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IDENTIFICATION OF CHROMOSOMES CARRYING GENES FOR RESISTANCE TO LOOSE SMUT OF BREAD WHEAT (*TRITICUM AESTIVUM* L.) IN INDIA

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ABSTRACT

The breadwheat variety Cadet and all its monosomic lines (except mono 3A) were inoculated with a mixture of loose smut chlamydospores. The inoculated seed was grown during normal (rabi) and off-season (summer) conditions. Cadet was found to be resistant to loose smut. Resistance was governed by at least three genetic factors located on chromosomes 1B, 3D and 7D. The gene on chromosome 3D was major. Genes on chromosomes 1 B and 7D were modifiers whose hemizygous ineffectiveness resulted in slight weakening of the resistance of the variety (only one late tiller of these susceptible plants developed the disease). While the gene on 1B was effective during both seasons, the effect of the one on 7D was not discernible during the normal season.

Key words: Monosomic analysis, loose smut, *Triticum aestivum*.

Loose smut of wheat, caused by *Ustilago nuda tritici* (Jens) Schaff., is an important disease in the main wheat belt of India. Floral infection is the rule. The mycelium remains dormant in the first season after infection. In the next season, it grows systemically and expresses itself at ear development. Usually all the spikelets in an ear are converted into a mass of spores resulting in total loss of grain. As all or most of the tillers of a plant are affected, even a small percentage of disease incidence causes considerable damage. Affected plants become a source of infection for healthy plants in the population.

It has been reported that in India resistance to this disease is governed by one dominant gene [1, 2] or by duplicate dominant genes [3]. As breadwheat is segmentally allohexaploid, a better understanding of inheritance can be obtained by aneuploid analysis. This publication contains information obtained by using Cadet monosomic lines.

MATERIALS AND METHODS

The breadwheat variety Cadet was found resistant to loose smut. Cadet and its twenty monosomic lines (excluding mono 3A) comprised the material for this study.

As pathogenic races of loose smut are not established [4], a composite inoculum collected from the northern parts of the country was used for studies with this smut.

Confirmed monosomic plants were inoculated with chlamydospores as described earlier [2] with the precaution that a minim of spore suspension was introduced into each floret with a hypodermic needle so as to avoid drenching the florets and ensure better seed set.

Part of the seed from the inoculated monosomic plants was grown in the following off-season (summer) and part in the normal rabi season next year at IARI Regional Station, Bhowali. The resistant and susceptible plants in each monosomic line were recorded individually (Table 1).

RESULTS AND DISCUSSION

As the variety Cadet is resistant to the smut, susceptibility was expected to manifest itself in the monosomic plants if resistance was ineffective in hemizygous or nullisomic conditions. Monosomic plants may range from 60–85% of the progeny derived from a monosomic plant [5, 6].

From the data presented in Table 1, it is clear that the variety Cadet is resistant to loose smut and lost its resistance in hemizygous condition of chromosome 1B, 3D and 7D. Obviously, the genes for loose smut resistance were located on these chromosomes. However, these resistance genes had different expressivity and penetrance in different monosomic populations.

In the population of mono 3D, the susceptible plants gave full expression of the disease (i.e. almost all the tillers were smutted with profuse sporulation) in both rabi and summer seasons. A modified reaction was observed in mono 1B and mono 7D. In these two populations the ear of only one late tiller of the susceptible plants developed the disease while all other tillers had normal ears. The smutted ear was small with visibly poor sporulation. The progenies of monosomics 1B and 7D gave similar reactions. However, mono 1B expressed the reaction in both the seasons whereas the effect of mono 7D was expressed only in summer. Individually the gene(s) on chromosome 1B and 7D in hemizygous condition had minor effect on the reduction of resistance of the variety.

The segregation pattern for resistance and susceptibility in the three critical monosomic populations showed that 3D segregated a high frequency of susceptible plants, i.e. 81% (111

Table 1. Loose smut incidence in inoculated monosomic lines of wheat variety Cadet

Variety or monosomic	Loose smut incidence						Total plants screened
	summer (off-season)			winter (rabi)			
	R	S	total	R	S	total	
Cadet	25	0	25	44	0	44	69
1A	43	0	43	65	0	65	108
1B*	55	10	65	73	15	88	153
1D	53	0	53	73	0	73	126
2A	51	0	51	72	0	72	123
2B	67	0	67	75	0	75	142
2D	39	0	39	80	0	80	119
3B	50	0	50	60	0	60	110
3D*	11	47	58	15	64	79	137
4A	33	0	33	37	0	37	70
4B	49	0	49	63	0	63	112
4D	71	0	71	85	0	85	156
5A	45	0	45	57	0	57	102
5B	51	0	51	77	0	77	128
5D	79	0	79	88	0	88	167
6A	35	0	35	47	0	47	82
6B	69	0	69	81	0	81	150
6D	55	0	55	75	0	75	130
7A	53	0	53	80	0	80	133
7B	63	0	63	85	0	85	148
7D*	37	9	46	50	0	50	96

*Critical line: R — symptomless; S — susceptible, susceptibility in mono 1B and 7D involved only one late tiller with sparse sporulation.

susceptible out of 137). The frequency of susceptible plants did not vary in the two diverse seasons (47 susceptible out of 58, i.e. 81% during rabi season; 64 susceptible out of 79, i.e. 81% during summer). This corresponds to the expected frequency of monosomic plants (60–85%), suggesting that all the monosomic plants were susceptible and disomic plants resistant. The chromosome numbers of a few resistant plants were checked and confirmed that they were disomes. In the case of 1B, the percentage of susceptible plants was very low but comparable in the two seasons (10 susceptible out of 65 (15.4%) during normal season; 15 susceptible out of 88 (17%) during summer). The mono 7D population also exhibited a

low frequency of susceptible plants: 9 susceptible out of 46 (19.6%) only during summer, but remained free from disease (all the 50 plants healthy) in rabi.

The results of this aneuploid analysis and conventional genetic analysis referred to above [1–3] confirm that loose smut resistance in wheat is governed by one major gene on chromosome 3D. However, the two minor genes on chromosomes 1B and 7D, as revealed by monosomic analysis, could not be detected by conventional genetics. As stated earlier these minor genes have very restricted effect on expression and incidence of the disease. Therefore, the modified reaction could be responsible for the smaller fraction of susceptible plants in the segregating population of the 1B and 7D monosomic plants.

The type of disease reaction described for mono 1B population is characteristic of another known variety, namely, Maria Escobar. It was observed earlier that 6 out of 86 plants (7.0%) of this variety exhibited disease only in one late tiller. In this context it may be inferred that varieties like Maria Escobar are resistant but the expression of resistance is dependent on the action of the modifier gene(s). Further this type of reaction may or may not appear in some seasons.

In all our genetic studies, conducted so far, the inoculum used was a composite mixture (collected from farmers' fields in North India). In spite of the fact that the inoculum used was a mixture, only one major gene has been detected for loose smut resistance. This suggests that either only a single race was present in the inoculum or all the racial components were avirulent for the gene(s) in Cadet. Nielsen [7] studied an Indian sample of smut and found it similar to the T_1 race of smut from Canada which carries a recessive virulence gene Utv_1 [8]. Further studies [9] indicate that Utv_1 is a complex locus — it is inherited as a unit and causes virulence on three differentials (Renfrew, Florence x Aurora, and Red Bobs) although there are races that cause virulence on only one of these differentials but not in others.

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