Agronomie (1992) 12, 303-306

Plant improvement

Generation means analysis of resistance to head smut in maize

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(Received 10 October 1991; accepted 27 November 1991)

Summary — Head smut, caused by *Sphacelotheca reilana* (Kühn) Clint, is a disease affecting maize (*Zea mays* L) in southwest France. Generation means analysis was used to determine the relative importance of additive, dominance, and epistatic effects controlling resistance of maize to head smut. The parental inbred lines, F_1, F_2, and first backcross generations of A632×A188 and LH74×LMZ66 were tested for head smut resistance at La Réole, France in 1990 and 1991. Additive effects accounted for 97% of the variation among A632×A188 generations and 88% of the variation among LH74×LMZ66 generations. Dominance and epistatic effects were minor compared with additive effects. The absence of entries × years interaction indicated that multiple-year screening of germplasm is not necessary. This result, as well as the preponderance of additive genetic effects, should enhance progress in selection for head smut resistance.

***Zea mays* L = maize / *Sphacelotheca reilana* / resistance / generation means analysis**

Résumé — Analyse des moyennes de génération pour la résistance au charbon nu chez le maïs. *Le charbon nu, causé par* Sphacelotheca reilana *(Kühn) Clint, est une maladie qui affecte le maïs (*Zea mays *L) dans le Sud-Ouest de la France. L'analyse des moyennes de générations fut utilisée pour déterminer la relative importance des effets d'additivité, de dominance, et d'épistasie contrôlant la résistance au charbon nu chez le maïs. Les lignées pures parentales, la F_1, la F_2, et les premières générations de backcross de A632×A188 et LH74×LMZ66 furent testées pour la résistance au charbon nu à La Réole, France en 1990 et 1991. Les effets d'additivité expliquent 97% de la variation parmi les générations A632×A188 et 88% de la variation parmi les générations LH74×LMZ66. Les effets de dominance et d'épistasie furent mineurs par rapport aux effets d'additivité. L'absence d'interaction génotype×année indique que des tests du germplasm sur plusieurs années ne sont pas nécessaires. Ce résultat, ainsi que la prépondérance des effets génétiques d'additivité, devraient permettre des progrès de sélection pour la résistance au charbon nu.*

Zea mays *L = maïs* / Sphacelotheca reilana / *résistance* / *analyse des moyennes de génération*

INTRODUCTION

Head smut in maize (*Zea mays* L) is caused by *Sphacelotheca reilana* (Kühn) Clint. Infected maize plants are characterized by black masses of spores on tassels and/or ears. Among INRA research stations, the disease was first reported at Clermont-Ferrand in 1982, in the Adour valley in 1983, and in the Loire in 1984 (Petitprez, personal communication). Head smut is an increasing problem in the southwest region of France and affects both maize inbred lines in seed production areas and hybrids in farmers' fields.

Genetic resistance to head smut among maize lines and hybrids has been found. Stromberg *et al* (1984) and Whythe and Ge-

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vers (1988) found that resistance to head smut is controlled primarily by additive genetic effects, whereas Frederiksen (1977) reported partial to complete dominance of genes controlling resistance.

The objective of this study was to determine the importance of additive, dominance, and epistatic effects controlling head smut resistance in 2 crosses of maize.

MATERIALS AND METHODS

In preliminary experiments conducted from 1986 to 1989 at La Réole, France, the maize inbred lines A632 and LH74 were identified as resistant whereas A188 and LMZ66 were identified as susceptible to head smut. The crosses A632 (P_1)×A188 (P_2) and LH74 (P_1)×LMZ66 (P_2) were used for generation means analysis. The parental inbred lines (P_1 and P_2), F_1, F_2, first backcross to P_1 (BC_1), and first backcross to P_2 (BC_2) were evaluated at La Réole in 1990 and 1991. In each year, the 12 entries were evaluated in a randomized complete block design with 4 replications. The entries were grown in 2-row plots, each 3 m long and spaced 0.80 m apart, at a plant population density of 75 000 plants ha^{-1}.

Data on the percentage of plants infected with head smut were taken on each plot prior to harvest. Because the incidence of disease infection was a threshold character, the percentage data were transformed into units of standard deviation from the mean as suggested by Falconer (1981). Analysis of variance, combined across years, was performed on the transformed data. Years and replicates were considered random whereas all other effects were considered fixed.

Generation means analyses were performed for the A632×A188 and LH74×LMZ66 entries. The genetic model used was:

$$Y = m + \alpha a + \beta d + \alpha^2 aa + 2\alpha\beta ad + \beta^2 dd$$

where Y is the mean of a particular generation, m is the population mean, and α and β are appropriate coefficients for the following pooled genetic effects: additive (a), dominance (d), additive × additive (aa), additive×dominance (ad), and dominance × dominance (dd). The expectations of the means of each generation are (Hayman, 1958): $Y_{P1} = m+a-0.5d+aa-ad + 0.25dd$; $Y_{P2} = m-a-0.5d + aa + ad + 0.25dd$; $Y_{F1} = m+0.5d = 0.25dd$; $Y_{F2}=m$; $Y_{BC1} = m+0.5a + 0.25aa$; and $Y_{BC2} = m-0.5a+0.25aa$. The variances among means of the A632×A188 and LH74×LMZ66 generations were analyzed and sequentially fitted to models for *a, d, aa, ad* and *dd* gene effects using least squares regression analysis. Point estimates of *m, a, d, aa, ad* and *dd* were obtained by solving the system of equations (Gamble, 1962).

RESULTS

The amount of natural head smut infection was sufficient for germplasm screening purposes in both 1990 and 1991 (table I). In both years, more than 90% of the plants of the susceptible inbred lines (A188 and LMZ66) were infected, whereas less than 10% of the plants of the resistant lines (A632 and LH74) showed any sign of infection. Based on a least significant difference ($P=0.05$), the entries differed in their resistance to head smut. The average percentage of infection among generations was significantly greater in A632×A188 (60.3%) than in LH74×LMZ66 (25.7%) (table II).

No significant difference was found between the average percentage of infected plants in 1990 and 1991 (table II). The non significant blocks/years mean square indicated that blocking was not effective in controlling error variance. The entries × years interaction mean square and the mean squares of its orthogonal components (*ie,* A632×A188 entries × years, LH74 × LMZ66 entires × years, and A632 × A188 *versus* LH74 × LMZ66 × years) were not significant. Therefore, the sums of squares for error and entries × years interaction were pooled to obtain a pooled error mean square with 77 degrees of freedom (Carmer *et al,* 1969). This pooled error term was used for all *F*-tests.

Significant differences in amount of head smut infection were found among the parental

Table I. Average percentage of plants infected with head smut at La Réole, France in 1990 and 1991.

Genotype	*1990*	*1991*	*Mean*
A632	7.8	6.8	7.3
A188	92.8	100.0	96.4
A632 × A188	92.3	78.5	85.4
(A632 × A188) F_2	59.0	64.8	61.9
(A632 × A188) A632	14.0	24.0	19.0
(A632 × A188) A188	93.3	90.8	92.0
LH74	0.0	0.8	0.4
LMZ66	97.5	99.3	98.4
LH74 × LMZ66	8.0	3.8	5.9
(LH74 × LMZ66) F_2	5.0	11.5	8.3
(LH74 × LMZ66) LH74	2.3	4.0	3.1
(LH74 × LMZ66) LMZ66	31.0	44.8	37.9
		$LSD_{0.05}$	21.0

inbred lines, F_1, F_2, and backcrosses for both the A632 × A188 and LH74 × LMZ66 sets of entries (table II). For the A632 × A188 generations, additive genetic effects were significant and accounted for 97% of the variation among genotypes; dominance and epistatic effects were not detected. For the LH74 × LMZ66 generations, additive genetic effects were significant and accounted for 88% of the variation among genotypes. Dominance effects were significant and accounted for 9% of the variation. The estimate of pooled dominance effects among the LH74 × LMZ66 generations was positive and smaller than the estimate of pooled additive effects (table III). The negative estimates of pooled additive × additive and additive × dominance epistatic effects were also significant. Epistasis accounted for only 3% of the variation among LH74 × LMZ66 generations (table II).

Table II. Generation means analysis of head smut resistance[a].

Source of variation	*df*	*Mean square*	R^2
Years	1	0.33	
Blocks/years	6	0.54	
Entries	11	37.99*	
A632 × A188 entries	(5)	25.50*	
Additive	(1)	123.54*	0.97
Dominance	(1)	0.67	0.01
Additive × additive	(1)	2.59	0.02
Additive × dominance	(1)	0.62	0.00
Dominance × dominance	(1)	0.10	0.00
LH74 × LMZ66 entries	(5)	43.72*	
Additive	(1)	191.63*	0.88
Dominance	(1)	19.32*	0.09
Additive × additive	(1)	3.00*	0.01
Additive × dominance	(1)	4.67*	0.02
Dominance × dominance	(1)	0.03	0.00
A632 × A188 *vs* LH74 × LMZ66	(1)	71.79*	
Entries × years	11	0.97	
A632 × A188 entries × years	(5)	1.41	
LH74 × LMZ66 entries × years	(5)	0.62	
A632 × A188 *vs* LH74 × LMZ66 × years	(1)	0.56	
Error	66	0.64	

*Significant at $P = 0.05$. For F-tests, pooled error mean square = 0.69 with 77 df. [a]Analysis based on level of infection expressed as units of standard deviation from the mean.

DISCUSSION

Results suggested that genes controlling resistance to head smut in the A632 × A188 and LH74 × LMZ66 sets of generations were different. The absence of dominance and epistatic effects indicated that head smut resistance was under additive genetic control in the A632 × A188 genotypes. In contrast, dominance and epistatic effects were detected among the LH74 × LMZ66 genotypes. The positive and relatively small (compared with pooled additive effects) estimate of pooled dominance effects suggested partial dominance of resistance to head smut among LH74 × LMZ66 generations. Partial dominance of resistance was counteracted by negative epistatic effects. However, these dominance and epistatic effects were minor compared with additive genetic effects. The preponderance of additive genetic effects indicated that resistance of hybrids to head smut can be predicted from parental data. If a breeder has 100 single-cross hybrids, obtained from crossing 10 lines of one heterotic group to 10 lines of a complementary heterotic group, all 100 single crosses need not be screened for head smut resistance. Rather, only the 20 parental inbreds need to be screened for head smut resistance, and the resistance of single crosses can be predicted as the mean of the parental values. The preponderance of additive genetic effects also suggested that recurrent selection for head smut resistance should be effective using intra-population improvement schemes, such as recurrent mass, half-sib, or selfed progeny selection (Miles *et al*, 1980).

Table III. Estimates of genetic effects for the A632 × A188 and LH74 × LMZ66 generations[a].

Cross	m	a	d	aa	ad	dd
A632 × A188	−0.31*	2.84*	−1.30	−0.79	0.44	−0.82
LH74 × LMZ66	1.66*	2.13*	0.58*	−1.09*	−1.21*	0.47

*Significant at $P = 0.05$. [a]Based on level of infection expressed as units of standard deviation from the mean. Pooled genetic effects are as follows: additive (*a*), dominance (*d*), additive × additive (*aa*), additive × dominance (*ad*), and dominance × dominance (*dd*).

The absence of entries × years interaction effects indicated that the relative levels of head smut resistance of parental lines and crosses remained consistent each year. Thus, multiple-year screening of lines and hybrids is not necessary if the level of head smut infection in a given year is sufficient for screening purposes.

In conclusion, resistance to head smut in the maize crosses studied was controlled primarily by additive genetic effects. Resistance of hybrids can be predicted from parental data obtained from a single year. These minimal screening requirements as well as the additive mode of inheritance should enhance progress from selection for head smut resistance.

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