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DETERMINATION OF GENE-CHROMOSOME ASSOCIATION FOR MILDEW RESISTANCE TO RACE 2 OF *AVENA SATIVA* L.

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Abstract

F_2 seedlings of monosomics I-XV of *A. sativa* cv. Manod x A. sativa cv. Mostyn (S. 242) and 41-chromosome I-XV of *A. sativa* cv. Manod x *A. sativa* cv. Cc 7718 were investigated for segregation of resistance to race 2 of mildew (*Erysiphe graminis* s. sp. *avenae*). There was no significant deviation from 3 resistant: 1 susceptible in a number of lines while a few lines showed significant deviation from 3 resistant: 1 susceptible but the deviation was due to higher number of susceptible plants. It seems that the gene controlling resistance to race 2 of mildew is not located atleast on any one of the monosomes so far available.

Introduction

Locating genes on specific chromosomes is made possible by using the mono- or trisomic lines (Thomas, 1976). The present study was undertaken to determine the association of gene responsible for resistance against the attack of the pathogen *Erysiphe graminis* s. sp. *avenae* (race 2) with one or more chromosome lines (1-XV) of *Avena sativa.*

Materials and Methods

The hexaploid cultivars of *Avena sativa*, employed in the present study were Manod, Mostyn (S. 242) and Cc 7718. The cultivar Manod was bred at the Welsh Plant Breeding Station, Aberystwyth, U.K. originated from *A. sativa* var. Sun II that in turn was produced from a cross between Star and Eagle varieties of this species at Svalof, Sweden and introduced into Great Britain in 1946. *Avena sativa cv. Mostyn (S. 242)* was also bred at the same station and released in 1968. It resulted from a complex cross involving *A. sterilis* var *ludoviciana* (the wild oat which contributed to mildew resistance to race 1, 2, 3 of mildew *E. graminis s. sp. avenae* (Hayes, 1970). *A. sativa cv. Cc 7718* involves Cc 4146 (*A. sativa* x *A. ludoviciana*) which provides resistance to race 1, 2 and 4 of mildew (Hayes, 1968).

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TABLE 1. Segregation for resistance to race 2 of mildew in the F_2 families from crosses of different monosomic lines of Sun II (susceptible) with the cultivars Mostyn/Cc 7718 (resistant).

Monosomic line	Monosomics X Mostyn				Monosomics X Cc 7718			
	Total plants	Resistant	Susceptible	χ^2 for 3 : 1	Total plants	Resistant	Susceptible	χ^2 for 3 : 1
I	57	41	16	0.29	49	38	11	0.17
II	60	35	25	8.89**	–	–	–	–
III	61	38	23	5.25*	50	37	13	0.03
IV	51	28	23	10.99***	53	34	19	3.33
V	61	35	26	10.10**	50	35	15	0.67
VI	58	34	24	8.30**	54	35	19	2.99
VII	52	36	16	0.92	63	36	27	10.71**
VIII	57	41	16	0.35	58	42	16	0.21
IX	61	47	14	0.41	61	45	16	0.05
X	60	32	28	15.02***	54	35	19	2.99
XI	44	20	24	20.48***	75	43	32	12.48***
XII	48	31	17	2.78	57	33	24	8.89**
XIII	–	–	–	–	56	27	29	21.43***
XIV	61	28	33	27.55***	59	22	37	44.75**
XV	61	22	29	49.32***	49	22	27	23.68**
Control	20	20	0	–	45	45	0	–

***Significant at 0.1% lavel

**Significant at 1% level

*Singnificant at 5% level

F_1 seeds of *A. sativa* cv. Manod (2n – 1 = 41) X *A. sativa* cv. Mostyn (S. 242) and *A. sativa* cv. Manod X *A. sativa* Cc 7718 (2n = 42) were obtained from Dr. H. Thomas of Welsh Plant Breeding Station Aberystwyth, U.K. Tests for mildew reaction of F_2 seedlings (derived from 2n = 41 F_1 plants were undertaken in a spore-proof green house. The seedlings were dusted at the 2 leaf stage with spores of mildew (race 2) from heavily infected spreader plants. The infected seedlings were scored after two weeks.

Results and Discussion

The data for the segregation of resistance to race 2 of mildew *E. graminis* s. sp. *avenae* in crosses between the monosomic I-XV of *A. sativa* cv. Manod and *A. sativa* cv. Mostyn (S. 242)/ *A. sativa* cv. Manod X *A. sativa* cv. Cc 7718 are presented in Table 1. Six lines out of fifteen lines showed significant deviation from 3 resistant: 1 susceptible but these are primarily the results of higher number of susceptible rather than resistant plants in F_2 families. It is therefore inferred that the gene controlling the resistance to mildew (race 2) is not located on any one of the monosomes respectively involved in the monosomic lines employed.

The common cultivated hexaploid oats that are susceptible to mildew could be considerably improved by selecting and breeding the cultivars/varieties with high or complete resistance to mildew genotypes of the wild oat in which the complete resistance have been known for a long time. Such wild species are *A. barbata* (4x = 28), A. *prostrata* (2n = 14) and *A. ventricosa* (2n = 14). The chromosomes of *A. barbata* bearing gene conferring resistance to pathogen has been successfully transferred to *A. sativa* by making addition/substitution lines (Jones & Aung, 1976; Aung & Thomas, 1976; 1978; Aung *et al,* 1977), or by inducing the chromosome of *A. barbata present in the telocentric line* of *A. sativa* to pair with 'sativa' chromosome in the presence of *A. longiglumis* (Cw 57) and then backcrossing with *A. sativa* for a couple of generations which resulted in lines that possessed 42-chromosomes and it was resistant to mildew but did not have 'barbata' chromosome (Powell & Thomas, 1979). The source of the gene responsible for resistance to mildew (race 2), *E. graminis* s. sp. *avenae* clearly confirm our finding that *A. sativa* lack such gene and consequently the known and available monosomics I-XV, that have *A. sativa* as their genetic background do not have gene which confers resistance against pathogen *E. graminis* s. sp. *avenae* (race 2) so that the gene conferring resistance to this race is not associated with any one of the monosome involved in the I-XV available monosomic lines.

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