{8389} in the March experiment, had significant ($P < 0.10$) differences between plants grown from *Wx* seeds and plants grown from *wxwx* seeds. The *wx*Δ1-9_{8389} population in the March experiment also had a significant difference for ICP using probit

TABLE 3. Differences between means of five components of partial resistance of maize plants grown from *Wx* seeds and plants grown from *wxwx* seeds in translocation × Mo17 F_2 populations[t]

	February				March				
Translocation	IE[u]	Probit[v]	ICP[w]	LN[x]	IE	Probit	ICP	LR[y]	LN
1-9c	−6.19	−0.43	0.09	−0.24	4.38	0.90	0.46	0.0332	−0.01
1-9_{4995}	11.40	0.00	0.32	−1.50	5.30	0.84	0.86	−0.0910	−0.19
1-9_{8389}	0.52	0.17	0.10	0.12	−11.04[+z]	1.26[+]	1.23	0.0862	−0.87
2-9b	30.60[+]	−0.04	0.00	1.66	1.90	0.27	−0.01	−0.1030	0.59
3-9c	16.62	0.60	−0.17	−0.44	−2.77	−0.06	0.11	0.0870	0.29
4-9b	−25.39	0.80	0.70	−0.42	9.60	0.21	0.30	0.1852	1.01
4-9_{5657}	−16.22	0.72	0.76	0.90	−4.72	0.14	0.49	−0.0498	−0.15
4-9g	−19.77	0.82[+]	0.53	0.15	0.68	−0.38	0.04	−0.1520	0.70
5-9a	3.73	0.32	−0.13	0.61	−2.76	0.21	0.33	0.1580	−1.05
5-9c	−9.83	0.21	0.21	−0.32	5.79	0.07	0.16	−0.0820	−0.15
6-9a	−8.70	0.58	0.30	0.10	−1.60	1.06	0.75	0.2073	0.25
6-9b	16.33	−0.89*	−0.85*	0.33	3.07	−0.29	−0.11*	0.1280	0.61
7-9a	11.21	−0.43	0.26	−0.56	−4.33	0.48	0.54	−0.0310	0.19
7-9_{4363}	−12.83	1.22	0.92	1.25	11.00	−2.82[+]	−1.25	0.0350	0.83
8-9d	−0.44	−0.24	−0.38	−0.91	12.82	0.70	0.91	−0.0725	0.82
8-9_{6673}	16.96	−0.27	−0.44	0.18	−4.07	−1.04[+]	−1.08	−0.1810	−0.36

[t] Plants were inoculated in the greenhouse with *Exserohilum turcicum* in February and March 1988 at Brookings, SD.
[u] IE = Infection efficiency. The number of infection points.
[v] ICP Probit = Incubation period. The day at which 50% of the lesions appeared based on probit analysis.
[w] ICP = Incubation period. The day, after inoculation, on which the first lesion appeared.
[x] LN = Lesion number. Number of lesions 15 days after inoculation.
[y] LR = Lesion expansion rate, measured in centimeters per day. This was not measured in the February experiment.
[z] + = $P \leq 0.10$. * = $P \leq 0.05$.

analysis. There were no significant differences for LN in either greenhouse experiment.

Three populations, *wx*Δ3-9c, *wx*Δ4-9g, and *wx*Δ6-9b, all had negative significant differences for DS ratings for both years (Table 2). This indicates that genes for resistance in Mo17 are on chromosomes 3, 4, and 6. The DS ratings taken during the week of 20 August 1987 and 18 August 1988 were the ratings used in the final analysis because ratings taken after that were beginning to be confounded with natural senescence. Two populations were not grown in 1988 due to insufficient seed. The $wx\Delta 8\text{-}9_{6673}$ population had a positive significant difference in DS in 1987, but no significant difference in 1988, and the *wx*Δ2-9b population had a positive significant difference in 1987 but a negative significant difference in 1988.

Mean differences for ICP, IR, LN, LS, and SP were often inconsistent between the 2 yr, and those measurements that were significantly different often did not correspond with significant differences in DS ratings. The F_2 populations involving $wx\Delta 4\text{-}9g$, *wx*Δ6-9b, and $wx\Delta 8\text{-}9_{6673}$ each had significant differences for two components that support the significant differences observed in their DS ratings in either 1987 or 1988. Mo17 has a gene (or genes) for resistance on the short arm of chromosome 4 based on a negative significant difference in DS ratings, and this is reflected in a longer ICP and fewer lesions in 1987. This resistance, based on DS ratings, was still expressed in 1988 and again was reflected by fewer lesions, although there were no detectable differences in ICP, and SP was significantly greater. DS ratings also indicated the presence of resistance genes on the long arm of chromosome 6 in both years, and this was supported by a longer ICP and fewer lesions in 1988. DS ratings for the *wx*Δ3-9c population indicate the presence of resistance genes on chromosome 3 in Mo17, and this is supported by a longer ICP and fewer lesions in 1988, but these data are contradicted by greater lesion size in 1987 and a faster lesion expansion rate in 1988. The population involving the short arm of chromosome 2 gave completely opposite results between the 2 yr. In 1987, a positive significant difference in the DS ratings was contradicted by lower spore production and fewer lesions, and in 1988 a negative significant difference in DS was contradicted by higher spore production. The F_2 population involving $wx\Delta 7\text{-}9_{4363}$ indicated genes for susceptibility in Mo17 on chromosome 7, as indicated by DS ratings and the significantly shorter ICP. The short arm of chromosome 6 appeared to have genes for resistance based on DS ratings, but this observation was not supported by data on any of the components of partial resistance. There were other significant differences between means for the various components in the other translocation × Mo17 F_2 populations, although none of them were associated with significant differences in DS ratings.

Mean differences in DS between *Wx* and *wxwx* adult plants in the field were significantly correlated ($P < 0.10$) with ICP in 1987 and in the combined data, and with LN in both years and in the combined data (Table 4). DS was not significantly associated with LR, LS, or SP in the field experiments. Differences in DS were not significantly correlated with any of the components measured on seedlings in the greenhouse (data not shown).

DISCUSSION

This particular series of chromosome translocation stocks is limited in its representation of the corn genome. Chromosome 10 is not represented at all and neither are the short arms of chromosomes 7 and 8 or the long arm of chromosome 2. The breakpoints for *wx*Δ3-9c are questionable (19). The short arms of chromosomes 1, 2, 4, 5, and 6 and the long arms of chromosomes 5 and 6 are only represented by a single breakpoint so that any resistance/susceptibility genes more than 50 cM from the breakpoint would not be detectable, although recombination values near the translocation site will be reduced.

The stage of plant growth can have a major effect on the expression of partial resistance (20,21). In the barley:*P. hordei* system, latent period was measured on six different cultivars that varied in their level of partial resistance (21). When latent period was measured on seedlings there was little difference between cultivars; however, when it was measured on the flag leaf, the length of the latent period accurately reflected the differences in resistance between cultivars. Hilu and Hooker examined the penetration and growth of *E. turcicum* in corn seedlings with no resistance and partial resistance (7). They observed that hyphal growth was slowed and that onset of lesion expansion (ICP) was delayed in seedlings with partial resistance. The *wx*Δ3-9c, *wx*Δ4-9g, and *wx*Δ6-9b translocation × Mo17 F_2 populations had significant positive differences for ICP in at least 1 yr in the field that also were reflected in differences in DS on adult plants. Similarly, a significant negative difference in ICP in the $wx\Delta 7\text{-}9_{4363}$ supported a significant positive difference in DS, indicating a possible gene for susceptibility on chromosome 7 in Mo17. Differences in the probit of ICP in these populations in the greenhouse did not necessarily support differences in DS seen in the field. The inconsistency between data from the different field and greenhouse experiments make conclusions about the association of ICP and DS ratings or chromosomal location of genes that might govern ICP in this pathosystem tentative.

LS and SP appear to be influenced more by the environmental conditions or by the level of stress on the host plant than by genetics of the host. Disease development was poor in 1988, and there were no significant differences between means for lesion size that year as opposed to six significant differences in the previous year. Four of these differences were not associated with a significant difference in DS ratings, and all of them indicated longer lesions were associated with genes from the resistant inbred Mo17. There were six significant differences for SP in 1988 compared with only two in 1987. In the *wx*Δ2-9b and *wx*Δ4-9g populations, SP was associated with significant differences in DS ratings but indicated higher sporulation on plants rated as resistant based on DS ratings. Because SP was measured on dried lesions in the lab, our ratings may not reflect sporulation as it occurs on live plants in the field. Although LS and SP may be a response to infection, they seem to be highly modified by conditions outside the scope of resistance reactions governed by the genetics of the host.

Data from the greenhouse or the field experiments were not consistent enough to definitively map to chromosome arm genes responsible for the expression of IE, ICP, LR, LN, LS, or SP as components of partial resistance to NLB. Of these components, ICP and LN appear to be most important in the partial resistance of Mo17 and deserve further study in future mapping experiments. Inconsistencies in the field data may be due to the differences in the weather experienced between the two field seasons. Plots were planted on approximately the same date but very dry weather delayed the onset of the epidemic and, consequently, plants were at a more advanced growth stage in 1988 when many of the data were recorded. DS ratings were fairly consistent between the two years because at the time the ratings were taken, plants

TABLE 4. Correlation coefficients among mean differences in components of partial resistance to *Exserohilum turcicum* and disease severity on adult plants between *Wx* and *wxwx* maize plants in Mo17 × translocation F_2 populations evaluated over 2 yr in the field[t]

Component	ICP[u]	LR[v]	LN[w]	LS[x]	SP[y]
vs DS[z] (1987)	−0.58*	···	0.53*	0.24	0.00
vs DS (1988)	−0.40	0.22	0.49+	0.17	−0.11
vs DS (combined)	−0.54**	0.22	0.41*	0.24	−0.02

[t] +, *, ** indicate correlation coefficient if significantly different from zero at the 0.10, 0.05, and 0.01 levels of probability, respectively.
[u] ICP = Incubation period. The number of days after inoculation until the first lesion appeared.
[v] LR = Lesion expansion rate, measured in centimeters per day.
[w] LN = Number of lesions 15 days after inoculation.
[x] LS = Lesion size. Length of lesions in centimeters.
[y] SP = Sporulation. Number of conidia produced per square millimeter of sporulating lesion.
[z] DS = Disease severity as estimated according to the Horsfall-Barratt scale. Ratings were taken the week of August 20.

were approximately the same age. The same argument can be used to explain the inconsistencies between the two greenhouse experiments. Even though infection was induced to progress at the same rate, the plants were older at the time of the second experiment. Different genes may be expressed at different ages of the plant, and there is also the possibility that the 1988 data may be reflecting the activity/inactivity of temperature-sensitive genes.

The assumption that the genes involved in the expression of these components act in a completely or partially dominant fashion may be confounding results. If any one of the genetic factors involved is recessive or incomplete in its penetrance (expression), the means derived from the plants grown from *Wx* seeds, two-thirds of which are heterozygous (*Wxwx*), would be skewed toward those from plants grown from *wxwx* seeds. If the experiments were conducted in the field, or if adequate seed sets could be produced in the greenhouse, plants could be rated at the end of the season for semi-sterility. Because semi-sterility is the result of the poor fertilization capabilities of heterozygous plants (1), data from these plants could be discarded and the means derived only from homozygous, normal plants. Unfortunately this would mean planting substantially more *Wx* F_2 seeds to have enough homozygous plants for statistical purposes after the heterozygous plants had been discarded.

If the experiments were to be repeated, a more complete series of translocation stocks would permit more accurate location of resistance genes. A more susceptible background than W23 used in the construction of the translocations would also be helpful. Plants should be inoculated at a more advanced growth stage and preferably in the controlled atmosphere of a greenhouse, in addition to field plots, to assess environmental effects. More precise chromosomal location of genes for partial resistance to NLB in Mo17 could be made using restriction fragment length polymorphism markers, which would also allow easy discrimination of heterozygotes; but the problems of environmental and development effects complicating the precise measurement of individual components of partial resistance remain.

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