GENETIC ANALYSIS OF RESISTANCE TO WHITE RUST IN INDIAN MUSTARD

D.Singh, H.Singh

Department of Plant Breeding, Haryana Agricultural University, Hisar-125004, India

Abstract

The present study on genetic analysis of resistance to white rust /Albugo candida/ an important but less understood disease of Indian Mustard /Brassica juncea L.Czern and Coss/ was carried out in 80 progenies developed through North Carolina mating design II /N.C.II/ in F3 population of a multiple cross [/RH30xDomo/xRIK78-6xRH30/x/RIK78-6xPrakasn/x/VarunaxTM2/]. The component parents of this cross represented early and late maturing genotypes of Indian and exotic origin.Parent Domo, RIK 78-6 and TM 2 possessed moderately to high degree of resistance whereas other parental lines were susceptible. From the genetic analysis of resistance to this disease it was intersting to note that the range of variability was considerably high, /0.67 to 69.07 per cent/. The values of additive genetic variance /δ_A^2/ and Dominance Variance /δ_D^2/ were significant. However, the magnitude of additive genetic variance was considerably higher than non-additive. The average degree of dominance indicated over dominance /$\bar{a}$=1.27/. The narrow sense heritability was 52.16 per cent. The expected genetic gain through full-sib family selection was 33.75 per cent and hence it could be possible to evolve high yielding and white rust resistance genotype /s/ from above base material.

Introduction

White Rust caused by Albugo candida (.Kunze is an endemic disease of Indian Mustard /Brassica juncea L.Czern and Coss/ in India. It affects many species of cruciferae.

The most common hosts are rapeseed-mustard, cabbage, cauliflower, radish horse, radish and sheperds /Petric 1973 , Platfor'd and Bernier 1976/. This disease produces two types of infection , local and systemic. Raised, white many isolated pastules on leaves and stem show local infection. In case of systemic infection, the fungus becomes systemic in host tissues and stimulates hypertrophy and hyperplasia which results in enlarged and distorted organs of flower. The normal seed development is prevented, resulting in major reduction in quantity and quality of seeds. This disease in recent years is among the chief factors responsible for the fluctuation in the seed yield over the years. There are varietal differences for disease /1986, Anonymous/. However, the literature is almost lacking for the genetic control of resistance to white rust. Therefore, present study was undertaken to find out the genetic control of resistance to this disease as an aid for evolving high yielding and white rust resistant genotypes in Indian Mustard.

Materials and methods

To develop the base material, the parents for white rust resistance were identified and the single crosses were made between high yielding susceptible parent x poor yielding resistant parent. From two single crosses the double crosses were made and subsequently a multiple cross consisting of 7 parents [/RH30xDomo/x/RIK78-6xRH30/x /RIK78-6xPrakash/x/VarunaxTM2/] was developed. The compoonent parent Domo, RIK 78-6 and TM 2 posses resistance to white rust. The other component parent RH 30, Prakash and Varuna are the high yielding cultivars of Indian Mustard but susceptible to white rust. The multiple cross was advanced to F3 population. In F3 population, one set of 4 females and 4 males were selected at random and each male with each female was crossed in all possible combinations. Such five sets, consisting of 80 biparental crosses were developed as per North Carolina mating design II /N.C.II/. The progenies of biparental crosses were grown in a compact

family block design with three replications. Each biparental cross progeny was represented with a single row of 5m length. Plants within r were spaced at 15 cm apart after 21 days of sowing. For adequate disease pressure the experiment was planted 20 days later /5th Nov./ than normal sowing time /15 Oct./. Artificial innoculation was done by crushing the infected leaves in sterilized distilled water and spraying of innoculum suspension over different biparental progenies. The observations were recorded on 50 leaves collected from different portions of plants in each progeny. The per cent disease was calculated according to formula of Gemawat and Prasad /1969/.

$$\text{Percent disease intensity} = \frac{\text{sum of all numerical rating}}{\text{total number of leaves x highest grading}} \times 100$$

The data per cent disease intensity was transformed by Angular transformation. The transformed data was analyzed as per N.C.II /Comstock and Robinson, 1952/.

Results and discussion

The analysis of variance for N.C.II for resistance to white rust in the biparental progenies presented in Table 1 indicated that the mean squares due to males were significant whereas the females in set were not significant. This revealed that males used had considerable variability for white rust whereas the non-significance of females in sets may be because of sampling error. The interaction in sets was highly significant indicating that considerable genetic variability was generated by biparental crosses for resistance to white rust.

Mean squares obtained from analysis of variance for N. C. II design was used to estimate the variance among males plus females and variance due to male x female interaction. Since males and females were equal in present case these variances were further utilized to estimate additive genetic variance /σ_A^2/ and dominance variance /σ_D^2/. These estimates are presented in Table 2.

The estimates of variance component /$\sigma_m^2 = \sigma_f^2$/ for white rust were higher than the variance due to

male x female interaction /σ^2_{mf}/. Thus, it is evident that the considerable genetic variation was present in the biparental progenies as well as in males and females used for developing biparental progenies. The estimates of additive /σ_A^2/ and dominance /σ_D^2/ revealed that the magnitude of additive genetic variance was higher than dominance /σ_D^2/. The dominance ratio /σ_D^2/σ_A^2 = 0.81/ also indicated the same. The estimates of average degree of dominance /$\bar{a}$=1.27/ indicated over dominance. This indicates the importance of both additive as well as dominance component of variance in control of resistance to white rust. The considerable high value of heritability and expected gain through fullsib family selection was observed for resistance to white rust. The selection for resistance to white rust in the intermated population rather than in F2 and F3 population is advocated because of the fact that biparental matings in F2 and advanced generation of intervarietal hybrids would obviate the harmful effects of liqkages and linkage disequilibirium and shuffle the desirable genes in one recombinant /Comstock and Robinson, 1952; Gates et.al., 1957 and Matzinger and Cockerham,1963/. The fullsib family selection would be the right choice looking into the kind of genetic variation obtained and the kind of material used in the present study.

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Table 1. Analysis of variance of N.C. II for resistance to white rust /Albugo candida/ in Indian Mustard.

Source of variation	d.f.	Mean squares
Sets	4	1394.988^{x}
Replication in sets	10	5.200^{x}
Females in sets	15	164.583
Males in sets	15	1296.497^{x}
Females x Males interaction in sets	45	297.247^{x}
Error	150	1.957

^{x} Significant at p = 0.05

Table 2. Estimates of genetic components, dominance ratio, average degree of dominance, heritability and expected genetic gain for fullsib family selection for resistance to white rust /Albugo candida/ in Indian Mustard.

Genetic components	Estimates
Additive genetic variance /σ_A^2/	487.026^{x} $\pm$ 152.19
Dominance variance /σ_D^2/	393.720^{x} $\pm$ 81.757
Dominance ratio /σ_D^2/σ_A^2/	0.81
Average degree of dominance $\bar{a} = \sqrt{2_D^{o-2}/\sigma_A^2}$	1.27
Haritability h^2	52.17
Expected genetic gain through full-sib family selection /Δ F/	33.75

^{x} Significant at p = 0.05