GENETIC ANALYSIS OF WHITE RUST *(ALBUGO CANDIDA)* RESISTANCE AND ISOLATION OF RESISTANT HIGH YIELDING GENOTYPES OF MUSTARD *(BRASSICA JUNCEA)*

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GENETIC ANALYSIS OF WHITE RUST *(ALBUGO CANDIDA)* RESISTANCE AND ISOLATION OF RESISTANT HIGH YIELDING GENOTYPES OF MUSTARD *(BRASSICA JUNCEA)*

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CERTIFICATE

This is to certify that the thesis entitled **GENETIC ANALYSIS OF WHITE RUST *(ALBUGO CANDIDA)* RESISTANCE AND ISOLATION OF RESISTANT HIGH YIELDING GENOTYPES OF MUSTARD *(BRASSICA JUNCEA)*** submitted to the Faculty of Post-Graduate School, Indian Agricultural Research Institute, New Delhi, in partial fulfilment of the requirements, for the award of the degree of **Doctor of Philosophy in Genetics, by Mr. Sanjeev Kumar Chauhan** embodies the result of *bona-fide* research work carried out by him under my guidance and supervision. No part of the thesis has been submitted by him for any other degree or diploma.

I further certify that such help or information, as have been availed of in this thesis, is duly acknowledged.

Date: October 16th, 1998

(R.N.RAUT)
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INTRODUCTION

Indian mustard, *Brassica juncea* L. Czern & Coss is India's second most important source of oilseeds and edible oil. Rape-seed mustard in India includes *B. campestris* (toria, brown sarson, yellow sarson), *B. juncea*, *B. carinata* and *B.napus* (gobhi sarson) but *B. juncea* occupies over 80% of the area and contributes 85% of the production. During the past three decades area, production and productivity of rapeseed-mustard has been expanding at a rapid rate largely in response to the continuing increase in demand for edible oil. The figures available for area, production and productivity for 1989-90 and 1996-97 respectively are 4.97 to 6.44 million hectares area, 4.13 to 6.90 million tonnes production and 839 to 1002 kg/ ha productivity (Kumar, 1998). In terms of area Rajasthan, Uttar Pradesh, Madhya Pradesh and Haryana are the four states accounting for more than 80% of the area with 5.54 million hectares out of a total of 6.85 million hectares during 1996-97. With increasing concern for higher production and productivity farmers in the above mentioned states have adopted intensive cultivation practices and frequently irrigate the crop. Mustard was traditionally a crop of marginal land, with the expansion and intensification of *Brassica* oilseed cultivation, pest and disease has become a constraint to profitable production.

Brassica oilseeds are attacked by many diseases, of which white rust [*Albugo candida* (Pers. ex Hook) Kuntze] and alternaria black spot (*Alternaria spp.*) are among the most widespread and difficult to control under Indian conditions. White rust of mustard is found nearly wherever mustard is grown, and most regularly causing extensive damage in recent

years due to congenial agro-climatic conditions. Most of the available cultivars of mustard are susceptible to white rust and the reported loss varies from 17 to 34 per cent (Kolte, 1985; Bisht , 1994) depending on the intensity of staghead formation. White rust causes loss in seed yield of Indian mustard upto the extent of 50% under late sown condition (Saharan *et al.*, 1984). Genetical resistance is the most economical and preferable method of reducing yield losses due to white rust. Genetic resistance can be most effectively utilized by knowledge of the identity of resistant genes, their mode of inheritance and stability under differing agro-climatic conditions. In view of its importance Brassica scientists all over the world have attempted to understand the genetics of white rust resistance in *B. juncea* (Thukral and Singh, 1986; Singh and Singh, 1987; Tiwari *et al.*, 1988; Yashpal *et al.*, 1991; Gadewadikar *et al.*, 1993; Paladhi *et al.*, 1993 and Yadav *et al.*, 1994), in *B. napus* (Fan *et al.*, 1983; Verma and Bhowmik, 1989; Liu and Rimmer, 1991; Ferriera *et al.*, 1995; Liu *et al.*, 1996), in *B. nigra* (Delwiche and Williams, 1977), in *B. rapa* (Pound and Williams, 1963; Cole *et al.*, 1996) and in interspecific crosses (Singh and Singh, 1988; Subudhi and Raut, 1994; Rao and Raut, 1994).

Conventional breeding has been effective in incorporating resistance to *B. juncea* and several resistant strains have been developed but so far no commercial cultivar is available in India having absolute resistance to white rust. Subudhi and Raut (1994), Rao and Raut (1994) and Vasanthi (1993) succeeded in incorporating white rust resistance from *B. napus* and *B. carinata* to *B. juncea*. Their mode of inheritance was studied by Sridhar (1996) and was found to be variable giving segregation ratios of 3R:1S or 1R:3S in F_2 population depending on the source of white rust resistance gene. Eventhough resistant gene/genes are now available in *B. juncea*, it is necessary to understand their mode of inheritance and also to combine resistance with yield and yield contributing characters for commercial

utilization as varieties. Therefore, the present studies were planned with following objectives:

1. Genetic analysis of white rust resistance derived from different sources. These were derived from intra- and interspecific crosses and now stable in *B. juncea*.

2. Variation in expression of resistance to white rust under different agro-climatic conditions and different genetic backgrounds.

3. Association of white rust resistance with leaf waxiness and yellow seed colour.

4. Estimation of gene effects for different yield component characters of selected crosses by use of generation mean analysis.

5. Identification of crosses/genotypes combining resistance to white rust and superior yield components.

REVIEW OF LITERATURE

The literature reviewed here includes relevant work done on usefulness of interspecific variability, disease resistance, leaf waxiness and seed colour studies in *Brassica.* Quantitative traits inheritance is reviewed only briefly.

Brassica is an agriculturally important genus containing over 30 cultivated and wild species with highly diverse morphology and wide ranging utility. In view of their cytogenetical relationship crosses have been attempted and characters transferred across species (Prakash and Hinata, 1980).

2.1 Usefulness of interspecific variability

Interspecific hybridization has an important role in plant breeding since it enables to recombine useful genes of the parental species which are not normally available within the limits of each species. Crosses involving the current cultivars of crop *Brassica* are more likely to produce desirable progenies than crosses involving wild species (Pawlowski, 1970). The usefulness of interspecific crossing however, depends on some of the progeny being fertile. Nonhomologous recombination between chromosomes of different genomes in interspecific or intergeneric hybrids is considered a good way to generate variability and transfer of desirable economic characters (Namai, 1978; Narain, 1979).

A large number of economically useful characters have been transferred through interspecific or intergeneric barriers. These include introgression of earliness from A genome of *B. juncea* to *B. napus* (Zaman, 1989), transfer of disease resistance traits such as Blackleg resistance from *B. juncea* to *B. napus* (Roy, 1984; Sacristan and Gerdemann, 1986). The results of interspecific hybridization between *B. juncea* and *B. carinata* envisaged the easy transfer of white rust resistance into high seed yield background through pedigree selection from an unadapted species, *B. carinata* to the adapted species, *B. juncea* (Singh and Singh, 1987; Singh *et al.*, 1988). The resistance, which is monogenic, with complete dominance in *B. carinata*, could be partially introgressed into *B. juncea* cultivars by selecting disease-free plants in advanced-segregating generations grown under heavy disease pressure, and their repeated backcrossing to *B. juncea* cultivars (Singh and Singh, 1988). White rust resistance from *B. napus* to *B. juncea* was transferred through interspecific hybridization of a synthetic line of *B. napus*, 'Indian Synthetic Napus 706' identified for resistance to white rust under artificial inoculation and widely grown variety of *B. juncea*, Varuna, susceptible to white rust by Rao and Raut (1994). All the F_1 were resistant indicating resistance carried by ISN 706 is expressed as a dominant phenotype and controlled by nuclear genes, segregation ratio in F_2 was 13R:3S. Progenies from the backcrosses to susceptible parent segregated in the ratio of 1R:1S and backcrosses to resistant parent gave all resistant plants. These ratios indicated the presence of digenic inheritance with dominant and recessive gene action. Subudhi and Raut (1994) also reported that white rust resistance was under digenic control in the studies involving F_1 and F_2 progenies of interspecific crosses between *B. juncea* (Pusa Barani) as female parent and 3 strains of *B. napus* (N368, N576 and N606) as male parent. Hybrids were having intermediate morphology but in the F_2 generation, segregation into types similar to either parent was observed in varying frequencies. Transfer of genes have been

reported for high linoleic acid from *B. juncea* to *B. napus* (Roy and Tarr, 1985), shattering resistance from *B. juncea* to *B. napus* (Prakash and Chopra, 1988), beet cyst nematode resistance to *B. napus* from related species (Lelivelt *et al.*, 1988), leaf waxiness from *B. napus* to *B. juncea* (Subudhi and Raut, 1994). From interspecific crossing of *B. juncea* x *B. napus*, high yielding rape varieties and also varieties with superior fatty acid composition were developed in USSR (Shpota and Podkolzina, 1980).

2.2 White rust resistance

Yield of *B. juncea* is highly affected by the diseases of this crop, mainly by white rust and *Alternaria* blight. These are two major diseases of mustard crop in India. white rust is caused by the Oomycetes fungus *Albugo candida* (Pers. Ex.Lev.) Kuntze, which produces localised white pustules on the lower surface of the leaves and other organs of the infected plants. Staghead (malformed inflorescence) may appear late in the growing season due to white rust and/or mixed infection of white rust and downy mildew [*Peronospora parasitica*]. White rust is reported to cause 17-34 per cent yield loss particularly due to the staghead phase (Bains and Jhooty, 1979; Kolte, 1985). This disease causes appreciable loss in seed yield of Indian mustard upto the extent of 50% under late sown conditions (Saharan *et al.*, 1984). The loss in oil in seed of infected plants has been found to range from 14.58 to 25.97 per cent (Ansari *et al.*, 1988). *B. carinata* and *B. napus* were found to be immune to white rust in contrast to susceptible *B. juncea* (Singh and Singh, 1987; Lakra, 1988).

The causal organism of white rust has a wide host range. The occurence of a large number of biological forms of *A. candida* was reported during the early half of this century (Hiura, 1930; Napper, 1933). To date atleast 10 physiologic "races' of *A. candida* have been identified and classified based on specificity to different crucifer species (Hill *et al.*, 1988;

Pound and Williams, 1963). However, races of *A. candida* do not exhibit an absolute adaptation to a particular host species, because most races also can infect some genotypes of related *Brassica* species (heterologous hosts), especially those sharing genomes with the *Brassica* species from which they were originally collected (homologous hosts). Several studies have suggested that *Brassica-Albugo* specificity occurs at the levels of genus, species within a genus (Delwiche and Williams, 1977; Hill *et al.*, 1988; Petrie, 1988; Pidskalny and Rimmer, 1985; Pound and Williams, 1963; Verma *et al.*, 1975), and cultivars within a species (S.R. Rimmer, unpublished data). The identified races of *A. candida* for different crucifers are race 1 on *Raphanus sativus* L., race 2 on *B. juncea*, race 3 on *Armoracia-rusticana*, race 4 on *Capsella-bursa-pastoris*, race 5 on *Sisymbrium officinale*, race 6 on *Rorippa islandica*, race 7 on *B. rapa* and race 8 on *B. nigra*. The existence of distinct races of *A. candida* was also observed by Singh and Bhardwaj (1984) and Lakra and Saharan (1988) on the basis of reaction of different isolates on sets of host differentials. Therefore, understanding the mode of inheritance for disease resistance is an important pre-requisite before attempting to breed resistant varieties.

Williams and Pound (1963) screened a large number of accessions and commercial varieties of radish at cotyledonary stage for resistance to *A. candida* race 1 and found that atleast 30 accessions and 2 varieties showed 20-100% resistance. Variety Chine Rose Winter (CRW) and Round Black Spanish (RBS) both displayed a monogenic dominant resistance. Resistance in CRW was manifested as hypersensitive reaction influenced by minor genes whereas RBS did not show any such visible disease expression.

Pound and Williams (1963) while studying the biological races of *A. candida* reported that the nature of white rust resistance in *B. oleracea* and *B. campestris* was of dominant and both monogenic and multigenic types.

Delwiche and Williams (1977) identified five recessive marker genes, cream coloured pollen (CPO), yellow-green plant (Ygl), yellow green cotyledon (Ygcl), lutescent plant (lu) and glabrous (gl) and dominant marker gene Ac7 for resistance to *Albugo candida* race 7 in *Brassica nigra* (L.) Koch.

Two Chinese lines of *B. napus* susceptible to race 7 of *A. candida*, GCL and 2282-9 and one Canadian resistant cultivar 'Regent' were crossed by Fan *et al.* (1983) to observe the inheritance pattern of resistance. They found that resistance to white rust races was conditioned by 3 independent dominant genes designated as Ac 7-1, Ac 7-2 and Ac 7-3. However, different F_2 and backcross ratios showed that the 'Regent' population was heterogeneous for resistance. They found that all 'Regent' plants were homozygous for resistance at two loci and some possibly carried resistance at third locus.

Thukral and Singh (1986) studied the inheritance of resistance in *B. juncea* to *A. candida* in the crosses EC 12749 x Prakash and EC 12749 x Varuna (Resistant x susceptible) under normal and late sown conditions and under artificial inoculation. The analysis of F_1, F_2 and backcross generations revealed the importance of additive, dominance and epistatic gene effects, dominance effects being particularly important. The disease was more prevalent under late sown conditions. They advocated the procedure for recurrent selection for improvement of resistance.

Singh and Singh (1987) evaluated resistance to *A. candida* in 80 *B. juncea* progenies from F_3 of the complex cross (RH30 x Domo) x (RIK 78-6 x Prakash) x (Varuna x TM2) where Domo, RIK78-6 and TM2 had moderate to high resistance while other lines were susceptible. Variation in resistance among the progenies was high (0.67-69.07%). They estimated that the additive genetic variance predominated in the control of the trait.

Narrow sense heritability was estimated to be 52.16%. The expected genetic gain through full-sib family selection was 33.76%.

In an attempt to transfer white rust resistance from *B. carinata* to *B. juncea*, interspecific hybridization was resorted to by Singh and Singh (1987) and found the hybrids to be resistant to white rust, with some additionally showing tolerance to *Alternaria brassicae*. From F_6 progenies of interspecific crosses between *B. juncea* and *B. carinata*, Singh *et al.* (1988) isolated derivatives with a high degree of resistance (96%) to white rust and high seed weight and oil content. Presence of independent single dominant genes for white rust resistance in *B. napus* was reported by Liu *et al.* (1987).

The inheritance of resistance to white rust (*A. candida*) race 2 in mustard was studied by Tiwari *et al.* (1988) in crosses involving one resistant and two susceptible cultivars. The study revealed that resistance is monogenic and could be easily transferred to adapted susceptible genotypes via backcrossing. The observed resistance to white rust in isolated experiments with various material has been reported to be monogenic (Singh and Singh, 1988) digenic (Liu *et al.*, 1987) or trigenic.

Verma and Bhowmik (1989) reported a duplicate, dominant nature of white rust resistance observed in the crosses between resistant and susceptible lines of *B. napus* against *B. juncea* pathotype. They found that F_1 was resistant and a 3:1 ratio of resistant: susceptible was demonstrated by test cross. The F_2 gave a characteristic 15 resistant: 1 susceptible ratio.

Information on genetic variation is derived from data on seed yield and reaction to white rust (*A. candida*) in the parents and 66 F_1 progenies from a 12 x 12 diallel cross involving susceptible and resistant *B. juncea* parents of Indian and exotic origin by Yashpal *et al* (1991). The material was grown under 4 environments (normal vs. late sown and natural vs.

artificially created epiphytotic conditions). Both additive and non additive components of variance were considered to be important for both the characters in all 4 environments. They suggested that pedigree selection can be exploited to simultaneously improve both the seed yield and white rust resistance of biparental hybrid material from Indian x exotic crosses. Partial dominance for resistance to *A. candida* was observed.

The inheritance of resistance to an isolate of *A. candida* collected from *B. carinata* was investigated by Liu and Rimmer (1991) using the *B. napus* lines. All F_1 plants derived from two crosses between the resistant line 2282-9 and the susceptible line DHF86-36 showed a resistant reaction. The segregation ratio of 3 resistant: 1 susceptible in the F_2 generation reveals that resistance of 2282-9 to the *B. carinata* isolate is conditioned by a single dominant resistance gene. This was confirmed when progenies derived from the backcross between F_1 plants and the susceptible parent segregated in a 1 resistant:1 susceptible ratio.

Gadewadikar *et al.* (1993) studied inheritance of resistance to white rust (*A. candida*) disease of Indian mustard by involving three exotic donors viz., L-6, L-4 and R-908 and national promising varieties Pusa Bold, Rohini, Krishna and RA-9. They showed that resistance was governed by monogenic dominant nuclear gene pair.

Paladhi *et al.* (1993) showed that the field resistance to *A. candida* was monogenic dominant by studying F_1 and F_2 generations of intervarietal crosses involving one resistant (PI-15) and four susceptible *B.juncea* genotypes.

Ten mustard lines with different degrees of resistance to *A. candida* were crossed in all possible combinations to study the genetic control of resistance. Forty-five experimental hybrids were produced during 1987-88

with two sowing dates such that the effects of different environmental variables could also be evaluated. Additive gene effects were more important than dominance effects in the inheritance of resistance. The lines EC12674B, EC126745, Domo-4 and EC 126746-1 were identified as parents for breeding disease resistant cultivars (Yadav *et al.*, 1994).

Subudhi and Raut (1994) reported that white rust resistance was under digenic control in the studies involving F_1 and F_2 progeny of cross between *B. juncea* (Pusa Barani) as female parent and 3 strains of *B. napus* (N 368, N576 and N 606) as male. Hybrids were having intermediate morphology but in the F_2 generation, segregation into types similar to either parent was observed in varying frequencies. A close association of parental species type and different grades of leaf waxiness with white rust resistance was noted. It was possible to recover waxy or medium waxy *B. juncea* types with white rust resistance, though in low frequencies.

Rao and Raut (1994) reported that resistance was expressed as a dominant phenotype and controlled by nuclear gene in all F_1 and F_1 M_1 in a cross of Indian synthetic *napus* (ISN 706) resistant under artificial inoculation to *A. candida* and widely grown *B. juncea* cv. Varuna. F_1 hybrid seeds were treated with 0.5% EDTA for 13h, and subsequently F_1, F_2, F_2M_2 and BC_1F_1 populations were grown. The F_2M_2 and F_2 populations showed 13 resistant: 3 susceptible ratio. Progenies of backcrossing to susceptible parents segregated as 1 resistant:1 susceptible and all those from backcrossing to resistant parents were resistant. These ratios indicated the presence of digenic inheritance with dominant and recessive gene interaction.

Ferreira *et al.* (1995) reported that the control of resistance to white rust (*A. candida*) attributed to a dominant allele at a single locus, designated ACA1, by the population of backcross (BCP_2) as well as F_1 derived double haploid (DH) and F_2 obtained from some single F_1 plant (produced from the

cultivar major X stellar) in rape. They suggested that other loci may be involved in the control of the intensity of sporulation of the fungus in the plant. The ACA1 locus was mapped with respect to RFLP loci in the F_1 DH population. Linkage between the ACA1 locus and 9 RFLP loci was observed on linkage group 9 of a rape RFLP linkage map (Ferreira *et al.*, 1995).

Cole *et al.* (1996) reported that genetic analysis for resistance to race 2 of *A. candida* in an F_2 population and a set of F_3 families both derived from a cross between *Brassica rapa* cultivars Per (resistant) and R-500 (susceptible) revealed that resistance is controlled by a dominant allele at a single locus. White rust resistance was associated with leaf pubescence, which also was governed by a dominant allele at a single locus. The resistance locus (ACA1) was mapped by linkage analysis with 144 RFLP loci segregating among the F_3 families. The ACA1 locus was mapped to linkage group 4 and was flanked by RFLP marker loci ec 2b3a (5.4 cM) and wg6c1a (5.0 cM). ACA 1 was liked to the leaf pubescence locus PUB 1 by 13.3 cM.

Liu *et al.* (1996) reported that resistance to *A. candida* race 7 in the Canadian *Brassica napus* cultivar Regent is controlled by three dominant genes, designated as Ac7-1, Ac7-2 and Ac7-3. Gene Ac7-3 is present in a heterozygous condition. Homozygous resistant BC_1F_3 lines carrying Ac7-1 or Ac7-2 were developed from a BC_1F_1 family in which segregation for white rust resistance fitted a 3R:1S ratio. To isolate the resistant genes, one BC_1F_3 line was assumed to have genotype Ac7-1 Ac7-1 ac7-2 ac7-2 and was used as a tester to cross with other selected BC_1F_3 lines. Progenies from test crosses were self pollinated and backcrossed to susceptible line 2281-9. Thus, two monogenic lines possessing Ac7-1 or Ac7-2 were developed as F_2 and backcross populations produced from four test crosses segregated to fit 15R:1S and 3R:1S ratios respectively, whereas the other four lines were homozygous resistant. The two single gene lines were used as testers to develop a monogenic line with Ac 7-3 from a BC_1F_1 family that

segregated in a 7R:1S ratio. These single genes are being incorporated into rapid cycling *B. napus* lines susceptible to several pathotypes of *A. candida* to develop isogenic differential lines. The monogenic lines can be used to study the mechanism (s) of resistance response conditioned by the individual genes. These lines also should facilitate molecular mapping of the loci in *B. napus* for resistance to *A. candida* race 7.

In a study Sridhar (1996) observed in the F_2 of 11 different crosses of *B. juncea* for resistance to white rust, segregation ratio of either 1R:3S or 3R:1S. Based on the mode of segregation in F_2 generation the resistance was found to be controlled by a single dominant gene showing monogenic dominant behaviour. In crosses where the segregation ratio for white rust was found to be 1R:3S, F_1 was susceptible, suggesting the presence of two doses of dominant genes are required for conferring resistance. In other words, the gene is haplo insufficient. The segregation ratio of 3R:1S clearly indicates the monogenic dominant behaviour of resistance to white rust in which case it is haplosufficient and the F_1 shows resistance. It was difficult to interpret the observation that in one case resistance was dominant and in the other susceptibility was dominant.

2.3 Association Studies with white rust resistance

2.3.1 Leaf Waxiness

Association of morphological trait with tolerance to aphid infestation or resistance to disease could be of immense importance in resistance breeding programme as it simplifies the identification and selection of resistant plants in segregating populations or even at early stages of growth. Such desirable association between nonwaxy or glossy nature of plants and aphid tolerance has been observed by Yadava *et al.* (1985a). Conn and

Tiwari (1989) reported the presence of an epicuticular wax layer conferring resistance to *Alternaria brassicae.*

In *B. juncea* a single recessive gene controlling, nonwaxy trait was reported by Yadava *et al.* (1985a), but dominant single gene control was indicated by Angadi *et al.* (1987) and diallelic locus with glossy stem trait dominant reported by Sengupta and Mukhopadhyaya (1981). In *B. napus* two recessive genes were controlling non-waxy trait as reported by XiRong *et al.* (1995) and Jianguo *et al.* (1995).

The role of surface waxes in reducing infection may be attributed to a physical or chemical barrier effect, an influence on leaf wettability or a combination of these factors.

Subudhi and Raut (1994) reported a close association of parental species type and different grades of leaf waxiness with white rust resistance. In the studies involving F_1 and F_2 progeny of cross between *B. juncea* (Pusa Barani) as female parent and 3 strains of *B. napus* (N368, N576 and N606), hybrids were of intermediate morphology but in the F_2 generation, segregation into types similar to either parent was observed in varying frequencies. It was possible to recover waxy or medium waxy *B. juncea* types with white rust resistance, though in low frequencies.

In *B. carinata* Jambhulkar and Raut (1995) reported crosses between lines with yellow or white flowers and waxy or glossy leaves reciprocally. F_1's showed intermediate expression between the two parents, indicating partial dominance. The F_2 segregation ratio confirmed monogenic inheritance with incomplete dominance. White flower colour and leaf waxiness conferred resistance to aphids and were closely associated with resistance to white rust (*A. candida*) and *Alternaria brassicae.* These traits could be transferred by simple breeding and selection programmes.

2.3.2 Yellow Seed Colour

Breeding for yellow seeded cultivars of mustard is considered an important objective because yellow seeds possess a thinner seed coat with smaller cell size than that of dark seeds resulting in higher oil percent and protein content and lower crude fibres (Stringham *et al.*, 1974; Woods, 1980; Shirzadegan and Robbelen, 1985; Abraham and Kotwal, 1995). The seed coat colour on rape and turnip rape is controlled by genes with pleiotropic effects resulting in a considerably improved oil content and reduced content of seed coat and fibre as compared to dark coloured seeds (Jonsson and Bengtsson, 1970). The inheritance of seed coat colour in different species of *Brassica* has been reported by various workers. In *B. juncea*, brown seed coat colour was reported as monogenic dominant over yellow (Nayar and George, 1970; Singh and Srivastava, 1974 and Chauhan *et al.*, 1995), duplicate dominant blackish brown over yellow (Vera *et al.*, 1979; Chauhan and Kumar, 1987) and maternal control, duplicate, digenic epistasis (Anand *et al.*, 1985; Rawat, 1989). In *B. napus* three independent dominant genes were reported for seed coat colour by Shrizadegan (1986) and in *B. carinata* monogenic dominant with incomplete dominance of brown over yellow was reported by Getinet *et al.* (1987) and two locus model with incomplete dominance of brown over yellow by Yousuf (1982).

There are reports indicating superiority of yellow seeded varieties over blackish and brown type in their tolerance to *Alternaria* blight and mustard aphid (Asthana *et al.*, 1975; Abraham and Bhatia, 1994).

In a report, Sridhar (1996) observed a relationship of yellow seededness with white rust resistance indicating that the genes determining yellow seed colour might have close linkage to the genes governing white rust resistance thereby providing yellow seed colour to serve as a marker in identifying white rust resistant plants in future breeding programme, in the

crosses involving one yellow seeded parent. This aspect requires further confirmation by detailed analysis.

2.4 Genetic Architecture

Choice of an appropriate strategy for yield improvement in any crop requires knowledge of nature and magnitude of gene action and association of characters with yield and also on the extent of environmental influence on the characters. This indicates the need for partitioning the overall variability into heritable and non-heritable components with the help of a suitable statistical technique. Fisher (1918) partitioned the heriditary variance for the metric trait into an additive portion resulting from the intra-allelic interaction and an epistatic part associated with interallelic interaction. Wright (1921, 1935) also defined additive genetic variance and variance due to dominance and epistatic deviation from the additive scheme resulting from the interaction of alleles and non-allelic genes. Cockerham (1954) further showed that epistatic variance can be partitioned into digenic interaction of the additive x additive, additive x dominance and dominance x dominance types and of higher order interactions. Such partitioning of variability into components requires raising of a number of related generations and their evaluation in an appropriate statistical design. A comprehensive summary of methods of estimating genetic variance has been presented by Cockerham (1963).

2.4.1 Nature of gene action

A comprehensive survey of the genetic architecture of yield and its component characters in *B. juncea* by different workers using different mating designs is presented in Table 1.

Table 1: Genetic architecture of yield and component characters in *B. juncea*

Character/ Nature of Gene action		References
1. Days to flowering		
Predominantly additive	:	Bhadouria *et al.* (1991), Singh *et al.* (1996).
Predominantly non-additive	:	Yadav (1992)
Additive and non-additive	:	Labana *et al.* (1978), Pradhan *et al.* (1993)
Partial dominance	:	Kumar *et al.* (1994).
2. Days to maturity		
Non-additive	:	Yadava *et al.* (1981)
Additive and non-additive	:	Labana *et al.* (1978), Jindal and Labana (1986)
3. Plant height		
Additive	:	Yadava *et al.* (1979); Singh *et al.* (1982); Gupta *et al.* (1987), Singh and Chauhan (1987), Pradhan *et al.* (1993).
Additive and non-additive	:	Singh *et al.* (1981), Chaudhary and Sharma (1982), Ram Dhari and Yadava (1983), Labana *et al.* (1984), Anand and Reddy (1987), Badwal and

		Labana (1987), Yadava *et al.* (1981), Yadava *et al.*(1993), Kumar *et al.* (1994).
Non-additive	:	Yadava (1983), Jain *et al.* (1988)
Dominance and Epistasis:	:	Kumar *et al.* (1998).

4. Number of Primary Branches

Additive and non-additive:		Singh *et al.* (1982), Yadava (1983), Ram Dhari and Yadava (1983), Singh *et al.* (1986), Singh and Chauhan (1987), Anand and Reddy(1987), Jindal and Labana (1986), Chaudhary *et al.*(1987), Badwal and Labana (1987), Yadav *et al.*(1992), Yadava *et al.* (1993), Diwakar and Singh (1993); Kumar *et al.* (1994).
Additive	:	Verma and Singh (1998).
Non-additive	:	Yadav *et al.* (1979), Labana *et al.* (1978), Gupta *et al.* (1987); Jain *et al.* (1988).
Dominance and Epistasis:		Chaudhary and Sharma (1982), Verma *et al.* (1992),Kumar *et al.* (1998).

5. Number of secondary branches

Additive and non-additive:	Ram Dhari and Yadava (1983), Labana *et al.* (1984), Chaudhary *et al.* (1987), Badwal and Labana (1987), Yadav *et al.* (1992), Yadava *et al.* (1993), Kumar *et al.* (1994).

Non-additive : Labana *et al.* (1978), Yadava *et al.* (1979), Singh *et al.* (1981), Singh *et al.* (1982), Gupta *et al.* (1987), Jain *et al.* (1988), Singh and Mital (1993).

Epistasis : Verma *et al.* (1992); Verma and Singh (1998).

Dominance and Epistasis: Kumar *et al.* (1998).

6. Length of main fruiting axis:

Additive and non-additive: Singh *et al.* (1981), Chaudhary and Sharma (1982), Ram Dhari and Yadava (1983), Rawat (1992), Singh and Mital (1993).

Non-additive : Anand and Reddy (1987), Gupta *et al.*, (1987), Singh *et al.* (1989).

Dominance and epistasis: Kumar *et al.* (1998).

7. Number of siliqua on main fruiting axis

Additive and non-additive: Singh *et al.* (1981), Labana *et al.* (1984), Verma *et al.* (1992),Singh and Mital(1993).

Non-additive : Anand and Reddy (1987), Gupta *et al.* (1987).

Dominance : Sachan and Singh (1988), Singh *et al.*(1996).

Dominance and epistasis: Kumar *et al.* (1998).

8. 1000-seed weight

Additive : Yadav (1992), Singh *et al.* (1996).

Additive and non-additive: Jindal and Labana (1986),Chaudhary *et al.* (1987), Anand and Reddy (1987),

Badwal and Labana (1987), Singh *et al.* (1989), Rishi Pal *et al.* (1993).

Non-additive : Yadava *et al.* (1979), Labana *et al.* (1978), Chauhan and Singh (1980), Yadava *et al.* (1981), Singh *et al.* (1986), Jain *et al.* (1988).

Epistasis : Verma and Singh (1998).

9. Seed yield per plant

Additive and non-additive: Ram Dhari and Yadava (1983), Jindal and Labana (1986); Chaudhary *et al.* (1987), Jain *et al.* (1988), Singh and Mital (1993).

Non-additive : Labana *et al.* (1978), Yadava *et al.* (1979), Govil *et al.* (1981), Yadava *et al.* (1981), Singh *et al.* (1981), Singh *et al.* (1982), Anand and Reddy (1987), Gupta *et al.* (1987).

Epistasis : Verma *et al.* (1992); Verma and Singh (1998).

10. Oil content

Additive and non-additive: Yadava *et al.* (1981), Govil *et al.* (1983).

Additive and dominance : Singh *et al.* (1986), Singh *et al.*(1989), Pahuja *et al.* (1996).

Non-additive : Govil *et al.* (1981), Badwal and Labana (1987).

Dominance : Thakral and Singh (1995).

Additive, dominance and Epistasis : Verma *et al.* (1992); Chauhan *et al.* (1996).

It was observed that yield and its components are controlled by varying proportion of additive and non-additive gene effects. The importance of different components of genetic variance differed according to cross and trait (Thukral *et al.*, 1986). Most of the characters are under the control of duplicate epistasis (Singh *et al.*, 1981; Ram Dhari and Yadava, 1983; Anand and Reddy, 1987; Jain *et al.*, 1988 and Kumar *et al.*, 1998). Thus for the exploitation of both additive and non-additive gene effects, reciprocal recurrent selection has been advocated by many workers by accumulation of favourable additive genes and also simultaneously maintaining heterozygosity in population for manifestation of dominance and epistasis gene effects.

MATERIALS AND METHODS

3.1 Materials

The materials included in the present study comprises of ten different strains of *Brassica juncea*, all showing resistance to white rust mostly derived from interspecific crosses and stabilised as *B. juncea* and a susceptible standard variety Pusa Bold.

The parentage of the strains included in this study, the suggested mode of inheritance of resistant genes carried by these lines from earlier studies and a brief description are given in Table-2, followed by a brief description of original parental lines. This provides a key to the different sources of white rust resistant genes.

Table 2: Parentage of strains, the nature and source of white rust resistance and their brief description

S. No.	Strain No.	Parentage	Segregation (R:S)	Description
1.	S-I	Varuna x AB-4-3	1:3	A white rust resistant strain along with tolerance to *Alternaria* blight (source: *B. carinata*).
2.	S-II	Varuna x WR-16-3-1	3:1	A high degree of white rust resistant strains showing segregation for leaf waxiness (source *B. napus*).

3.	S-III	EC287711 x AB-5	1:3	A white rust resistant strain along with tolerance to *Alternaria* blight, low erucic acid and segregation for leaf waxiness (source *B. carinata*).
4.	S-IV	EC287711 x WR-16-3-6	3:1	A high degree of white rust resistant strain, having low erucic acid and segregation for leaf waxiness (source *B. napus*).
5.	S-V	VSL-1 x AB-5	1:3	A high yielding white rust resistant strain (source *B. carinata*).
6.	S-VI	VSL-1 x WR-16-3-6	3:1	A high yielding, highly white rust resistant strain with segregation for leaf waxiness (source *B. napus*).
7.	S-VII	PSR-7 x AB-10-1	3:1	Another high yielding white rust resistant strain, showing segregation for leaf waxiness (source *B. carinata*).
8.	S-VIII	PSR-7 x AB-5	1:3	A high yielding white rust resistant strain with tolerance to *Alternaria* blight. (source *B. carinata*).
9.	S-IX	Varuna x PCR05	-	A yellow seeded, high oil content and white rust resistant strain (source *unknown*).
10.	S-X	Varuna x PCR04	-	A yellow seeded, high oil content and white rust resistant strain (source unknown).
11.	Pusa Bold		Suscep-tible	A popular bold seeded variety released for cultivation by IARI

Brief description of original parental strains are given below:

Female Parents

Varuna	:	Most widely grown mustard variety of the country.
VSL-1	:	A high yielding strain developed by crossing Varuna with synthetic *Brassica juncea*.
PSR-7		A high yielding strain developed by inter-varietal crossing
EC287711	:	Exotic introduction for low erucic acid.

Male parents

WR-16-3-1 and WR-16-3-6	:	White rust resistant strains developed from interspecific crosses of *B. juncea* with *B. napus* and stabilized as *B. juncea*.
AB-4-3, AB-5 and AB-10-1	:	White rust resistant, *Alternaria* blight tolerant and yellow seeded strains derived from interspecific hybridization between *B. juncea* with *B.carinata* and stabilized as *B. juncea*.

PCR-04 and PCR-05 .: White rust resistant yellow seeded strains received from Project Coordinator Rapeseed and Mustard.

3.2 Methods

3.2.1 Crossing programme

Two row lines of several resistant strains to white rust available in the section were grown during Rabi 1995-96 at IARI Farm, New Delhi. The growing conditions were made congenial for the development of white rust. Ten selected plants from each resistant strain and a susceptible strain Pusa Bold were crossed in a diallel manner excluding reciprocals to obtain 55 possible F_1s. Crossing work was undertaken during 1995-96 rabi season at IARI Farm, New Delhi. Emasculation and pollination was done in standard manner. Buds from plant to be used as female were emasculated one day before anthesis after clipping of the immature buds and opened flowers. To ensure sufficient amount of seeds, nearly 800-1000 buds were pollinated per cross combination. Pollination was done in the next morning by dusting fresh pollen of just dehisced anthers collected from plants of male parents on the stigmatic surface of emasculated buds of female parents. Butter paper bags were used to protect the emasculated buds from foreign pollens. Soon after pollination, again bags were placed with appropriate tagging. Scoring of disease resistance was done at flowering and podding stage. Parents were also selfed and seeds were harvested individually for each cross combination.

3.2.2 Raising F_1, F_2 and Backcross generations

All the F_1s along with their parents were advanced during summer, 1996 (June-Sept.) in off-season nursery at IARI regional station, Wellington (The Nilgiris), Tamilnadu. A single row was grown for each F_1 and parents. Spacing was 45 cm from row to row and 10-15 cm from plant to plant within the row. Wellington (T.N.) is supposed to be a hot-spot for white rust infection due to its congenial climatic conditions for pathogen of white rust disease and suspected availability of various races of pathogen in the surrounding flora. Disease reactions for white rust were scored only at full podding stage. Seeds were harvested from the plants for each F_1 and parents only from 12 selected crosses to represent specific combinations. Most crosses (43 in number) were rejected as they did not confirm to the requirement of the study.

During 1996-97 rabi season, all the parents, crossed seeds of all hybrids F_1 and F_2 populations from resistant plants of F_1s harvested at Wellington, were grown at IARI, New Delhi field. A single 3 meter long row was allotted for all parents and F_1s but 2 rows for each of the three selected resistant F_1 plants for raising F_2 population due to limited availability of land. Here, also spacing was maintained at 45 cm from row to row and 10-15 cm between plant to plant within the row. All 12 resistant F_1s which were selected at Wellington were backcrossed to both the parents to obtain B_1 and B_2 seeds. Disease reactions for white rust were scored at both late vegetative and full podding stage. Observations were also recorded for leaf waxiness for all the generations. During this season segregation for white rust resistance could not be worked out because of the small population size of F_2. Seeds were harvested from selected plants for all generations. Precautions were taken to avoid out crossing and also to eliminate any chance mixture of off plant by roguing. The parental material raised always bred true for its original plant type and resistance or susceptibility.

3.2.3 Raising off season nursery

To understand the behaviour of white rust resistance in different regions of the country and to assess if there is any differential action due to ecological differences, a multilocation testing experiment was carried out during summer, 1997. To facilitate the process, F_2 population of selected 12 crosses were grown in off-season (June-Sept., 1997) nursery at IARI regional station, Wellington (The Nilgiris), Tamil Nadu and Himachal Pradesh Krishi Vishwa Vidyalaya, Regional Station at Kukumseri (Lahaul), H.P.

At Wellington, 10 rows, each of 2 meter length were grown for F_2 population of each cross, maintaining the spacing from row to row and plant to plant within the row at 45 cm and 10-15 cm respectively. Disease reactions were scored at full podding stage and seeds were harvested from resistant plants only.

At Kukumseri (Lahaul),H.P. only 3 rows, each of 3 meter in length were grown for F_2 population of each cross, maintaining the spacing from row to row and plant to plant at 45 cm and 10-15 cm respectively. Disease reactions for white rust were scored at full podding stage and seeds were harvested only from resistant plants.

3.2.4 Replicated trial of 5 generations

During 1997-98 rabi season, parents (P_1 and P_2), F_1, B_1 and B_2 generations of selected 10 crosses after eleminating two due to insufficient seed were grown in randomized block design with 3 replications. The ten cross combinations were evaluated for this study as indicated in the layout plan given on the following page. Three meter long single rows were alloted to each generation of each cross and parents in each replication. Row to row spacing was 45 cm and plants within row were spaced 10-15 cm.

Layout plan for replicated trial

		R I	R II	R III
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 2 (S-II-3x S-IV-2)	Cross - 1	Cross - 7
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 12 (S-VI-8xS-IX-6)	Cross - 3	Cross - 10
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 10 (S-IV-9xS-X-4)	Cross - 4	Cross - 12
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 1 (S-II-2xS-III-2)	Cross - 11	Cross - 8
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 6 (S-III-3xS-IV-3)	Cross - 8	Cross - 1
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 8 (S-IV-6xS-VII-4)	Cross - 12	Cross - 6
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 11 (S-VI-10x Pusa Bold)	Cross - 6	Cross - 4
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 3 (S-II-6xS-VII-2)	Cross - 2	Cross - 11
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 7 (S-III-9xS-X-3)	Cross - 10	Cross - 3
5 rows	P_1 P_2 F_1 B_1 B_2	Cross - 4 (S-II-7xS-VIII-2)	Cross - 7	Cross - 2

Normal cultural practices were followed without any plant protection measures. Although during 1997-98 rabi season white rust infection was most severe, artificial inoculation of *A. candida* spores was done to ensure sufficient development of disease. Disease reactions for white rust were scored at both late vegetative and full podding stage. Date of sowing was 16th October, 1997 and harvesting was done throughout March, 1998 depending on the maturity of materials.

3.2.5 Raising F_2 and F_3 populations

F_2 populations for all the selected 12 F_1s were grown separately without replication at IARI, New Delhi. Beds were of 3 meter width and 10 rows were alloted for each cross at random. F_2 populations were raised from two selected resistant F_1 plants from each cross. Resistant F_1 plants were selected during previous rabi season at Delhi and summer season of 1996 at Wellington.

F_3 populations were also raised from selected resistant F_2 plants having good yield attributes at Wellington and New Delhi. These F_2 plant progenies from above locations were grown at IARI, New Delhi during 1997-98 rabi season only in separate plots. Beds were of 3 meter width and 1 row was allotted for each selected F_3 progeny. Spacing was maintained from row to row and plant to plant within the row as 45 cm and 10-15 cm respectively.

3.3 Artificial epiphytotics

Although white rust infection was very severe during 1997-98 rabi season due to congenial weather conditions, to ensure adequate pressure of white rust in the experimental area, artificial inoculation was done in the replicated trial F_2 and F_3 plots described in the preceding two subsections.

The inoculum was prepared by collecting white rust infected leaves from highly infested areas and putting them in pure water to get fungal sporangia dissolved in it. This sporangial suspension was kept for three hours to allow germination of fungal spores. The inoculum thus prepared was sprayed three times during evening hours starting from 5 weeks after sowing and then at weekly intervals. The spraying was done thoroughly so as to cover the plants uniformly, particularly on the lower side of the leaves. Congenial weather conditions were created by frequent irrigation of the plots and filling the water in the channels around the plots during the period of inoculation.

3.4 Recording Observations

3.4.1 Disease resistance

For the white rust resistance inheritance studies, resistance was scored using a standard 0-5 disease scoring scale for white rust. Category **O** was defined as the complete absence of the white rust pustules and categories **1** and **2** were also classified as resistant with very low infection which are likely to be hypersensitive. Other categories **3,4** and **5** were defined as moderate to high infection of disease with more rust pustules and later development of staghead as susceptible. Details of scoring technique are listed in Table 3. In Fig.1 are presented leaves corresponding to **0** to **5** infection intensity.

The disease reactions for all the generations throughout the course of study were scored using **0-5** scale and for calculating segregation ratios **0** to **2** are counted as resistant and **3** to **5** as susceptible (Table 3).

3.4.2 Qualitative characters

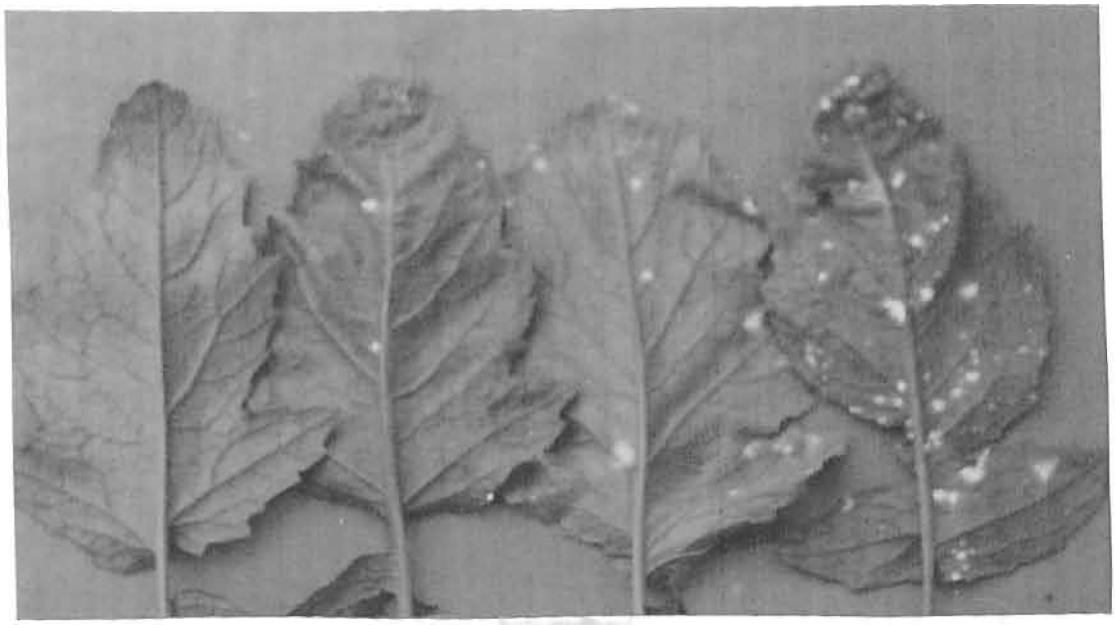
Some of the parents used had waxy leaves and some had yellow seed colour. With the idea of relating these qualitative characters with

Fig.1: Leaves and inflorescence showing different grades of white rust infection

a. Four leaves showing 0, 1, 2, and 3 grades of white rust infection in ascending order from left to right

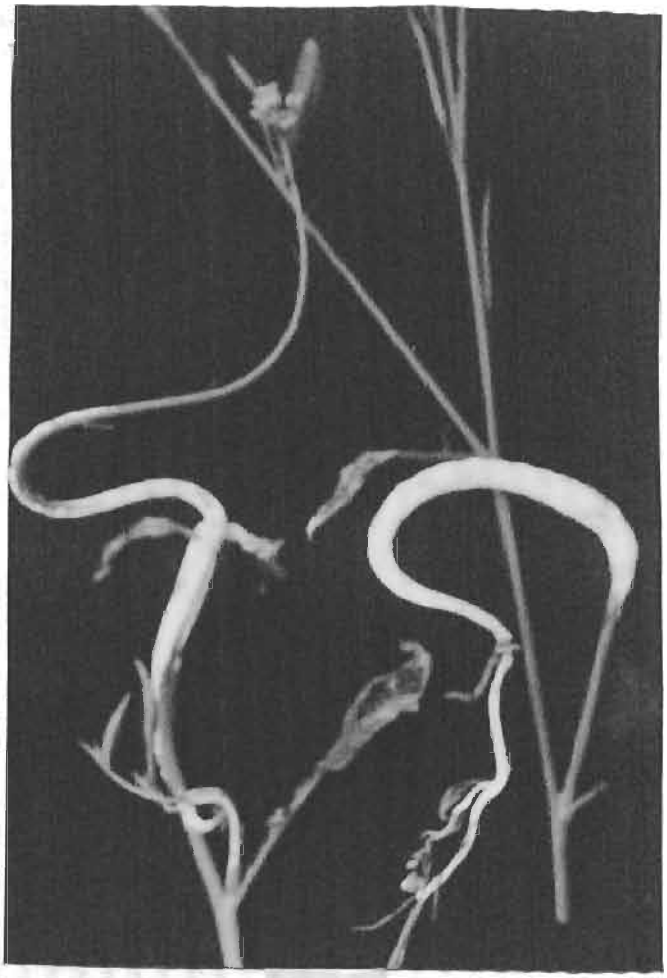
b. Two leaves showing grades 4 (right) and 5 of white rust infection

c. Inflorescence showing malformed floral parts (staghead) from highly infected white rust susceptible plants



a

b



c

disease resistance observations were recorded on leaf waxiness and seed colour in different generations. Frequency of plants with waxy and non-waxy leaves in different generations viz., F_1, F_2, F_3, B_1 and B_2 were recorded for some selected crosses during 1997-98, rabi season at IARI, New Delhi. Resistant and susceptible parents frequencies were classified under each category.

Frequency of plants with yellow and brown seed colour in different generationsviz., P_1, P_2, F_1, F_2, B_1, and B_2 were noted for some selected crosses during 1997-98 rabi season at IARI, New Delhi and have also resistant and susceptible plants were classified under each.

Table 3: Visual disease scoring scale (0-5) used for grading of resistant and susceptible plants based on intensity of symptoms development

Grade	Description	Disease Reaction
0	Complete absence of the white rust pustules on the leaves and other parts of plant.	R
1	Below 5% intensity of disease infection confined to lower leaves of plant	R
2	5-15% intensity of disease infection confined to lower leaves of plant	R
3	15-40% intensity of disease infection on whole plant	S
4	40-70% intensity of disease infection on whole plant	S
5	above 70% intensity of disease infection with staghead formation	S

3.4.3 Quantitative Characters

Data regarding quantitative characters were noted from five competitive plants from P_1, P_2, F_1, B_1, and B_2 generations of each cross in each replication. In F_2 population data were recorded for 15 randomly selected plants from each F_1 plant progeny. The observations on the following ten characters of the material grown in replicated trial along with F_2 generation were recorded.

	Characters	Abbreviation
1.	Days to 50% flowering	DF
2.	Days to maturity	DM
3.	Plant height (cm)	PH
4.	Number of primary branches	PB
5.	Number of secondary branches	SB
6.	Length of the main fruiting axis (cm)	LM
7.	Number of siliquae on main fruiting axis	SM
8.	1000-seed weight (g)	SW
9.	Seed yield per plant (g)	SY
10.	Oil content (%)	OC

Statistical analysis

To study the mode of inheritance of white rust resistance in F_2 population Chi-square test was used. Yate's correction was followed, while calculating X^2 value for one degree of freedom. Partition X^2 test was used to test the deviation and heterogeneity for the segregation of white rust resistance at different locations. All these tests were carried out as per standard procedure given by Panse and Sukhatme (1985).

Gene effects were estimated based on generation mean analysis according to the procedure outlined by Hayman (1958) which is as follows for a six parameter model.

$\hat{m}$ = Mean effect $= \bar{F}_2$

$[\hat{d}]$ = Additive gene effect $= \overline{BC}_1 - \overline{BC}_2$

$[\hat{h}]$ = Dominance gene effect $= 2\overline{BC}_1 + 2\overline{BC}_2 - 4\bar{F}_2 + \bar{F}_1 - 1/2\ \bar{P}_1 - 1/2\ \bar{P}_2$

$[\hat{i}]$ = Additive x additive type of gene action $= 2\overline{BC}_1 + 2\overline{BC}_2 - 4\bar{F}_2$

$[\hat{j}]$ = Additive x dominance type of gene action =

$\overline{BC}_1 - 1/2\ \bar{P}_1 - \overline{BC}_2 + 1/2\ \bar{P}_2$

$[\hat{l}]$ = Dominance x dominance type of gene action =

$= \bar{P}_1 + \bar{P}_2 + 2\bar{F}_1 + 4\bar{F}_2 - 4\overline{BC}_1 - 4\ \overline{BC}_2$

Variances of the estimates of these parameters were calculated as follows:

$V_m = V_{\bar{F}2}$

$V_{[d]} = V_{\bar{BC}1} + V_{\bar{BC}2}$

$V_{[h]} = 4V_{\bar{BC}1} + 4V_{\bar{BC}2} + 16V_{\bar{F}2} + V_{\bar{F}1} + 1/4\ V_{\bar{P}1} + 1/4\ V_{\bar{P}2}$

$V_{[i]} = 4V_{\bar{BC}1} + 4V_{\bar{BC}2} + 16V_{\bar{F}2}$

$V_{[j]} = V_{\bar{BC}1} + 1/4\ V_{\bar{P}1} + V_{\bar{BC}2} + 1/4\ V_{\bar{P}2}$

$V_{[l]} = V_{\bar{P}1} + V_{\bar{P}2} + 4V_{\bar{F}1} + 16V_{\bar{F}2} + 16V_{\bar{BC}1} + 16V_{\bar{BC}2}$

Standard errors were calculated by taking square-root of the variances calculated above.

't' values were calculated by the following formulae:

$t_{(m)} = m/SE_{[m]}$

$t_{[d]} = [d]/SE_{[d]}$

$t_{[h]} = [h]/\ SE_{[h]}$

$t_{[i]} = [i]/SE_{[i]}$

$t_{[j]} = [j]/SE_{[j]}$

$t_{[l]} = [l]/SE_{[l]}$

The significance of each parameter is tested by 't' test comparing the calculated 't' with Table 't' value at appropriate degree of freedom.

Ranking of the selected 10 crosses were carried out by employing the Duncan's Multiple Range Test using means of the two parents and their F_1 hybrid for different characters.

RESULTS

The results of the present study are based on the observations recorded and analysis carried out for inheritance of white rust resistance growing the appropriate material at three different locations viz. Wellington (T.N.), Kukumseri (HP) and Delhi. Along with disease resistance quantitative data was recorded on P_1, P_2, F_1, F_2, B_1 and B_2 generations and estimation of gene effects for yield and its components were made and resistant high yielding genotypes or cross combinations identified by comparing their means for different characters. It also included association studies of leaf waxiness and yellow seed colour with white rust resistance.

The results obtained are presented under following headings:

White rust resistance:

- Inheritance studies
- Variation in expression of white rust resistance in different crosses
- Variation in expression of white rust resistance under 0-5 scale at different locations
- Association of leaf waxiness with white rust resistance
- Association of yellow seed colour with white rust resistance

Estimation of gene effects:

Identification of desirable crosses/recombinants:

4.1 White rust resistance

4.1.1 Inheritance studies

White rust caused by *A. candida* is the most important disease of mustard in India. The disease, when severe, causes heavy losses in the yield of this crop. The disease is characterised by prominent white to creamy yellow pustules which often coalesce to form patches on the under side of leaves. Susceptible plants sometimes form stagheads when the infection is severe (Fig. 2)..

Genetic analysis or inheritance was carried out only for 12 selected crosses which represent specific combination to fulfill the objectives of the present study.

The crosses between two parents both having resistance to white rust yielded all resistant plants in F_1. The F_2 population segregated into resistant and susceptible types, (Fig.3) within each there were different grades of resistance or susceptibility. Data regarding segregation pattern classifying into resistant or susceptible category in two crosses (No.1 and 6) are given in Tables 4 and 5. Chi-square test showed a close fit to 13 Resistant:3 Susceptible ratio. This ratio of 13R:3S in F_2 was found in agreement at all three locations by the use of χ^2 test of heterogeneity. F_3 progenies raised from individual F_2 plants were either true breeding with all resistant plants or segregated into resistant and susceptible plants. In back cross with parent one and parent two all the plants were resistant. It indicates the digenic

Table 4. Response and segregation pattern of different generations for white rust resistance in Cross No. 1 (S-II-2xS-III-2) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	24	-	-	-	-
P_2	R	Delhi	22	-	-	-	-
F_1	R	Delhi	32	-	-	-	-
F_2	Seg.	Wellington	70	22	13:3	2.577	0.10-0.20
F_2	Seg.	Kukumseri	49	7	13:3	0.682	0.30-0.50
F_2	Seg.	Delhi	190	31	13:3	5.858	0.01-0.02
Deviation (associated with 1 d.f.)						2.430	0.10-0.20
Heterogeneity (associated with 2 d.f.)						3.860	0.10-0.20
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	34	-		2	100
	Seg.	Delhi	61	31		4	66.3
F_3 (W)	R	Delhi	48	-		2	100
	Seg.	Delhi	39	6		2	86.7
B_1	R	Delhi	20	-		-	100
B_2	R	Delhi	18	-		-	100

Table 5. Response and segregation pattern of different generations for white rust resistance in Cross No. 6 (S-III-3xS-IV-3) at different locations.

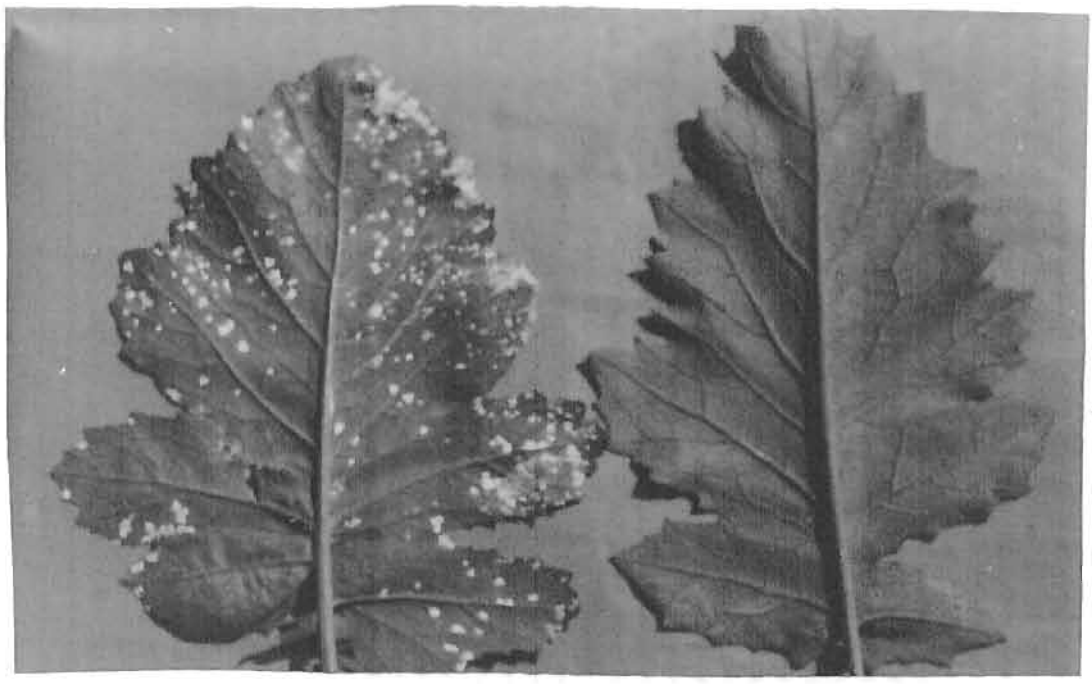
Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	18	-	-	-	-
P_2	R	Delhi	21	-	-	-	-
F_1	R	Delhi	29	-	-	-	-
F_2	Seg.	Wellington	66	24	13:3	3.210	0.05-0.10
F_2	Seg.	Kukumseri	41	12	13:3	0.302	0.50-0.70
F_2	Seg.	Delhi	227	49	13:3	0.320	0.50-0.70
Deviation (associated with 1 d.f.)						0.375	0.50-0.70
Heterogeneity (associated with 2 d.f.)						4.272	0.10-0.20
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	88	-		5	100
	Seg.	Delhi	14	5		1	73.7
F_3 (W)	R	Delhi	19	-		2	100
B_1	R	Delhi	32	-		-	100
B_2	R	Delhi	24	-		-	100

Fig.2: Symptoms of white rust disease on leaves, plants and inflorescence

a. A leaf from a resistant plant (right) and a susceptible plant (left) showing prominent white rust pustules

b. Showing comparision of resistant (left) and susceptible plants to white rust. The susceptible plants had several stagheads

c. Inflorescences showing staghead formation due to severe infection of white rust



a

b

c

Fig.3: Plants showing resistance and susceptibility to white rust in F_2 population of Cross No. 1 and 6

a. A plant showing resistance to white rust in F_2 population

b. A plant showing susceptibility to white rust in F_2 population. Leaves clearly show the disease symptoms

a

b

inheritance of white rust resistance with two different and totally independent genes with some interaction. It means one parent was having the monogenic dominant gene and other parent was having the monogenic recessive gene for white rust resistance. There can be other interpretations to arrive at the same ratio which aspect will be treated in the discussion section.

In two other crosses (No.2 and 3) between resistant parents F_1 generation had all resistant plants. The data regarding segregation pattern classifying into resistant or susceptible category (Fig. 4) in these two crosses are given in Tables 6 and 7. Chi-square test showed a close fit to 3 Resistant:1 Susceptible ratio in F_2. This ratio 3R:1S in F_2 was found in agreement at all three locations showing highly significant χ^2 test of heterogeneity. At Delhi, one F_1 plant progeny bred true for white rust resistance, yielding only resistant plants in F_2. F_3 single plant progenies had either all resistant types or segregated into resistant and susceptible plants with high percentage of resistant plants. In backcross with parent one (P_1) and parent two (P_2) all the plants were resistant. It indicates the monogenic inheritance involving same gene for white rust resistance.

Five crosses (No.4,8,9,11 and 12) involving one resistant and one susceptible parent showed all the resistant plants in F_1. It indicates complete dominance of resistance over susceptibility. The data regarding segregation pattern of these crosses in different generations are given in Tables 8,9,10,11 and 12. Chi-square test showed a close fit to 3 Resistant:1 Susceptible ratio in F_2. This ratio 3R:1S in F_2 was found in agreement at all three locations showing significant values of χ^2 test of heterogeneity. F_3 single plant progenies showed either all resistant types or segregated into resistant and susceptible plants with majority of resistant plants (Fig.5). In backcross with resistant plant all the plants were resistant and in backcross

Table 6. Response and segregation pattern of different generations for white rust resistance in Cross No. 2 (S-II-3xS-IV-2) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	24	-	-	-	-
P_2	R	Delhi	20	-	-	-	-
F_1	R	Delhi	19	-	-	-	-
F_2	Seg.	Wellington	94	28	3:1	0.175	0.50-0.70
F_2	Seg.	Kukumsari	40	15	3:1	0.055	0.80-0.90
F_2	R	Delhi	41	-	-	-	-
	Seg.	Delhi	81	29	3:1	0.048	0.80-0.90
Deviation (associated with 1 d.f.)						0.001	0.95-1.00
Heterogeneity (associated with 2 d.f.)						0.533	0.70-0.80
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	65	-		3	100
	Seg.	Delhi	23	31		3	42.6
F_3 (W)	R	Delhi	74	-		3	100
	Seg.	Delhi	18	19		1	48.6
B_1	R	Delhi	21	-		-	100
B_2	R	Delhi	54	-		-	100

Table 7. Response and segregation pattern of different generations for white rust resistance in Cross No. 3 (S-II-6xS-VII-2) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	26	-	-	-	-
P_2	R	Delhi	23	-	-	-	-
F_1	R	Delhi	21	-	-	-	-
F_2	Seg.	Wellington	70	27	3:1	0.278	0.50-0.70
F_2	Seg.	Kukumseri	37	15	3:1	0.231	0.50-0.70
F_2	R	Delhi	117	-	-	-	-
	Seg.	Delhi	85	29	3:1	0.000	1.00
Deviation (associated with 1 d.f.)						0.559	0.30-0.50
Heterogeneity (associated with 2 d.f.)						0.279	0.80-0.90
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	78	-		4	100
	Seg.	Delhi	20	14		2	58.8
F_3 (W)	R	Delhi	92	-		4	100
B_1	R	Delhi	22	-		-	100
B_2	R	Delhi	35	-		-	100

Table 8. Response and segregation pattern of different generations for white rust resistance in Cross No. 4 (S-II-7xS-VIII-2) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	19	-	-	-	-
P_2	S	Delhi	-	17	-	-	-
F_1	R	Delhi	21	-	-	-	-
F_2	Seg.	Wellington	62	26	3:1	0.755	0.30-0.50
F_2	Seg.	Kukumseri	38	18	3:1	1.167	0.20-0.30
F_2	Seg.	Delhi	170	67	3:1	1.183	0.20-0.30
Deviation (associated with 1 d.f.)						3.470	0.05-0.10
Heterogeneity (associated with 2 d.f.)						0.370	0.80-0.90
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	70	-		3	100
	Seg.	Delhi	30	22		3	57.7
F_3 (W)	R	Delhi	54	-		2	100
	Seg.	Delhi	19	14		2	57.6
B_1	R	Delhi	24	-		-	100
B_2	Seg.	Delhi	17	15		-	53.1

Table 9. Response and segregation pattern of different generations for white rust resistance in Cross No. 8 (S-IV-6xS-VII-4) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	18	-	-	-	-
P_2	S	Delhi	-	22	-	-	-
F_1	R	Delhi	22	-	-	-	-
F_2	Seg.	Wellington	63	26	3:1	0.633	0.30-0.50
F_2	Seg.	Kukumseri	35	15	3:1	0.427	0.50-0.70
F_2	Seg.	Delhi	118	41	3:1	0.019	0.80-0.90
Deviation (associated with 1 d.f.)						1.007	0.30-0.50
Heterogeneity (associated with 2 d.f.)						0.555	0.70-0.80
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	33	-		3	100
	Seg.	Delhi	26	17		3	60.5
F_3 (W)	R	Delhi	3	-		1	100
B_1	R	Delhi	23	-		-	100
B_2	R	Delhi	25	18		-	58.1

Table 10. Response and segregation pattern of different generations for white rust resistance in Cross No. 9 (S-IV-8xS-IX-4) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	S	Delhi	-	23	-	-	-
P_2	R	Delhi	19	-	-	-	-
F_1	R	Delhi	23	-	-	-	-
F_2	Seg.	Wellington	93	30	3:1	0.003	0.95-0.98
F_2	Seg.	Kukumseri	35	16	3:1	0.791	0.30-0.50
F_2	Seg.	Delhi	153	44	3:1	0.611	0.30-0.50
Deviation (associated with 1 d.f.)						0.010	0.90-0.95
Heterogeneity (associated with 2 d.f.)						1.865	0.30-0.50
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	44	-		3	100
	Seg.	Delhi	36	15		3	70.6
F_3 (W)	R	Delhi	32	-		2	100
	Seg.	Delhi	23	10		2	69.7
B_1	-	Delhi	-	-		-	-
B_2	-	Delhi	-	-		-	-

Table 11. Response and segregation pattern of different generations for white rust resistance in Cross No. 11 (S-IV-10xPusa Bold) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	20	-	-	-	-
P_2	S	Delhi	-	23	-	-	-
F_1	R	Delhi	21	-	-	-	-
F_2	Seg.	Wellington	65	29	3:1	1.419	0.20-0.30
F_2	Seg.	Kukumseri	35	17	3:1	1.256	0.20-0.30
F_2	Seg.	Delhi	163	49	3:1	0.308	0.50-0.70
Deviation (associated with 1 d.f.)						0.450	0.30-0.50
Heterogeneity (associated with 2 d.f.)						2.940	0.20-0.30
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	51	-		4	100
	Seg.	Delhi	18	21		2	46.2
F_3 (W)	R	Delhi	16	-		1	100
	Seg.	Delhi	32	27		3	54.2
B_1	R	Delhi	25	-		-	100
B_2	Seg.	Delhi	25	21		-	54.3

Table 12. Response and segregation pattern of different generations for white rust resistance in Cross No. 12 (S-VI-8xS-IX-6) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	23	-	-	-	-
P_2	S	Delhi	-	20	-	-	-
F_1	R	Delhi	21	-	-	-	-
F_2	Seg.	Wellington	53	22	3:1	0.537	0.30-0.50
F_2	Seg.	Kukumseri	36	16	3:1	0.641	0.30-0.50
F_2	Seg.	Delhi	127	54	3:1	2.005	0.10-0.20
Deviation (associated with 1 d.f.)						3.896	0.02-0.05
Heterogeneity (associated with 2 d.f.)						0.034	0.98-0.99
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	64	-		3	100
	Seg.	Delhi	32	27		3	54.2
F_3 (W)	R	Delhi	7	-		2	100
B_1	R	Delhi	34	-		-	100
B_2	Seg.	Delhi	15	12		-	55.6

Fig.4: Plants showing resistance and susceptibility to white rust in Cross No. 2 and 3

a. Plant showing resistance to white rust with clean leaves

b. Plant showing susceptibility to white rust with white pustules on under side of leaves

a

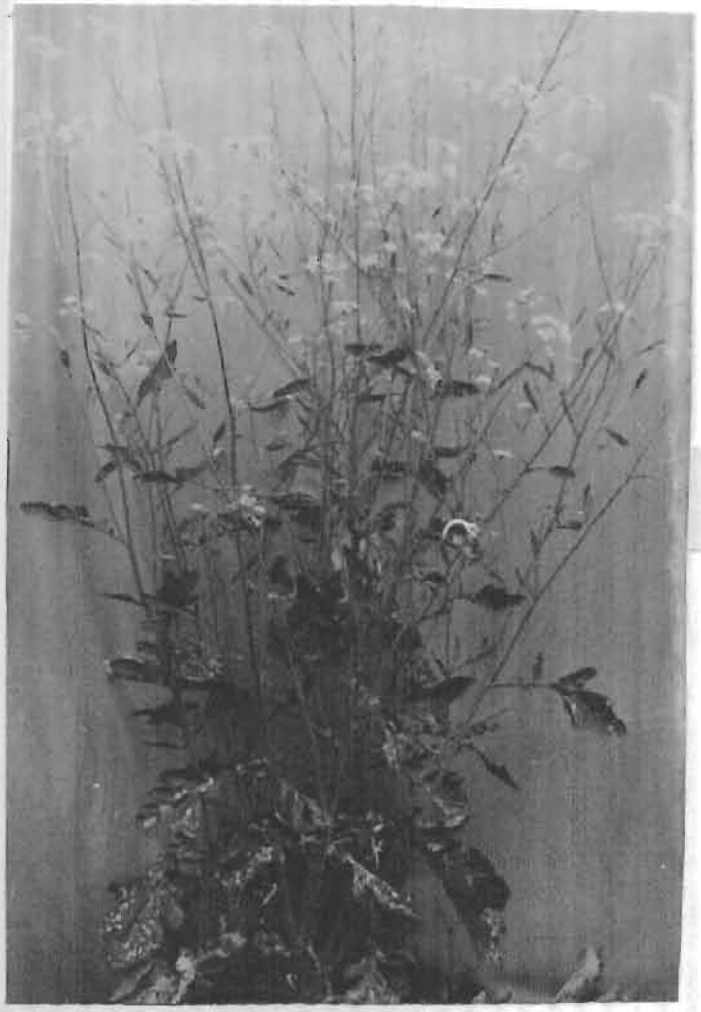
b

Fig.5: Representative plants showing resistance and susceptibility to white rust taken from F_3 population of crosses 4, 8, 9, 11 and 12

a. Plant showing resistance to white rust with clean leaves

b. Plant showing susceptibility to white rust with infected leaves

a



b

with susceptible plant, segregation into resistant and susceptible plants were found in almost equal frequencies. It indicates the monogenic inheritance of white rust resistance in all these crosses.

In another cross (No.7) involving one resistant and one susceptible parent (Fig.6) the resulting F_1 plants were all susceptible. It indicates the dominance of susceptibility over resistance. Alternatively single dose of this dominant resistant gene is not enough to confer resistance. Data regarding segregation pattern into resistant and susceptible are given in Table 13. Chi square test showed a close fit to 1 Resistant:3 Susceptible ratio in F_2. This ratio 1R:3S was found in agreement at all three locations with high probability of significance of χ^2 test of heterogeneity. F_3 single plant progenies showed either all resistant types or segregation into resistant and susceptible plants with the low percentage of resistant plants than susceptible ones. Backcross with parent one showed segregation into resistant and susceptible plants with about 1R:1S ratio. In backcross with parent two (P_2) all the plants were susceptible type. It indicates the monogenic recessive nature of white rust resistance gene or inability of the heterozygotes to confer resistance.

In one cross between two resistant parents (Cross No.5), F_1 generation had all resistant plants. The data regarding segregation pattern classifying into resistant or susceptible category in this cross are given in Table 14. Chi-square test showed a close fit to 15 Resistant:1 Susceptible ratio in F_2 at Delhi and 3 Resistant :1 Susceptible ratio in F_2 at Wellington and Kukumseri. F_3 single plant progenies showed either all resistant types or segregated into resistant and susceptible plants (Fig. 7) with high percentage of resistant plants. Although backcrosses were attempted with both the parents, their behaviour could not be studied because of failure of germination of backcrossed seeds. It indicates that at Delhi resistant genes

Table 13. Response and segregation pattern of different generations for white rust resistance in Cross No. 7 (S-III-9xS-X-3) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	18	-	-	-	-
P_2	S	Delhi	-	16	-	-	-
F_1	S	Delhi	-	19	-	-	-
F_2	Seg.	Wellington	22	48	1:3	1.210	0.20-0.30
F_2	Seg.	Kukumseri	9	32	1:3	0.070	0.70-0.80
F_2	Seg.	Delhi	45	117	1:3	0.530	0.30-0.50
Deviation (associated with 1 d.f.)						1.170	0.20-0.30
Heterogeneity (associated with 2 d.f.)						1.240	0.50-0.70
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	4	-		1	100
	Seg.	Delhi	17	27		3	38.6
F_3 (W)	R	Delhi	13	-		2	100
	Seg.	Delhi	11	14		2	44.0
B_1	Seg.	Delhi	10	8		-	55.6
B_2	S	Delhi	-	22		-	0.0

Table 14. Response and segregation pattern of different generations for white rust resistance in Cross No. 5 (S-II-9xS-X-2) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	R	Delhi	17	-	-	-	-
P_2	R	Delhi	20	-	-	-	-
F_1	R	Delhi	29	-	-	-	-
F_2	Seg.	Wellington	52	31	3:1	6.110	0.01-0.02
F_2	Seg.	Kukumseri	42	20	3:1	1.376	0.20-0.30
F_2	Seg.	Delhi	78	8	15:1	0.896	0.30-0.50
Deviation (associated with 1 d.f.)						0.036	0.80-0.90
Heterogeneity (associated with 2 d.f.)						18.750	
					F3 progeny rows		%R-plants
F_3 (D)	R	Delhi	66	-		4	100
	Seg.	Delhi	32	14		2	69.6
F_3 (W)	R	Delhi	27	-		2	100
B_1	-	Delhi	-	-		-	-
B_2	-	Delhi	-	-		-	-

Fig.6: Resistant and susceptible parental plants to white rust involved in Cross No. 7

a. A resistant plant representing parent S-III-9

b. A susceptible plant representing parent S-X-3

a

b

Fig.7: Representative plants showing resistance and susceptibility to white rust from F_3 population of Cross No. 5

a. Plant showing resistance to white rust with clean leaves

b. Plants showing susceptibility to white rust with diseased leaves and stagheads

a

b

present in both the parents expressed in F_2 but at Wellington and Kukumseri the resistant gene present in parent two (S-X-2) failed to express and behaved as susceptible due to some reason. At Delhi this cross shows digenic duplicate nature of the white rust resistance gene and at Wellington and Kukumseri monogenic dominant nature of white rust resistance gene was observed.

In one more cross (No.10) between a resistant and a susceptible parent (Fig. 8), F_1 generation had all the resistant plants. The data regarding segregation pattern into resistant or susceptible category are given in Table 15. Chi-square test showed a close fit to 3 Resistant:1 Susceptible ratio in F_2 at Delhi and 1 Resistant :3 Susceptible ratio in F_2 at Wellington and Kukumsari. F_3 single plant progenies showed segregation into resistant and susceptible plants with high percentage of resistant plants only. In backcross with resistant parent all the plants were resistant and backcross with susceptible parent showed segregation into about 1R:1S ratio. It indicates the monogenic dominant nature of white rust resistance gene at Delhi and monogenic recessive nature of white rust resistance gene at Wellington and Kukumseri locations. The reason for reversal of ratios at different locations will be interpreted in the discussion section.

4.1.2 Variation in the expression of white rust resistance in different crosses

Parents involved in these selected 12 crosses under study carried the resistant gene for white rust incorporated from different sources either through interspecific or intraspecific hybridization. The crosses between these parents, therefore, were showing the variation in the degree of expression of white rust resistance or susceptibiity.

Table 15. Response and segregation pattern of different generations for white rust resistance in Cross No. 10 (S-IV-9xS-X-4) at different locations.

Generation	Disease Reaction	Location	R	S	Ratio	χ^2	P-range
P_1	S	Delhi	-	18	-	-	-
P_2	R	Delhi	16	-	-	-	-
F_1	R	Delhi	34	-	-	-	-
F_2	Seg.	Wellington	21	73	1:3	0.227	0.50-0.70
F_2	Seg.	Kukumseri	16	40	1:3	0.215	0.50-0.70
F_2	Seg.	Delhi	182	54	3:1	0.457	0.30-0.50
Deviation (associated with 1 d.f.)						68.67	-
Heterogeneity (associated with 2 d.f.)						135.30	-
					F3 progeny rows		%R-plants
F_3 (D)	Seg.	Delhi	38	13		2	74.5
F_3 (W)	Seg.	Delhi	20	8		2	71.4
B_1	Seg.	Delhi	18	16		-	52.9
B_2	R	Delhi	22	-		-	100

Fig.8: Parental plants to Cross No. 10 showing resistant and susceptible reaction to white rust

a. A resistant plant of parental strain S-X-4

b. A susceptible plant of parental strain S-IV-9

a

b

Data presented in Table 16A, shows that in the five crosses the female parent (S-II, Fig.9) was resistant in all crosses but these five crosses showed different segregation ratios in F_2 (13R:3S, 3R:1S and 15R:1S) depending on the source of resistance gene of the male parent and whether male parent used was susceptible or resistant.

Data presented in Table 16B shows that in the two crosses female parent (S-III, Fig.10) was resistant in all crosses but these two crosses showed the different segregation ratio in F_2 (13R:3S and 1R:3S) depending on the source of resistance gene of male parent and whether it was susceptible or resistant.

Data presented in Table 16C shows that individual plants of strain IV used as female parent was resistant in two crosses and susceptible in two crosses (Fig. 11), but out of these four crosses, three crosses showed 3Resistant: 1 Susceptible segregation ratio in F_2 at all locations except one cross which showed 3 Resistant:1 Susceptible segregation ratio at Delhi and 1 Resistant:3 Susceptible at Wellington and Kukumseri in F_2 depending on the source of resistance gene in male parent and whether it was susceptible or resistant.

Data presented in Table 16D shows that in this cross female parent was resistant and male parent was susceptible. In F_2 this cross showed 3 Resistant:1 Susceptible segregation ratio.

4.1.3 Variation in expression under 0-5 scale

In the present study resistant and susceptible plants were scored using the 0-5 scale for white rust as indicated in section 3.4.1 and Fig.1. For the test of goodness of fit to specific F_2 ratios plants under the grades **0** to **2** were classified as resistant and under **3** to **5** grades as susceptible.

Table 16. Variation in the expression of resistance to white rust in different crosses.

Cross No./ Parents	Disease reactions of different generations								
	P_1	P_2	F_1	F_2					
				Location	R	S	Ratio	χ^2	P-range
A.									
1. (S-II-2xS-III-2)	R	R	R	Wellington	70	22	13:3	2.577	0.10-0.20
				Kukumseri	49	7	13:3	0.682	0.30-0.50
				Delhi	190	31	13:3	5.858	0.01-0.02
2. (S-II-3xS-IV-2)	R	R	R	Wellington	94	28	3:1	0.175	0.50-0.70
				Kukumseri	40	15	3:1	0.055	0.80-0.90
				Delhi	41	-	-	-	-
					81	29	3:1	0.048	0.80-0.90
3. (S-II-6xS-VII-2)	R	R	R	Wellington	70	27	3:1	0.278	0.50-0.70
				Kukumseri	37	15	3:1	0.231	0.50-0.70
				Delhi	117	-	-	-	-
					85	29	3:1	0.000	1.00
4. (S-II-7xS-VIII-2)	R	S	R	Wellington	62	26	3:1	0.755	0.30-0.50
				Kukumseri	38	18	3:1	1.167	0.20-0.30
				Delhi	170	67	3:1	1.183	0.20-0.30
5. (S-II-9xS-X-2)	R	R	R	Wellington	52	31	3:1	6.110	0.01-0.02
				Kukumseri	42	20	3:1	1.376	0.20-0.30
				Delhi	78	8	15:1	0.896	0.30-0.50
B.									
6. (S-III-2xS-IV-3)	R	R	R	Wellington	66	24	13:3	3.210	0.05-0.10
				Kukumseri	41	12	13:3	0.302	0.50-0.70
				Delhi	227	49	13:3	0.320	0.50-0.70
7. (S-III-9xS-X-3)	R	S	S	Wellington	22	48	1:3	1.210	0.20-0.30
				Kukumseri	9	32	1:3	0.070	0.70-0.80
				Delhi	45	117	1:3	0.530	0.30-0.50

Contd...

Cross No./ Parents	Disease reactions of different generations								
	P_1	P_2	F_1	F_2					
				Location	R	S	Ratio	χ^2	P-range
C.									
8. (S-IV-6xS-VII-4)	R	S	R	Wellington	63	26	3:1	0.633	0.30-0.50
				Kukumseri	35	15	3:1	0.427	0.50-0.70
				Delhi	118	41	3;1	0.019	0.80-0.90
9. (S-IV-8xS-IX-4)	S	R	R	Wellington	93	30	3:1	0.003	0.95-0.98
				Kukumseri	35	16	3:1	0.791	0.30-0.50
				Delhi	153	44	3:1	0.611	0.30-0.50
10. (S-IV-9xS-X-4)	S	R	R	Wellington	21	73	1:3	0.227	0.50-0.70
				Kukumseri	16	40	1:3	0.215	0.50-0.70
				Delhi	182	54	3:1	0.457	0.30-0.50
11. (S-IV-10xPusa Bold)	R	S	R	Wellington	65	29	3:1	1.419	0.20-0.30
				Kukumseri	35	17	3:1	1.256	0.20-0.30
				Delhi	163	49	3:1	0.308	0.50-0.70
D.									
12. (S-VI-8xS-IX-6)	R	S	R	Wellington	53	22	3:1	0.537	0.30-0.50
				Kukumseri	36	16	3:1	0.641	0.30-0.50
				Delhi	127	54	3:1	2.005	0.10-0.20

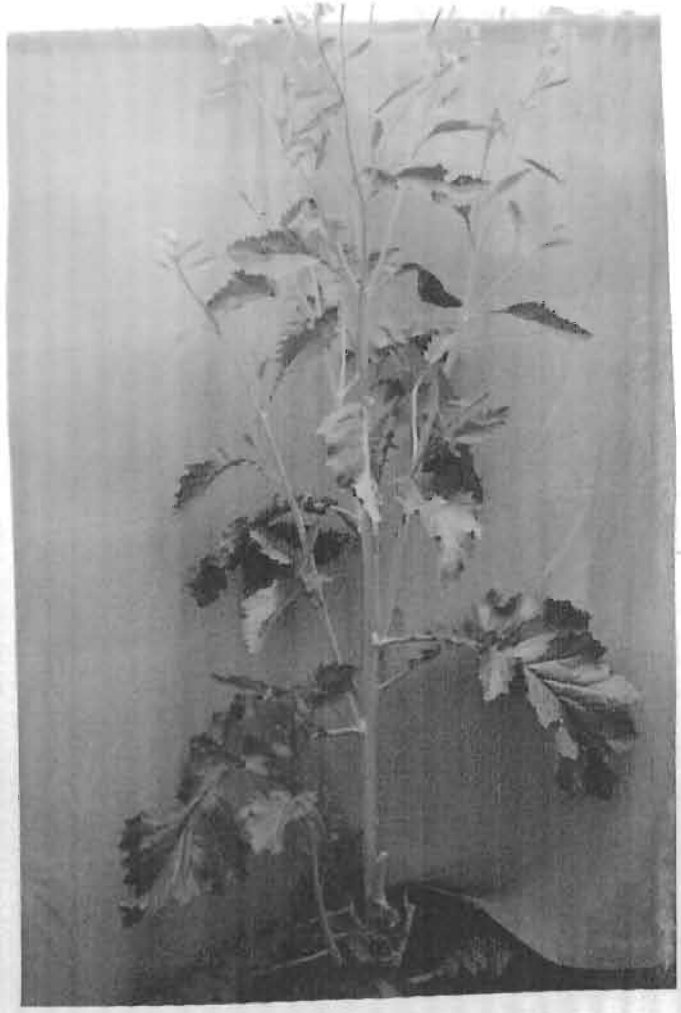
Fig.9: A representative white rust resistant plant of female parental strain S-II used in Cross Nos. 1, 2, 3, 4 and 5

Fig.10: A representative white rust resistant plant of female parental strain S-III used in Cross Nos. 6 and 7

Fig.11: Resistant and susceptible plants of strain IV used as female parent in four different crosses

a. A resistant plant of the strain IV used as female parent in Cross No. 8 and 11

b. A susceptible plant of the strain IV used as female parent in Cross No. 9 and 10



a

b

Two crosses showed same 13R:3S segregation ratio in F_2 (Table 17). But at different locations there is wide variation in percentage of resistant and susceptible plants under different disease scoring grades. At Wellington, both the crosses showed high percentage of resistant plant under grade **1**. At Kukumseri, cross No.1 showed highest percentage of resistant plants under grade **0** but in cross No.6 highest percentage of resistant plants were under grade **1**. At Delhi, both the crosses showed highest percantage of resistant plants under grade **0**. Likewise, percentage of susceptible plants were about equal at Wellington under grades **3,4** and **5**, but at Kukumseri under grade **4** and at Delhi the highest percentage of susceptible plants were under grade **3** in cross No.1. In cross No. 6, the highest percentage of susceptible plants were under grade **5** at Wellington and Delhi but under grade **4** at Kukumseri. Although, over all locations the highest percentage of resitant plants were under the grade **0** in both the crosses and percentages of susceptible plants were higher under grade **3** in cross No.1 and under grade **5** in cross No. 6.

It indicates that depending on the availability of congenial ecological conditions and differentiation in races of pathogen, the plants under one grade can change into a comparatively low resistance or more susceptible grade.

Same pattern of variation in the percentage of resistance and susceptible plants under different grades at different locations were observed for the crosses showing same segregation ratio in F_2 at different locations are given in Table 18 and 19.

4.1.4. Association of leaf waxiness with white rust resistance

Leaf waxiness is an important qualitative trait in *B. napus* and *B. carinata* which mechanically prevents the establishment of disease spores.

Table 17. Distribution of resistant and susceptible plants for white rust scored in 0 to 5 scale for crosses showing similar segregation ratios in F_2 at three locations.

Cross No.	Segregation Ratio (R : S)	Location	Frequency of Resistant plants under different grades				Frequency of susceptible plants under different grades			
			0	1	2	Total	3	4	5	Total
1.	13:3	Wellington	25	41	4	70	7	8	7	22
			(35.7)	(58.6)	(5.7)		(31.8)	(36.4)	(31.8)	
		Kukumseri	42	7	0	49	1	4	2	7
			(85.7)	(14.3)	(0.0)		(14.3)	(57.1)	(28.6)	
		Delhi	155	18	17	190	30	0	1	31
			(81.6)	(9.5)	(8.9)		(96.8)	(0.0)	(3.2)	
		Over locations	222	66	21	309	38	12	10	60
			(71.8)	(21.4)	(6.8)		(63.3)	(20.0)	(16.7)	
6.	13:3	Wellington	5	54	7	66	5	3	16	24
			(7.6)	(81.8)	(10.6)		(20.8)	(12.5)	(66.7)	
		Kukumseri	6	27	8	41	3	6	3	12
			(14.6)	(65.9)	(19.5)		(25.0)	(50.0)	(25.0)	
		Delhi	212	15	8	235	20	11	18	49
			(90.2)	(6.4)	(3.4)		(40.8)	(22.4)	(36.8)	
		Over locations	223	96	23	342	28	20	37	85
			(65.2)	(28.1)	(6.7)		(32.9)	(23.5)	(43.6)	

Figures in parenthesis indicate percent plants

Table 18. Distribution of resistant and susceptible plants for white rust scored in 0 to 5 scale for crosses showing similar segregation ratios in F_2 at three locations.

Cross No.	Segregation Ratio (R : S)	Location	Frequency of Resistant plants under different grades				Frequency of susceptible plants under different grades			
			0	1	2	Total	3	4	5	Total
2.	3:1	Wellington	13	22	59	94	8	14	6	28
			(13.8)	(23.4)	(62.8)		(28.6)	(50.0)	(21.4)	
		Kukumseri	9	23	8	40	2	8	5	15
			(22.5)	(57.5)	(20.0)		(13.3)	(53.4)	(33.3)	
		Delhi	70	6	5	81	13	7	9	29
			(86.4)	(7.4)	(6.2)		(44.8)	(24.1)	(31.1)	
	True Breeding	Delhi	27	12	2	41	-	-	-	-
			(65.9)	(29.3)	(4.8)					
		Over locations	119	63	74	256	23	29	20	72
			(46.5)	(24.6)	(28.9)		(31.9)	(40.3)	(27.8)	
3.	3:1	Wellington	29	35	6	70	8	9	10	27
			(41.4)	(50.0)	(8.6)		(29.6)	(33.3)	(37.1)	
		Kukumseri	12	22	3	37	5	7	3	15
			(32.4)	(59.5)	(8.1)		(33.3)	(46.7)	(20.0)	
		Delhi	67	8	10	85	10	12	7	29
			(78.8)	(9.4)	(11.8)		(34.5)	(41.4)	(24.1)	
	True Breeding	Delhi	115	2	0	117	-	-	-	-
			(98.3)	(1.7)	(0.0)					
		Over locations	223	67	19	309	23	28	20	71
			(72.2)	(21.7)	(6.1)		(32.4)	(39.4)	(28.2)	

Figures in parenthesis indicate percent plants

Cross No.	Segregation Ratio (R : S)	Location	Frequency of Resistant plants under different grades				Frequency of susceptible plants under different grades			
			0	1	2	Total	3	4	5	Total
4.	3:1	Wellington	17	40	5	62	6	4	16	26
			(27.4)	(64.5)	(8.1)		(23.1)	(15.4)	(61.5)	
		Kukumseri	15	a18	5	38	1	4	13	18
			(39.5)	(47.4)	(13.1)		(5.6)	(22.2)	(72.2)	
		Delhi	137	17	16	170	18	18	31	67
			(80.6)	(10.0)	(94)		(26.9)	(26.9)	(46.2)	
		Over locations	169	75	26	270	25	26	60	111
			(62.6)	(27.8)	(9.6)		(22.5)	(23.4)	(54.1)	
8.	3:1	Wellington	12	19	32	63	7	9	10	26
			(19.0)	(30.2)	(50.8)		(26.9)	(34.6)	(38.5)	
		Kukumseri	9	23	3	35	3	9	3	15
			(25.7)	(65.7)	(8.6)		(20.0)	(60.0)	(20.0)	
		Delhi	106	7	5	118	19	10	12	41
			(89.8)	(5.9)	(4.3)		(46.3)	(24.4)	(29.3)	
		Over locations	127	49	40	216	29	28	25	82
			(58.8)	(22.7)	(18.5)		(35.4)	(34.1)	(30.5)	
9.	3:1	Wellington	30	61	2	93	8	7	15	30
			(32.3)	(65.6)	(2.1)		(26.7)	(23.3)	(50.0)	
		Kukumseri	12	19	4	35	1	6	9	16
			(34.3)	(54.3)	(11.4)		(6.3)	(37.5)	(56.2)	
		Delhi	101	21	31	153	16	20	8	44
			(66.0)	(13.7)	(20.3)		(36.4)	(45.4)	(18.2)	
		Over locations	143	101	37	281	25	33	32	90
			(50.9)	(35.9)	(13.2)		(27.8)	(36.7)	(35.5)	

Table 18. contd...

Cross No.	Segregation Ratio (R : S)	Location	Frequency of Resistant plants under different grades				Frequency of susceptible plants under different grades			
			0	1	2	Total	3	4	5	Total
11.	3:1	Wellington	19	33	13	65	6	15	8	29
			(29.2)	(50.8)	(20.0)		(20.7)	(51.7)	(27.6)	
		Kukumseri	10	20	5	35	5	5	7	17
			(28.6)	(57.1)	(14.3)		(29.4)	(29.4)	(41.2)	
		Delhi	117	20	26	163	16	11	22	49
			(71.8)	(12.3)	(15.9)		(32.8)	(22.4)	(44.8)	
		Over locations	146	73	44	263	27	31	37	95
			(55.5)	(27.8)	(16.7)		(28.4)	(32.6)	(39.0)	
12.	3:1	Wellington	0	48	5	53	3	4	15	22
			(0.0)	(90.6)	(9.4)		(13.6)	(18.2)	(68.2)	
		Kukumseri	13	20	3	36	3	7	6	16
			(36.1)	(55.6)	(8.3)		(18.8)	(43.8)	(37.4)	
		Delhi	71	27	29	127	45	7	2	54
			(55.9)	(21.3)	(22.8)		(83.3)	(13.0)	(3.7)	
		Over locations	84	95	37	216	51	18	23	92
			(38.9)	(44.0)	(17.1)		(55.4)	(19.6)	(25.0)	

Figures in parenthesis indicate percent plants

Table 19. Distribution of resistant and susceptible plants for white rust scored in 0 to 5 scale for crosses showing similar segregation ratios in F_2 at three locations.

Cross No.	Segregation Ratio (R : S)	Location	Frequency of Resistant plants under different grades				Frequency of susceptible plants under different grades			
			0	1	2	Total	3	4	5	Total
7.	1:3	Wellington	0	12	10	22	26	12	10	48
			(0.0	(54.6)	(45.4)		(54.2)	(25.0)	(20.8)	
		Kukumseri	0	7	2	9	2	7	23	32
			(0.0)	(77.8)	(22.2)		(6.3)	(21.9)	(71.8)	
		Delhi	9	7	29	45	55	16	46	117
			(20.0)	(15.6)	(64.4)		(47.0)	(13.7)	(39.3)	
		Over locations	9	26	41	76	83	35	79	197
			(11.8)	(34.2)	(54.0)		(42.1)	(17.8)	(40.1)	

Figures in parenthesis indicate percent plants

In the process of incorporating disease resistance from *B. napus* and *B. carinata* to *B. juncea* the leaf waxiness was also incorporated to the parental lines of the present study. Any one out of two parents involved in the 12 selected cross combinations in the present study was waxy but with varying intensity.

Data presented in the Tables 20, 21, 22, 23 and 24 indicates that all the plants in F_1 generations of these five crosses were waxy and resistant except cross No.7 given in Table 23 where waxy plants were also susceptible (Fig. 12a). In the segregating generations viz., F_2, F_3, B_1 and B_2 for almost all five crosses, percentage of resistant plants were high under waxy category. Although non-waxy plants were also showing resistance to white rust in these crosses the frequency was comparatively low. It indicates that white rust resistance was enhanced by leaf waxiness in these crosses.

On the other hand, the data presented in Tables 25 to 31 shows that all the plants in F_1 generation were resistant and waxy indicating the involvement of one waxy parent in all these crosses. Segregating generations viz., F_2, F_3, B_1 and B_2 for almost all the crosses showed equal percentage of resistant plants under waxy and non-waxy categories (Fig.12b). It indicates that white rust resistance is not dependent on waxiness in these crosses because of low intensity of waxiness and nature of resistant gene involved in non-waxy plants which show resistance even without being associated with waxiness.

4.1.5 Association of yellow seed colour with white rust resistance

In the present study an attempt was made to study the association of yellow seed colour (Fig. 13) with white rust resistance because yellow seed

Table 20. Association of leaf waxiness with white rust resistance for different generations in Cross No. 5 (S-II-9xS-X-2).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	17	-	100	-	-	-
P_2	R	-	-	-	20	-	100
F_1	R	29	-	100	-	-	-
F_2	Seg.	50	-	100	28	8	77.8
F_3(D)	Seg.	80	5	94.1	18	9	66.7
F_3(W)	R	24	-	100	3	-	100
B_1	-	-	-	-	-	-	-
B_2	-	-	-	-	-	-	-

Table 21. Association of leaf waxiness with white rust resistance for different generations in Cross No. 6 (S-III-3xS-IV-3).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	18	-	100	-	-	-
P_2	R	-	-	-	21	-	100
F_1	R	29	-	100	-	-	-
F_2	Seg.	166	27	86.0	61	22	73.5
F_3(D)	Seg.	71	-	100	33	5	86.8
F_3(W)	R	15	-	100	4	-	100
B_1	R	32	-	100	-	-	-
B_2	R	12	-	100	12	-	100

Table 22. Association of leaf waxiness with white rust resistance for different generations in Cross No. 7 (S-III-9xS-X-3).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	18	-	100	-	-	-
P_2	S	-	-	-	-	16	0.0
F_1	S	-	19	0.0	-	-	-
F_2	Seg.	31	42	42.5	14	75	15.7
F_3(D)	Seg.	19	14	57.6	2	13	13.3
F_3(W)	Seg.	24	14	63.2	-	-	
B_1	Seg.	10	8	55.6	-	-	-
B_2	S	-	8	0.0	-	14	0.0

Table 23. Association of leaf waxiness with white rust resistance for different generations in Cross No. 9 (S-IV-8xS-IX-4).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	S	-	-	-	-	23	0.0
P_2	R	19	-	0.0	-	-	-
F_1	R	23	-	100.0	-	-	-
F_2	Seg.	97	19	83.6	56	25	69.1
F_3(D)	Seg.	35	5	87.5	45	10	81.8
F_3(W)	Seg.	32	2	94.1	23	8	74.2
B_1	-	-	-	-	-	-	-
B_2	-	-	-	-	-	-	-

Table 24. Association of leaf waxiness with white rust resistance for different generations in Cross No. 11 (S-IV-10x Pusa Bold).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	20	-	100	-	-	-
P_2	S	-	-	-	-	23	0.0
F_1	R	21	-	100	-	-	-
F_2	Seg.	101	21	82.8	62	28	68.9
F_3(D)	Seg.	54	11	83.1	15	10	60.0
F_3(W)	Seg.	40	-	100	8	27	22.9
B_1	R	25	-	100	-	-	100
B_2	Seg.	12	10	54.5	13	11	54.2

Table 25. Association of leaf waxiness with white rust resistance for different generations in Cross No. 1 (S-II-2xS-III-2).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	24	-	100	-	-	-
P_2	R	-	-	-	22	-	100
F_1	R	32	-	100	-	-	-
F_2	Seg.	152	22	87.4	38	9	80.9
F_3(D)	Seg.	59	15	79.7	36	16	69.2
F_3(W)	Seg.	68	-	100	19	6	76.0
B_1	R	20	-	100	-	-	-
B_2	R	9	-	100	9	-	100

Table 26. Association of leaf waxiness with white rust resistance for different generations in Cross No. 2 (S-II-3xS-IV-2).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	-	-	-	24	-	100
P_2	R	20	-	100	-	-	-
F_1	R	19	-	100	-	-	-
F_2	Seg.	89	18	83.2	33	11	75.0
F_3(D)	Seg.	67	20	77.0	21	11	65.6
F_3(W)	Seg.	80	14	85.1	12	5	70.6
B_1	R	10	-	100	11	-	100
B_2	R	54	-	100	-	-	-

Table 27. Association of leaf waxiness with white rust resistance for different generations in Cross No. 3 (S-II-6xS-VII-2).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	-	-	-	26	-	100
P_2	R	23	-	100	-	-	-
F_1	R	21	-	100	-	-	-
F_2	Seg.	160	20	88.9	42	9	82.4
F_3(D)	Seg.	77	12	86.5	21	2	91.3
F_3(W)	R	73	-	100	19	-	100
B_1	R	10	-	100	12	-	100
B_2	R	35	-	100	-	-	-

Table 28. Association of leaf waxiness with white rust resistance for different generations in Cross No. 4 (S-II-7xS-VIII-2).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	19	-	100	-	-	-
P_2	S	-	-	-	-	17	0.0
F_1	R	21	-	100	-	-	-
F_2	Seg.	128	42	75.3	42	25	62.7
F_3(D)	Seg.	74	9	89.2	26	13	66.7
F_3(W)	Seg.	54	7	88.5	19	7	73.1
B_1	R	24	-	100	-	-	-
B_2	Seg.	8	8	50.0	9	7	56.3

Table 29. Association of leaf waxiness with white rust resistance for different generations in Cross No. 8 (S-IV-6xS-VII-4).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	-	-	-	18	-	100
P_2	S	-	22	0.0	-	-	-
F_1	R	22	-	100	-	-	-
F_2	Seg.	58	17	77.3	60	24	71.4
F_3 (D)	Seg.	29	8	78.4	30	9	76.9
F_3 (W)	R	3	-	100	-	-	-
B_1	R	13	-	100	10	-	100
B_2	Seg.	25	18	58.1	-	-	-

Table 30. Association of leaf waxiness with white rust resistance for different generations in Cross No. 10 (S-IV-9xS-X-4).

Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	S	-	-	-	-	18	0.0
P_2	R	16	-	100	-	-	-
F_1	R	34	-	100	-	-	-
F_2	Seg.	128	31	80.5	54	23	70.1
F_3(D)	Seg.	19	6	76.0	19	7	73.1
F_3(W)	Seg.	12	3	80.0	8	5	61.5
B_1	Seg.	8	7	53.3	10	9	52.6
B_2	R	22	-	100	-	-	-

Table 31. Association of leaf waxiness with white rust resistance for different generations in Cross No. 12 (S-VI-8xS-IX-6).

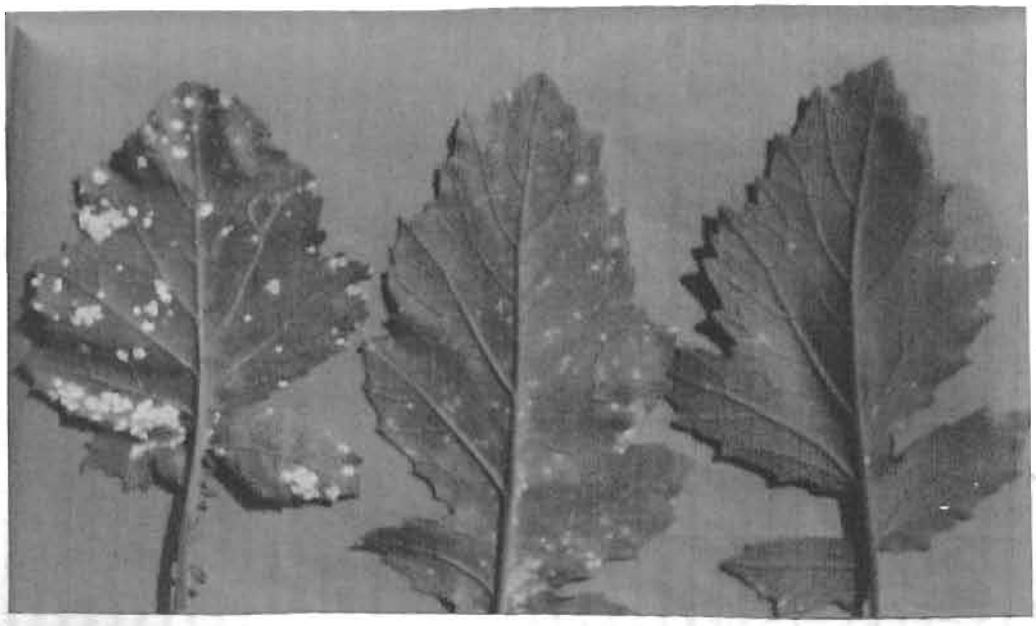
Generation	Disease reaction	Waxy			Non-waxy		
		Frequency		%	Frequency		%
		R	S	R-plants	R	S	R-plants
P_1	R	23	-	100	-	-	-
P_2	S	-	-	-	-	20	0.0
F_1	R	21	-	100	-	-	-
F_2	Seg.	94	35	72.9	33	19	63.5
F_3(D)	Seg.	75	21	78.1	21	6	77.8
F_3(W)	R	7	-	100	-	-	-
B_1	R	34	-	100	-	-	-
B_2	Seg.	10	1	90.9	5	11	31.3

Fig.12: Leaves showing the association of leaf waxiness with white rust resistance

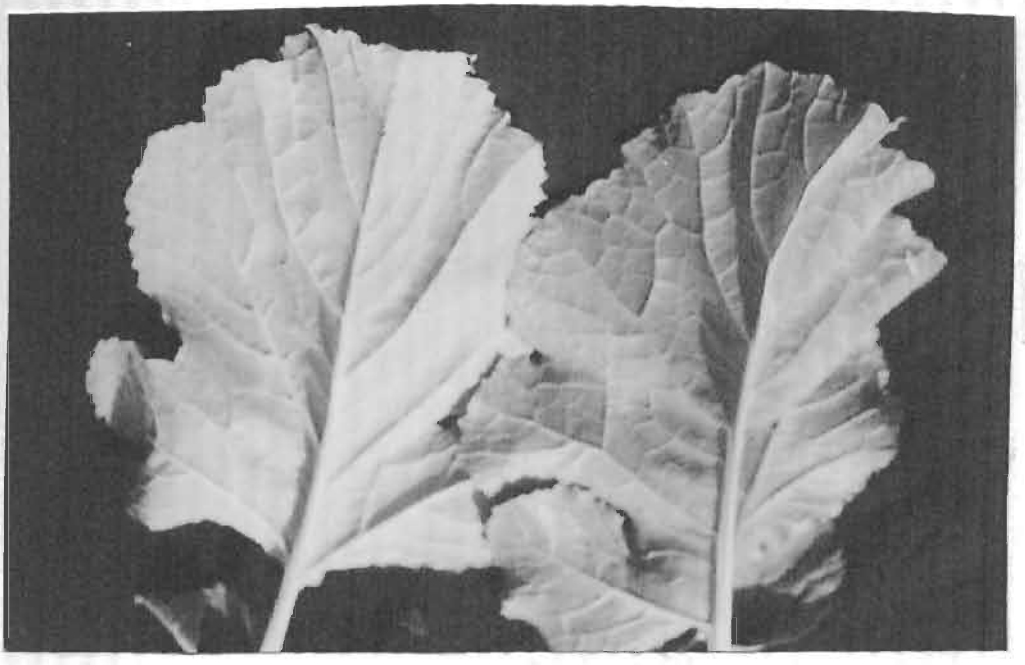
a. Waxy leaf (extreme right) showing the resistance to white rust and non-waxy leaves with prominent white rust pustules

b. Waxy resistant leaf (left) and non-waxy resistant leaf (right) showing complete absence of white rust pustules

Fig.13: Seeds with yellow seed coat colour (left) and brown seed coat colour (right). These are two extreme seed colours but there is a gradation between two in a segregating generation



a



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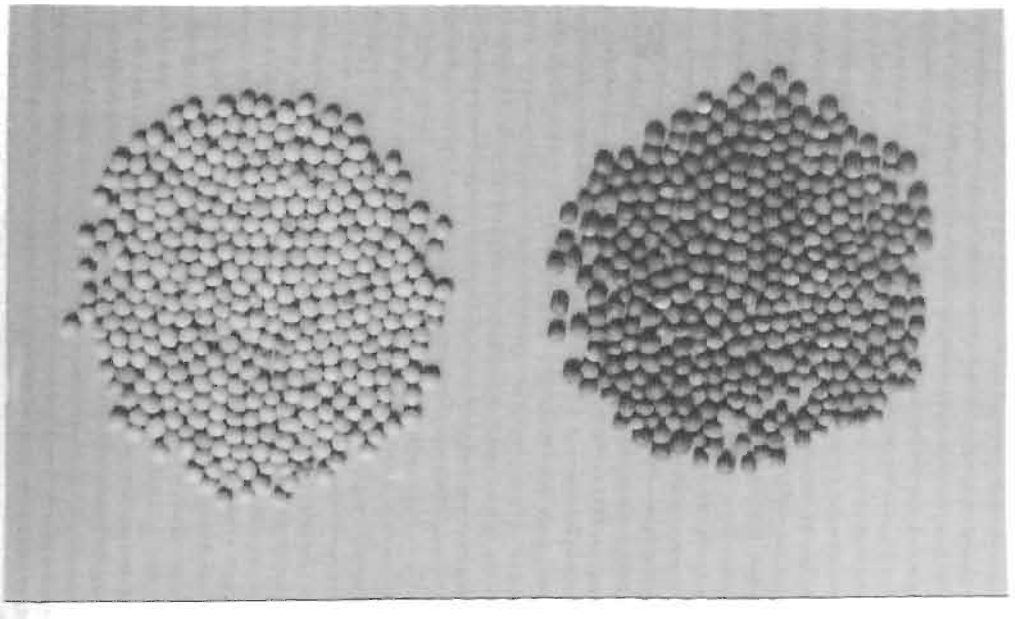


coat colour could be used as a marker to identify the resistant plant in the segregating population and also yellow seeds yield high oil percent because of low crude fibres. Data presented in Table 32 shows that three crosses involving one yellow seeded parent had all resistant plants with brown seed in F_1 generation except in cross No.7 where all the F_1 plants were susceptible. In cross No.7 and 12 the original yellow seeded parent was susceptible and only in cross No.10 it was resistant. F_2 and backcrosses populations segregation ratio showed that the percentage of brown seeded and yellow seeded resistant plants were almost equal in all three crosses. It indicates that there was no association between yellow seed colour and white rust resistance in the material used in the present study.

4.2 Estimation of gene effects

A knowledge of the nature of genetic variation and mode of inheritance of complex quantitative trait is essential to assess genetic potential and formulate effective selection strategy. Thus, the examination of the nature and magnitude of gene effects of quantitative traits is one such important exercise for which generation mean analysis was performed following procedure given by Hayman (1958).

The gene effects were estimated for traits such as days to flowering (DF), days to maturity (DM), plant height (PH), number of primary branches (PB), number of secodary branches (SB), length of main fruiting axis (LM),siliqua on main fruiting axis (SM), seed yield per plant (SY), 1000-seed weight (SW) and per cent oil content (OC) in 10 selected crosses. The six parameters mean effect (**m**), additive (**d**), dominance (**h**), additive x additive (**i**), additive x dominance or vice-versa (**j**) and dominance x dominance epistasis (**l**) were estimated in these 10 selected crosses. The gene effects along with their standard errors and significance are given in Tables 33 to 42 and are described below characterwise.

Table 32. Association of yellow seed colour with white rust resistance in some crosses involving a yellow seeded parent.

Cross No./ Parents	Seed colour	Brown			Yellow		
	Disease reaction	Frequency		%	Frequency		%
	Generation	R	S	R-plants	R	S	R-plants
7.	P_1	18	-	100.0	-	-	-
(S-III-9xS-X-3)	P_2	-	-	-	-	16	0.0
	F_1	-	19	0.0	-	-	-
	F_2	40	105	27.6	5	12	29.4
	B_1	10	8	55.6	-	-	-
	B_2	-	10	0.0	-	12	0.0
10.	P_1	-	18	0.0	-	-	-
(S-IV-9xS-X-4)	P_2	-	-	-	16	-	100.0
	F_1	34	-	100.0	-	-	-
	F_2	161	48	77.0	21	6	77.8
	B_1	18	16	52.9	-	-	-
	B_2	10	-	100.0	12	-	100.0
12.	P_1	23	-	100.0	-	-	-
(S-VI-8xS-IX-6)	P_2	-	-	-	-	20	0.0
	F_1	21	-	100.0	-	-	-
	F_2	89	32	73.5	38	16	70.4
	B_1	34	-	100.0	-	-	-
	B_2	8	4	66.7	7	8	46.7

Days to flowering:

The data from Table 33 indicates that additive (**d**), dominance (**h**), additive x additive (**i**) and dominance x dominance (**l**) interaction components made significant contribution for expression of this trait in three crosses, such as in cross No.2 additive (**d**) and dominance effects were significant, in cross No.6 epistatic components **i** and **l** were significant and in cross No.8 additive (**d**) and dominance x dominance (**l**) effects were significant. In only cross No.6, **l** was with negative sign and significant rest crosses were significant with positive sign. From the sign of **h** and **l** it was noticed that complementary epistasis is operating in only cross No. 10 and 12 which will ease the selection of this trait. In rest of the crosses, duplicate epistasis was observed.

Days to maturity:

The dominance (**h**) effect and additive x additive (**i**) interaction was observed to be important contributor for variation of this trait in cross No.1 and 12 only, **h** and **i** are negative with higher magnitude in cross No.12 and lower magnitude in cross No.1 (Table 34). Epistasis seems to be of duplicate type in most of the crosses except cross No.7 and 10 which shows complementary epistasis.

Plant height:

The data presented in Table 35 indicates that all types of gene actions viz., **d, h, i, j** and **l** made significant contribution for expression of this trait. In cross No. 1, 3 and 10, **d, h, i** and **l** were significant and negative but in cross No.3 & 10, **d** and **l** were positive and in cross No.1, only **l** was positive. In cross No.2 **d, h** and **i** were positive and highly significant. In cross no.4,

Table 33. Estimates of gene effects for days to flowering in different crosses.

Cross No.	$\hat{m}$	$\hat{d}$	$\hat{h}$	$\hat{i}$	$\hat{j}$	$\hat{l}$
1.	52.33** ± 0.33	-1.67 ± 2.11	6.33 ± 4.55	6.00 ± 4.42	-1.67 ± 2.20	-11.33 ± 8.81
2.	54.33** ± 0.33	5.33* ± 2.40	5.00* ± 2.40	4.00 ± 4.99	4.33 ± 2.52	-6.00 ± 10.18
3.	54.00** ± 0.58	1.33 ± 1.33	2.50 ± 3.78	2.67 ± 3.53	0.50 ± 1.62	-0.99 ± 6.42
4.	55.00** ± 0.58	-0.33 ± 3.35	-1.83 ± 7.24	-2.00 ± 7.09	-2.50 ± 3.52	3.67 ± 13.92
6.	54.67** ± 0.33	-2.00 ± 1.37	3.83 ± 3.39	6.67* ± 3.06	0.50 ± 1.81	-17.00* ± 6.38
7.	57.33** ± 1.20	0.67 ± 1.45	-5.00 ± 6.04	-4.00 ± 5.62	0.00 ± 2.45	4.67 ± 8.74
8.	54.67** ± 0.88	3.33** ± 1.20	-1.33 ± 4.30	-1.33 ± 4.27	-1.00 ± 1.28	13.33* ± 6.06
10.	56.67** ± 2.03	-1.33 ± 1.76	-6.00 ± 8.91	-1.33 ± 8.84	-0.33 ± 1.94	-1.33 ± 10.95
11.	53.33** ± 0.88	-3.00 ± 3.04	1.67 ± 7.13	6.00 ± 7.02	-4.67 ± 3.15	-7.33 ± 12.88
12.	59.33** ± 0.33	-5.00* ± 2.16	-3.17 ± 4.80	0.67 ± 4.52	-2.50 ± 2.33	-9.67 ± 9.32

**, ** : significant at 5% and at 1% level, respectively.*

Table 34. Estimates of gene effects for days to maturity in different crosses.

Cross No.	^ m	^ d	^ h	^ i	^ j	^ l
1.	143.67** ±0.33	-0.67 ± 0.94	5.33* ± 2.45	5.33* ± 2.31	-0.67 ± 1.05	-6.67 ± 4.32
2.	145.67** ±0.33	1.00 ± 1.97	0.67 ± 4.42	0.67 ± 4.16	0.67 ± 2.16	-1.33 ± 8.54
3.	145.67** ±0.67	0.33 ± 0.88	3.50 ± 3.27	2.00 ± 3.20	0.50 ± 0.96	-5.67 ± 4.63
4.	146.33** ±0.67	0.67 ± 1.33	-3.17 ± 3.85	-2.67 ± 3.77	0.17 ± 1.54	5.00 ± 6.16
6.	146.00** ±0.58	-1.67 ± 0.88	2.00 ± 3.02	3.33 ± 2.91	1.67 ± 1.00	-6.67 ± 4.52
7.	146.00** ±1.15	0.67 ± 0.67	1.17 ± 4.87	1.33 ± 4.81	0.17 ± 0.87	1.67 ± 5.57
8.	145.00** ±0.00	0.67 ± 0.94	0.00 ± 2.13	0.00 ± 1.89	-0.33 ± 1.20	1.33 ± 4.27
10.	146.33** ±1.45	0.00 ± 2.05	-2.00 ± 7.19	0.00 ± 7.12	1.00 ± 2.11	-4.00 ± 10.26
11.	144.67** ±0.33	-2.33 ± 1.20	3.67 ± 2.85	4.67 ± 2.75	-2.67 ± 1.25	-7.33 ± 5.21
12.	149.00** ±0.00	-1.00 ± 0.94	-9.50** ±2.29	-8.67** ±1.89	-0.17 ± 1.12	7.00 ± 4.58

**, ** : significant at 5% and at 1% level, respectively.*

Table 35. Estimates of gene effects for plant height (cm) in different crosses.

Cross No.	$\hat{m}$	$\hat{d}$	$\hat{h}$	$\hat{i}$	$\hat{j}$	$\hat{l}$
1.	219.57** ± 5.43	-17.93** ± 4.91	-86.68** ± 24.02	-88.93** ± 23.85	-3.62 ± 5.26	72.17* ± 29.85
2.	212.67** ± 3.01	20.77** ± 6.10	63.42** ± 17.29	43.67* ± 17.14	0.88 ± 6.18	-52.97 ± 27.60
3.	227.67** ± 1.01	14.23* ± 6.54	-63.15** ± 17.26	-65.40** ± 13.70	-0.12 ± 6.69	123.63** ± 33.80
4.	228.13** ± 2.38	-5.97 ± 3.82	-89.97** ± 12.60	-84.20** ± 12.22	-35.30** ± 3.89	103.00** ± 19.04
6.	230.53** ± 1.47	-44.77** ± 6.22	-88.92** ± 14.51	-85.13** ± 13.76	-37.18** ± 7.43	125.17** ± 27.18
7.	211.90** ± 1.08	-0.43 ± 5.67	-33.28* ± 13.49	-25.67 ± 12.13	-4.22 ± 5.93	58.70* ± 25.94
8.	214.40** ± 2.91	13.13 ± 10.92	47.22 ± 24.87	26.80 ± 24.75	8.48 ± 11.04	-80.57 ± 45.45
10.	246.43** ± 1.11	11.53* ± 5.36	-103.05** ± 13.91	-113.87** ± 11.60	6.78 ± 6.06	112.90** ± 26.74
11.	202.13** ± 2.47	4.07 ± 31.68	19.90 ± 13.57	21.33 ± 12.31	-9.60* ± 4.18	7.27 ± 21.08
12.	249.27** ± 0.52	16.33* ± 7.88	-59.05** ± 16.47	-71.07** ± 15.90	18.92* ± 8.23	40.77 ± 32.74

*, **: *significant at 5% and at 1% level, respectively.*

h, i, j and **l** were highly significant but **l** was only positive, in cross No.6, all **d, h, i, j** and **l** were negative and highly significant but only **l** was positive, in cross No.7, **h** and **l** were significant with low magnitude and **h** was negative but **l** was positive, in cross No.11, only **j** was significant and negative with low magnitude, and in cross No.12 **d, h, i, j** gene effects were observed significant but **h** and **i** were negative with higher magnitude and **d** and **j** were positive with low magnitude. Epistasis seems to be duplicate type almost in all the crosses except cross No.11 which shows complementary type epistasis.

Number of primary branches:

Data from the Table 36 indicated that dominance (**h**), additive x additive (**i**) and dominance x dominance (**l**) interaction made significant contribution for expression of this trait in most of the crosses. In cross No. 1 and 12, **h** and **i** gene effects were positive and significant, in cross No.2 and 8, **h, i** and **l** were significant both positive and negative, in cross number 4 and 10, **h** was positive and significant, in cross No.6, **j** was negative and signifiant, in cross No.7, **l** was positive and significant, in cross No.11 all the types of gene actions were operating significantly as **d, j** and **l** negative and **h** and **i** positive sign. Epistasis is observed as duplicate type in almost all the crosses except cross No.3 which shows complementry type.

Number of secondary branches

It was observed from Table 37 that in cross no.1, 2, 8, 10 and 12, the **h, i** and **l** were significant with higher magnitude and both positive and negative. In cross No.3 and 4, **h** and **i** were significant and positive but **h** has higher magnitude than **i**, in cross no.6, **j** was negative and significant, in cross no.7, **h** was negative and significant and **l** was positive and

Table 36. Estimates of gene effects for number of primary branches in different crosses.

Cross No.	$\hat{m}$	$\hat{d}$	$\hat{h}$	$\hat{i}$	$\hat{j}$	$\hat{l}$
1.	8.47** ± 0.12	-0.33 ± 0.42	6.27** ± 1.60	5.20** ± 0.97	0.19 ± 0.83	-3.20 ± 3.10
2.	11.87** ± 0.13	-0.13 ± 0.84	-15.47** ± 1.79	-17.33** ± 1.77	1.53 ± 0.87	22.93** ± 3.47
3.	7.83** ± 0.08	-0.70 ± 0.75	1.55 ± 1.69	1.13 ± 1.54	-1.05 ± 0.89	5.30 ± 3.33
4.	7.23** ± 0.16	0.00 ± 0.92	6.70** ± 2.06	3.73 ± 1.93	-1.00 ± 0.93	-3.80 ± 4.00
6.	7.77** ± 0.23	-2.23 ± 1.08	-1.20 ± 2.51	-0.33 ± 2.35	-3.03* ± 1.22	4.67 ± 4.75
7.	9.13** ± 0.43	0.27 ± 0.47	-3.82 ± 2.44	-2.27 ± 1.94	-2.15 ± 1.53	10.30* ± 3.88
8.	6.07** ± 0.19	0.17 ± 1.10	11.10** ± 2.38	11.27** ± 2.32	-0.40 ± 1.19	-11.53* ± 4.58
10.	6.63** ± 0.20	0.47 ± 0.40	4.25** ± 1.22	2.53 ± 1.14	0.08 ± 0.41	-1.43 ± 1.99
11.	6.30** ± 0.38	-1.20* ± 0.40	7.80** ± 1.85	7.60** ± 1.71	-1.97** ± 0.53	-11.20** ± 2.60
12.	5.73** ± 0.58	2.10 ± 1.42	16.27** ± 3.75	14.20** ± 3.66	-0.07 ± 1.43	-12.53 ± 6.35

**, ** : significant at 5% and at 1% level, respectively.*

Table 37. Estimates of gene effects for number of secondary branches in different crosses.

Cross No.	^ m	^ d	^ h	^ i	^ j	^ l
1.	26.23** ± 0.49	-3.99 ± 2.60	54.68** ± 7.82	49.20** ± 5.57	8.33 ± 4.27	-40.70* ± 15.24
2.	50.53** ± 0.62	3.83 ± 3.98	-73.23** ± 8.74	-83.26** ± 8.35	8.93 ± 4.13	114.07** ±16.94
3.	22.47** ± 0.52	-1.23 ± 3.13	35.25** ± 7.13	33.00** ± 6.60	-1.38 ± 3.67	-0.10 ± 13.79
4.	27.07** ± 0.58	-3.87 ± 6.36	48.10** ±13.96	31.73* ± 12.94	-7.80 ± 6.38	-41.93 ± 27.63
6.	28.63** ± 0.49	-11.13 ± 5.61	7.73 ± 12.32	12.27 ± 11.38	-15.37* ± 6.67	-3.20 ± 24.41
7.	39.40** ± 2.41	3.07 ± 4.41	-28.93* ± 13.57	-31.60 ± 13.08	-3.40 ± 5.18	64.80** ±21.39
8.	19.60** ± 2.55	1.10 ± 5.43	72.87** ±16.29	73.40**±14.92	4.97 ± 6.16	-83.60** ±27.35
10.	23.50** ± 1.68	-6.07 ± 3.43	51.02** ±10.34	34.53** ± 9.61	-7.35 ± 3.46	-40.03* ± 17.09
11.	21.23** ± 1.98	-5.93 ± 3.98	45.77** ±11.78	43.33** ±11.23	-10.77* ± 4.32	-63.00** ±19.16
12.	23.93** ± 3.06	5.07 ± 4.92	72.97** ±15.90	64.80** ±15.71	-4.10 ± 4.96	-53.00* ± 23.72

**, ** : significant at 5% and at 1% level, respectively.*

significant with higher magnitude and in cross no. 11, **h**, **j** and **l** were significant but **j** and **l** were negative and **j** had lower magnitude than other three types of gene actions. Duplicate type epistasis was observed in all the crosses.

Length of main fruiting axis

It was observed from Table 38 that in cross No.2, **d**, **h** and **i** types of gene action were significant but **h** and **i** had higher magnitude than **d**, in cross No. 3, **h** and **j** were positive and significant with low magnitude, in cross No.6, **d** was negative and significant, in cross No. 7 and 8 all types of gene actions were operating but **h**; **i** and **l** had higher magnitude than **d** and **j**, and in cross No.10, **d** and **j** were negative and significant. All the crosses were showing duplicate type epistasis.

Number of siliqua on main fruiting axis

Data from Table 39 indicated that in cross No.2, **h** and **i** type of gene action was positive and significant, in cross No.3, only **h** was positive and significant, in cross No.6, **d** was negative and significant, in cross No.7, all types of gene actions were operating but **h** and **i** were negative and **d**, **j** and **l** were positive and significant, in cross No.8, **h**, **i** and **l** were significant but **l** was negative, in cross No.10, only **j** type of gene actions was negative and significant and in cross No.12, **d** and **i** types of gene action were negative and significant. Duplicate type of epistasis was observed in all the crosses except cross No.1.

Seed yield

It was observed from Table 40 that all types of gene actions were operating in different crosses. In cross No.1, **d**, **h** and **i** were significant but

Table 38. Estimates of gene effects for length of main fruiting axis (cm) in different crosses.

Cross No.	^ m	^ d	^ h	^ i	^ j	^ l
1.	71.50** ± 0.96	-8.76 ± 7.45	-13.28 ± 15.67	-14.86 ± 15.39	-7.38 ± 7.77	6.70 ± 30.62
2.	70.63** ± 0.20	8.33* ± 3.19	36.36** ± 6.58	31.46** ± 6.45	5.17 ± 3.24	-10.99 ± 13.09
3.	76.60** ± 1.51	7.30 ± 4.06	29.10* ± 10.57	23.13 ± 10.13	9.50* ± 4.07	-20.47 ± 18.36
4.	71.06** ± 0.58	-3.87 ± 8.26	32.47 ± 17.47	21.73 ± 16.68	-4.00 ± 8.27	-65.07 ± 34.70
6.	81.27** ± 0.87	-11.03* ± 4.39	-6.00 ± 10.36	-11.80 ± 9.44	-6.40 ± 5.53	21.47 ± 19.83
7.	85.73** ± 1.58	4.43* ± 1.82	-68.77** ± 7.69	-62.73** ± 7.30	9.67** ± 2.89	99.67** ±10.81
8.	64.53** ± 0.44	12.10* ± 5.41	81.30** ±11.35	80.47** ±10.97	10.30* ± 5.99	-127.26** ±22.50
10.	85.20** ± 1.31	-13.07* ± 4.57	19.03± 12.22	7.73 ± 10.53	-13.90* ± 4.96	-9.00 ± 22.71
11.	80.50** ± 1.85	-5.07 ± 6.57	-25.58 ± 15.31	-18.53 ± 15.09	-4.78 ± 6.81	28.23 ± 27.80
12.	90.57** ± 1.15	-6.57 ± 7.06	-16.95 ± 15.44	-23.13 ± 14.85	0.02 ± 7.80	14.70 ± 29.85

**, ** : significant at 5% and at 1% level, respectively.*

Table 39. Estimates of gene effects for number of siliqua on main fruiting axis in different crosses.

Cross No.	^ m	^ d	^ h	^ i	^ j	^ l
1.	54.76** ± 0.49	-7.06 ± 4.98	2.05 ± 10.43	-3.86 ± 10.17	-3.48 ± 5.11	4.83 ± 20.57
2.	53.00** ± 0.40	4.10 ± 2.13	34.08** ± 5.40	27.67** ± 4.56	7.12 ± 3.44	-5.89 ± 10.43
3.	60.77** ± 0.67	2.70 ± 4.33	20.60* ± 9.15	17.80 ± 9.07	6.17 ± 4.45	-24.80 ± 17.70
4.	55.83** ± 0.08	1.37 ± 6.98	4.72 ± 14.90	-4.07 ± 13.97	-0.98 ± 7.00	-0.90 ± 29.83
6.	60.80** ± 0.59	-14.27** ± 4.35	-8.00 ± 10.09	-11.60 ± 9.01	-13.13 ± 5.77	39.73 ± 19.77
7.	64.67** ± 1.39	6.63* ± 3.04	-38.78** ± 9.03	-36.07** ± 8.22	10.45* ± 3.84	74.23** ±15.29
8.	49.07** ± 0.68	9.27 ± 5.05	60.70**±11.15	64.40** ±10.47	4.67 ± 5.18	-85.13** ±21.80
10.	64.97** ± 1.03	-8.80 ± 4.74	20.48 ± 10.66	11.87 ± 10.34	-11.08* ± 5.02	-25.63 ± 20.10
11.	58.87** ± 0.43	-4.13 ± 3.96	-7.53 ± 8.51	-2.67 ± 8.11	-8.57 ± 4.54	4.93 ± 16.77
12.	70.20** ± 0.80	-8.63** ± 2.56	-10.02 ± 7.53	-16.87* ± 6.03	-4.22 ± 3.47	16.97 ± 13.99

**, ** : significant at 5% and at 1% level, respectively.*

Table 40. Estimates of gene effects for seed yield/plant (g) in different crosses.

Cross No.	^ m	^ d	^ h	^ i	^ j	^ l
1.	19.77** ± 2.22	-4.07* ± 1.92	61.30**±11.72	55.46** ± 9.69	8.33 ± 4.17	-22.20 ± 17.65
2.	35.30** ± 1.19	-7.30 ± 6.25	-4.99 ± 14.20	-27.67 ± 13.38	3.48 ± 6.38	83.97**±27.18
3.	18.87** ± 0.32	7.37* ± 2.81	51.43** ± 8.08	43.13** ± 5.77	13.70* ± 4.95	7.93 ± 15.99
4.	18.57** ± 1.45	-12.90 ± 13.92	128.88**±30.78	103.00**±28.43	-16.08 ± 13.92	-131.63**±60.73
6.	20.20** ± 1.77	-18.73** ± 2.80	53.08**±10.42	52.40** ± 9.03	-22.48** ± 5.00	-43.50* ± 16.85
7.	27.33** ± 0.56	8.37 ± 6.68	34.71* ± 14.40	26.73 ± 13.55	-5.62 ± 7.09	48.83 ± 28.54
8.	17.73** ± 2.84	-1.17 ± 1 0.66	88.87* ± 24.33	97.13**±24.16	4.63 ± 10.95	-144.93**±44.50
10.	14.60** ± 0.32	-25.57** ± 7.22	165.32**±15.91	135.00**±14.50	-30.74** ± 7.45	-180.90**±31.75
11.	25.23** ± 2.21	-11.30* ± 4.73	50.75**±17.42	45.53**±12.94	-20.95* ± 7.11	-64.30 ± 31.30
12.	16.20** ± 3.72	14.87* ± 6.67	147.05**±23.15	111.60**±20.00	4.25 ± 6.84	-115.16**±38.45

**, ** : significant at 5% and at 1% level, respectively.*

d was negative, in cross No.2, only **l** was positive and significant, in cross No.3 and 11, **d, h, i** and **j** were significant, in cross No.4 and 8, **h, i** and **l** were significant but **l** was negative, in cross No.6 and 10, all types of gene actions were observed but only **h** and **i** type gene effects were positive, in cross No.7, only **h** was significant and positive, and in cross No.12, **d, h, i** and **l** type of gene effects were significant but **l** was negative in magnitude. Duplicate type epistasis was observed in almost all the crosses but in cross No.3 and 7, complementary type of epistasis was observed.

Seed weight

The data from Table 41 indicated that in cross No.1, **h, i and l** type of gene effects were observed significant but dominance x dominance (**l**) type gene action was in higher magnitude than dominance (**h**) and additive x additive (**i**) type of gene actions. In cross No.3, only additive x dominance (**j**) type of gene action was significant with high magnitude. In cross No.8, **h** and **i** type gene action was observed with high magnitude, in cross No.10, **d, h, i** and **l** types of gene actions were significant but **l** has higher magnitude than **d, h** and **i**. In cross No.11, **h, i** and **l** were significant but **l** was negative, and in cross No.12, **d** and **j** type of gene effects were positive and significant. All the crosses were showing duplicate type epistasis except cross No.7 and 12 which showed complementary type epistasis.

Oil content

It was observed from Table 42 that additive (**d**), dominance (**h**), additive x additive (**i**) and dominance x dominance (**l**) type gene actions had greater role in controlling of this trait. In cross No.1, dominance (**h**) gene effect was observed with negative magnitude. In cross No.6, the additive (**d**)

Table 41. Estimates of gene effects for 1000-seed weight (g) in different crosses.

Cross No.	^ m	^ d	^ h	^ i	^ j	^ l
1.	3.01** ± 0.14	-0.06 ± 0.18	-1.85* ± 0.75	-1.68* ± 0.65	0.23 ± 0.25	2.48* ± 1.18
2.	3.15** ± 0.03	0.25 ± 0.40	-1.38 ± 0.84	-1.68 ± 0.82	0.21 ± 0.41	2.38 ± 1.67
3.	3.16** ± 0.09	0.52 ± 0.39	1.49 ± 0.92	0.77 ± 0.87	1.31** ± 0.43	-0.69 ± 1.73
4.	3.48** ± 0.10	0.12 ± 0.33	-1.20 ± 0.85	-0.91 ± 0.77	-0.58 ± 0.34	1.04 ± 1.57
6.	2.83** ± 0.09	0.03 ± 0.24	0.14 ± 0.62	-0.09 ± 0.60	0.12 ± 0.29	-0.23 ± 1.08
7.	2.49** ± 0.08	0.11 ± 0.39	0.01 ± 0.99	0.20 ± 0.86	0.37 ± 0.43	1.91 ± 1.89
8.	2.39** ± 0.04	-0.13 ± 0.46	2.69* ± 0.99	2.17* ± 0.94	-0.43 ± 0.48	-2.53 ± 1.96
10.	3.82** ± 0.04	-0.84* ± 0.28	-1.53* ± 0.66	-1.75** ± 0.57	-0.21 ± 0.30	2.90* ± 1.30
11.	3.69** ± 0.05	-0.40 ± 0.25	2.19** ± 0.63	1.49** ± 0.52	-0.38 ± 0.26	-3.35** ± 1.21
12.	3.38** ± 0.19	1.47** ± 0.14	0.39 ± 0.87	-1.01 ± 0.80	1.15** ± 0.15	0.77 ± 1.16

**, ** : significant at 5% and at 1% level, respectively.*

Table 42. Estimates of gene effects for percent oil content in different crosses.

Cross No.	$\hat{m}$	$\hat{d}$	$\hat{h}$	$\hat{i}$	$\hat{j}$	$\hat{l}$
1.	38.17** ± 0.49	-0.63 ± 0.83	0.73 ± 2.62	-6.67* ± 2.58	1.40 ± 0.91	-1.73 ± 3.98
2.	37.07** ± 0.43	0.00 ± 1.02	-1.78 ± 2.79	-2.67 ± 2.68	-0.42 ± 1.25	5.70 ± 4.70
3.	37.10** ± 0.40	-0.50 ± 0.35	3.12 ± 1.78	2.20 ± 1.76	0.65 ± 0.41	-2.10 ± 2.21
4.	37.03** ± 0.62	0.13 ± 1.14	0.33 ± 3.45	1.07 ± 3.36	0.93 ± 1.18	-2.00 ± 5.41
6.	37.57** ± 0.19	-2.03* ± 0.74	4.25* ± 1.77	3.00 ± 1.66	-1.55 ± 0.84	-5.30 ± 3.29
7.	39.03** ± 0.35	-2.83** ± 0.53	4.92* ± 1.83	5.93** ± 1.75	-0.98 ± 0.61	-10.10** ± 2.75
8.	37.90** ± 0.84	0.43 ± 0.58	2.52 ± 3.58	1.00 ± 3.55	0.68 ± 0.69	-3.83 ± 4.18
10.	37.30** ± 0.59	-1.47** ± 0.45	2.52 ± 2.56	1.73 ± 2.51	-0.92 ± 0.51	-2.63 ± 3.11
11.	38.53** ± 0.75	-0.63 ± 0.82	-3.02 ± 3.47	-3.27 ± 3.43	-1.15 ± 0.90	4.97 ± 4.60
12.	37.20** ± 0.70	0.97 ± 1.33	3.92 ± 3.93	2.87 ± 3.86	0.68 ± 1.48	-7.97 ± 6.19

**, ** : significant at 5% and at 1% level, respectively.*

and dominance (**h**) gene effects were significant but **d** was negative. In cross No.7, **d**, **h**, **i** and **l** type of gene action was observed significant but **d** and **l** were negative. In cross No.10, only additive (**d**) effect was observed significant and negative. In all the crosses duplicate type epistasis was observed.

4.3 Identification of desirable crosses and selection of high yielding resistant recombinants

Comparision of the means of different characters of different crosses or genotypes by the Duncan's multiple range test provides the rank for a high mean value for a character in a genotype or cross. With the help of this test, better performing genotypes or crosses showing the top rank for all yield contributing characters can be identified.

It was observed from Table 43 that all the 10 selected crosses were showing very good performance for yield and its components except some characters in some crosses. Two crosses were showing better performance for almost all the characters. Cross No.3 (Fig. 14) was showing A, AB or ABC rank for all the nine characters except oil content (BC) and cross No.7 was showing A, AB or ABC rank for eight characters out of ten characters studied except days to flowering (BC) and days to maturity (C), although the means of these two characters were not having so wide range but they were showing slightly higher mean values. This cross yielded useful segregants with resistance (Fig. 15a) and high productivity as well as susceptible plants (Fig. 15b). Therefore, based on the mean values of different characters, these two crosses i.e. cross No.3 and 7 can be identified for isolating better performing recombinants for yield and its components.

Both these crosses and some others have in fact yielded useful segregants (Fig.16) in F_3 showing white rust resistance and superior yield

Table 43. Mean and rank of different characters in different crosses

Cross No.	DF		DM		PH		PB		SB		LM		SM		SY		SW		OC	
	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank
1.	52.4	A	144.7	A	192.8	C	10.1	AB	39.7	A	65.5	C	53.1	B	40.98	AB	2.821	BC	37.57	BC
2.	54.6	AB	145.7	ABC	218.0	AB	8.6	BCD	35.7	AB	82.8	A	64.3	A	37.86	AB	2.859	BC	37.01	BC
3.	55.1	ABC	145.0	AB	225.5	A	9.6	AB	38.6	A	82.1	A	63.0	A	41.03	AB	3.249	ABC	37.52	BC
4.	54.8	ABC	146.3	BC	212.7	AB	7.6	CD	29.7	BCD	63.9	C	52.1	B	32.84	B	3.340	AB	37.19	BC
6.	54.2	AB	146.2	BC	219.9	A	8.9	BCD	34.7	ABC	79.8	A	64.3	A	35.41	B	2.683	C	37.53	BC
7.	56.6	BC	147.1	C	215.0	AB	10.8	A	39.4	A	80.3	A	65.3	A	51.58	A	3.097	ABC	39.64	A
8.	57.3	BC	145.3	AB	204.3	BC	8.8	BCD	35.5	ABC	72.8	B	60.6	A	31.44	B	2.757	C	37.19	BC
10.	56.4	BC	145.7	ABC	215.9	AB	7.3	D	28.0	CD	84.9	A	63.1	A	31.82	B	3.636	A	37.38	BC
11.	55.2	ABC	145.3	AB	214.9	AB	7.3	D	26.7	D	79.5	A	59.6	A	31.06	B	3.476	A	38.10	B
12.	57.8	C	146.6	BC	221.9	A	9.3	ABC	41.7	A	81.6	A	64.9	A	37.30	B	2.832	BC	36.47	C

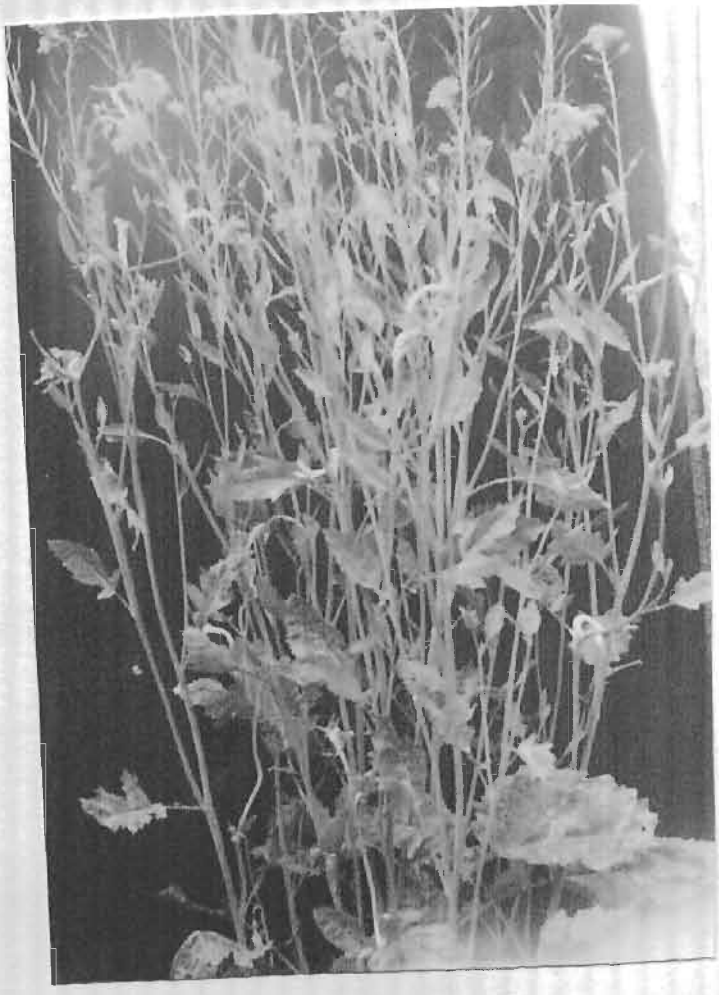
Fig.14: Productive and resistant plants of Cross No. 3 showing healthy growth and earliness

Fig.15: Resistant and susceptible plants to white rust in Cross No. 7

a. Resistant segregants showing productive growth but slight lateness

b. A susceptible segregant showing white rust disease symptoms on leaves and several staghead

a



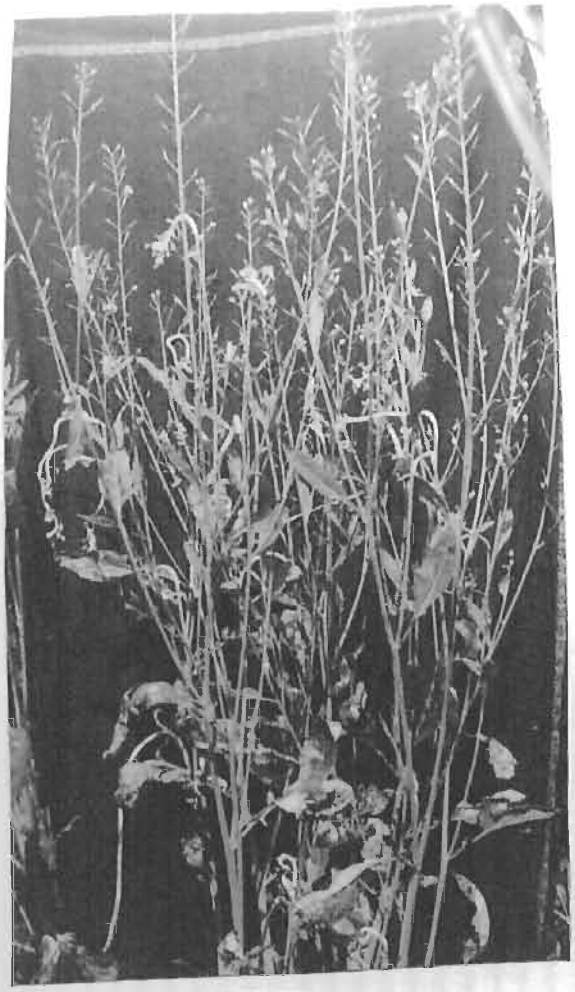
b

Fig.16: Productive and white rust resistant segregants compared to a highly susceptible parental line

a. A highly susceptible plant from parental line showing severely diseased leaves and full of stagheads

b. Resistant segregants showing no disease symptoms on leaves or inflorescence

c. Highly productive, early maturing, disease resistant segregant in F_3 generation



a

b

c

attributes. Many useful recombinants for seed yield selected on a visual basis are breeding true for white rust resistance. By using systematic breeding and selection procedure as suggested in the previous section more useful genotypes can be isolated.

DISCUSSION

Biologically based technologies represent the primary means of plant disease management worldwide; contrary to public perceptions, synthetic pesticides play a relatively minor role compared to biologically based methods for plant disease management. Host plant resistance always will be among the most important biologically based method of plant disease management. The use of genes accessible within sexually compatible populations of crop plants., as practiced by plant breeders for most of this century, continues to account for most of the progress, but is also a maturing technology. The use of alien genes, i.e., genes from wild relatives and progenitors of crop plants, although practised by plant breeders since around the middle of this century, is still a very young technology and holds promise as a means to greatly expand the circle of diseases (Jones *et al.*, 1995). It is important to recognise that the principles of plant breeding are the same regardless of the source of the gene(s) or method used to transfer the genes into a variety for commercial use. Indeed, in most cases, the newer techniques are used initially to create parents with the useful new genes for resistance and then later the variety is developed by conventional methods.

During earlier studies using different techniques white rust resistant strains of *B. juncea* were developed in Brassica cytogenetics section of Genetics Division, IARI, New Delhi (Subudhi and Raut, 1994; Rao and Raut, 1994; Vasanthi, 1993; Sridhar, 1996). It is necessary to properly characterise these genes with regard to their mode of inheritance and stability under different agroclimatic conditions. These two aspects formed

the basic objectives of the present studies.

In a recent publication Downey and Chopra (1998) identified plant protection as a major emerging trend in oleiferous brassica breeding. In China, well adapted, disease resistance canola quality winter and spring *B. napus* cultivars have been released but such varieties in *B. juncea* are not available in the Indian subcontinent. In the absence of resistant varieties the loss in production can be devastating in a bad year like Rabi 1997-98 in Northern India. The climatic factors of rainfall, humidity, temperature etc. were quite favourable for the development of white rust disease (Appendix I). This resulted in widespread infection of mustard by white rust and other diseases in north and north-eastern part of the country. The yield loss in zone II comprising states of Punjab, Haryana, Rajasthan and Delhi was to the extent of over 40%. Appendix II tabulates data for yield loss of standard varieties of mustard during 1998 compared to 1997 which was a normal year. Even in zone III comprising states to Rajasthan, Uttar Pradesh and Madhya Pradesh the yield loss was significant (Anonymous 1997, 1998). These observations establish beyond doubt the importance of incorporating disease resistance more specifically white rust resistance to mustard cultivars for making them more stable. This is because of the fact that resistant genes to white rust are now available in *B. juncea*, their mode of inheritance is not complex and conventional breeding can result in having high yielding white rust resistant varieties in the near future. Results obtained during the present study are useful steps in this direction. Some of the significant findings are discussed in the following pages.

5.1 White rust resistance

Lack of resistance to diseases in our currently grown cultivars is one important reason for high fluctuation of rapeseed-mustard production and productivity from year to year in the country. Among the diseases, white rust

is most serious under Indian conditions. Therefore, knowledge of the inheritance pattern of resistance to this disease is essential for choosing a suitable strategy for incorporation of resistant genes in existing cultivars. At early segregating generations of F_2 and F_3 also, we could select some promising lines showing resistance because only a few major genes were conferring resistance and also frequently associated with phenotypic markers.

Perusal of data on white rust in two crosses (Tables 4 and 5) indicated involvement of two different and totally independent genes with dominant and recessive gene interaction because of close agreement to 13R:3S in F_2 at all locations. Resistance reaction in F_1 hybrids of both crosses may be due to epistasis or dominance. In cross No. 1, female parent (S-II) and male parent (S-III) were carrying the monogenic dominance and monogenic recessive gene for resistance, depending on the source of the gene. In cross No.6, female parent (S-III) and male parent (S-IV) were carrying the monogenic recessive gene and monogenic dominant gene respectively for resistance, depending on the source of the gene. Therefore, in both these crosses, Ac_1 only is conferring resistance to white rust but Ac_2 for susceptibility. Because of dominant epistasis of Ac_1 and also epistatic interaction of ac_2 when recessive homozygous this ratio of 13R:3S was obtained (Ac_1- $-$ $-$, $ac_1ac_1ac_2ac_2$: resistant, $ac_1ac_1Ac_2$ $-$: susceptible, Table 44). Segregation from F_3 family of a few randomly selected plants and all resistant plants obtained in both B_1 and B_2 partly confirm this. These results are in agreement with Rao and Raut (1994), Subudhi and Raut (1994), for similar segregation ratios obtained in interspecific crosses. Variation in expression of resistance under 0-5 disease scoring scale at different locations might be due to the availability of congenial agroclimatic conditions in hills and prevalence of different races of pathogens, the plants may develop more disease symptoms and change into a comparatively low resistance or more susceptible grade (Table 17). This is because Wellington

Table 44: Proposed genetic constitution of parents, F_1 and F_2 generations of crosses showing segregation ratio of 13 R : 3S for white rust resistance.

Cross No./ Parents	Generation	Genotype	Phenotype	Location
1. (S-II-2xS-III-2)	P_1	$Ac_1\ Ac_1\ Ac_2\ Ac_2$	Resistant	Delhi
	P_2	$ac_1\ ac_1\ ac_2\ ac_2$	Resistant	Delhi
	F_1	$Ac_1\ ac_1\ Ac_2\ ac_2$	Resistant	Delhi and Wellington
	F_2	$Ac_1 - - -$ $ac_1\ ac_1\ ac_2\ ac_2$	Resistant	Over all locations
		$ac_1\ ac_1\ Ac_2 -$	Susceptible	
6. (S-III-3xS-IV-3)	P_1	$ac_1\ ac_1\ ac_2\ ac_2$	Resistant	Delhi
	P_2	$Ac_1\ Ac_1\ Ac_2\ Ac_2$	Resistant	Delhi
	F_1	$Ac_1\ ac_1\ Ac_2\ ac_2$	Resistant	Delhi and Wellington
	F_2	$Ac_1 - - -$ $ac_1\ ac_1\ ac_2\ ac_2$	Resistant	Over all locations
		$ac_1\ ac_1\ Ac_2 -$	Susceptible	

Table 45: Proposed genetic constitution of parents, F_1 and F_2 generations of crosses showing segregation ratio of 3 R : 1S for white rust resistance.

Cross No./ Parents	Generation	Genotype	Phenotype	Location
2. (S-II-3xS-IV-2)	P_1	$Ac_1\ Ac_1$	Resistant	Delhi
3. (S-II-6xS-VII-2)	P_2	$Ac_1\ ac_1$	Resistant	Delhi
	F_1	$Ac_1\ Ac_1$, $Ac_1\ ac_1$	Resistant	Delhi and Wellington
	F_2	$Ac_1\ Ac_1$ or	Resistant	Delhi
		$Ac_1 -$ $ac_1\ ac_1$	Resistant Susceptible	Over all locations

and Kukumseri are located on hills providing favourable temperature, humidity and sunlight conditions for the development of pathogen spores and may be having variation in pathogen flora as compared to Delhi.

Data on white rust in two crosses (Table 6 and 7) indicated involvement of same white rust resistance gene because of close agreement to 3R:1S ratio in F_2 at all locations, involving both resistant parents. It means one parent in both the crosses (No.2 and 3) was heterozygous (Table 45). All the resistant plants in F_1 shows the dominance of resistance over susceptibility. Monogenic dominant gene controlling resistance to white rust is in agreement with Thukral and Singh (1986); Tiwari *et al.* (1988), Singh and Singh (1988) and Yadav *et al.* (1994). True breeding nature of one F_1 plant progeny and segregation to 3R:1S in F_2 by other F_1 plant progeny confirm our inferences. Backcrosses of F_1 with both the parents resulted in all resistant plants because the F_1 plants had the genotype Ac_1Ac_1. In F_3 both type of pattern either true breeding for resistance or segregation into resistant and susceptible plants with higher frequency of resistant plants further confirms our conclusion drawn from the results.

Data on white rust in five crosses (Table 8 to 12) indicated the dominance of resistance over susceptibility showing monogenic dominant inheritance (3R:1S in F_2 at all locations) of white rust resistance involving resistant female and susceptible male parents in cross No. 4,8,11,12 and male parent was resistant and female parent susceptible in cross No.9 (Table 46). In this cross, the source of resistance gene was different (Ac_3Ac_3) but in rest of the four crosses the source of resistant gene was same (Ac_1Ac_1). True breeding resistant plants or segregation into resistant and susceptible plant in F_3 progenies, B_1 and B_2 confirm the monogenic dominant inheritance of white rust resistance. These results are in agreement with the findings of Tiwari *et al.* (1988), Liu *et al.* (1987), Yashpal *et al.* (1991) and Yadav *et al.* (1994).

Table 46: Proposed genetic constitution of parents, F_1 and F_2 generations of crosses showing segregation ratio of 3 R : 1S for white rust resistance.

Cross No./ Parents	Generation	Genotype	Phenotype	Location
4. (S-II-7xS-VIII-2)	P_1	$Ac_1 Ac_1 Ac_2 Ac_2$	Resistant	Delhi
	P_2	$ac_1 ac_1 Ac_2 Ac_2$	Susceptible	Delhi
	F_1	$Ac_1 ac_1 Ac_2 ac_2$	Resistant	Delhi and Wellington
	F_2	$Ac_1 Ac_1 Ac_2 Ac_2$ $Ac_1 ac_1 Ac_2 Ac_2$	Resistant	Over all locations
		$ac_1 ac_1 Ac_2 Ac_2$	Susceptible	
8. (S-IV-6xS-VII-4)	P_1	$Ac_1 Ac_1$	Resistant	Delhi
11. (S-IV-10x Pusa Bold)	P_2	$ac_1 ac_1$	Susceptible	Delhi
	F_1	$Ac_1 ac_1$	Resistant	Delhi and Wellington
	F_2	Ac_1 – $ac_1 ac_1$	Resistant Susceptible	Over all locations
9. (S-IV-8xS-IX-4)	P_1	$ac_1 ac_1 ac_3 ac_3$	Susceptible	Delhi
	P_2	$ac_1 ac_1 Ac_3 Ac_3$	Resistant	Delhi
	F_1	$ac_1 ac_1 Ac_3 ac_3$	Resistant	Delhi and Wellington
	F_2	$ac_1 ac_1 Ac_3 Ac_3$ $ac_1 ac_1 Ac_3 ac_3$	Resistant	Over all locations
		$ac_1 ac_1 ac_3 ac_3$	Susceptible	
12. (S-VI-8xS-IX-6)	P_1	$Ac_1 Ac_1 ac_3 ac_3$	Resistant	Delhi
	P_2	$ac_1 ac_1 ac_3 ac_3$	Susceptible	Delhi
	F_1	$Ac_1 ac_1 ac_3 ac_3$	Resistant	Delhi and Wellington
	F_2	$Ac_1 Ac_1 ac_3 ac_3$ $Ac_1 ac_1 ac_3 ac_3$	Resistant	Over all locations
		$ac_1 ac_1 ac_3 ac_3$	Susceptible	

The nature of gene in cross No.7 (Table 13) was assumed to be monogenic recessive resistant to white rust, giving 1R:3S ratio in F_2 at all locations, involving one resistant and one susceptible parent. It indicates that susceptibility is dominant over resistance because F_1 had all susceptible plants. Therefore, resistance was controlled by a recessive homozygous gene (ac_2ac_2). Another interpretation of the same result can be made assuing inability of the heterozygous to confer resistance and only the dominant homozygous was resistant. Two dose of dominant gene (Ac_2Ac_2) are required to confer resistance to white rust in this cross and Ac_2ac_2, ac_2ac_2 are susceptible (Table 47). Assuming monogenic recessive behaviour of resistance to white rust our results are in agreement to Singh and McIntosh (1986), Knott and McIntosh (1978) as they had reported monogenic recessive nature of stem rust resistance genes Sr8b and Sr17 respectively in wheat and Zhang *et al.* (1996) reported Xa 13 gene for Bacterial leaf blight in rice. As no recessive gene conferring resistance in Brassica has been reported so far we are inclined to favour the interpretation of heterozygous susceptible. If we assume monogenic dominant nature of resistance gene with two dose then our results are in agreement to Gadewadikar *et al.* (1993), they reported that white rust resistance in *B. juncea* was under the control of a dominant gene pair. Segregation into resistant and susceptible or all resistant plants in F_3 progenies and all the susceptible plants in backcross with susceptible parent and segregation into resistant and susceptible type, in backcross with resistant parent partially confirm our hypothesis of haplo insufficient for resistance. In view of this, our interpretation appears more plausible.

Data on white rust in cross No.5 (Table 14) involving two resistant parents indicated the digenic duplicate nature of white rust resistance at Delhi and monogenic dominant behaviour at Wellington and Kukumseri showing 15R:1S and 3R:1S ratio in F_2 respectively. It indicates that resistant

Table 47: Proposed genetic constitution of parents, F_1 and F_2 generations of cross showing segregation ratio of 1R : 3S for white rust resistance.

Cross No./ Parents	Generation	Genotype	Phenotype	Location
7. (S-III-9xS-X-3)	P_1	$ac_2\,ac_2\,ac_4\,ac_4$	Resistant	Delhi
	P_2	$Ac_2\,Ac_2\,ac_4\,ac_4$	Susceptible	Delhi
	F_1	$Ac_2\,ac_2\,ac_4\,ac_4$	Susceptible	Delhi and Wellington
	F_2	$ac_2\,ac_2\,ac_4\,ac_4$	Resistant	Over all
		$Ac_2\,Ac_2\,ac_4\,ac_4$ $Ac_2\,ac_2\,ac_4\,ac_4$	Susceptible	locations
		or		
	P_1	$Ac_2\,Ac_2\,ac_4\,ac_4$	Resistant	Delhi
	P_2	$ac_2\,ac_2\,ac_4\,ac_4$	Susceptible	Delhi
	F_1	$Ac_2\,ac_2\,ac_4\,ac_4$	Susceptible	Delhi and Wellington
	F_2	$Ac_2\,Ac_2\,ac_4\,ac_4$ $Ac_2\,ac_2\,ac_4\,ac_4$	Resistant	Over all
		$ac_2\,ac_2\,ac_4\,ac_4$	Susceptible	locations

gene present in both the parents expressed in F_2 but at Wellington and Kukumseri the resistant gene present in parent two (S-X-2) failed to express and behaved as susceptible because of the prevalence of different racesof pathogen, which knockdown the resistance gene (Ac_4Ac_4) at both these locations (Table 48). Digenic duplicate nature of white rust resistance was reported by Fan *et al.* (1983), Verma and Bhowmik (1989). In north Indian plains only race 2 of *A. candida* has been reported but in hills of Nilgiris and Himalayas occurence of other race cannot be ruled out in view of their characteristic flora.

One more cross (No.10) between a resistant and susceptible parent showed monogenic dominant nature (3R:1S) at Delhi and monogenic recessive nature or monogenic dominant requiring two dose of dominant gene for resistance (1R:3S) at Wellington and Kukumseri. Resistant reaction in F_1 hybrid was observed at Delhi indicating that only one dose of dominant resistant gene is sufficient to confer resistance (haplo sufficient) but at Wellington and Kukumseri two dose of dominant gene is required to confer resistance. Therefore, gene in heterozygous condition was unable to confer resistance to white rust (Table 49) because of agroclimatic conditions most congenial for the development of disease at these two locations.

Variation in the expression of white rust resistance in different crosses (Table 16A,B,C,D) was due to different sources of resistant gene which was incorporated either through inter-specific or intra-specific hybridization and whether male parent was resistant or susceptible and also availability of congenial agroclimatic conditions.

Variation in expression of white rust resistance in different crosses under 0-5 scale at different locations (Table 17,18,19) was due to the availability of congenial agroclimatic conditions and different races of pathogen by which the plants under one grade were changed into

Table 48: Proposed genetic constitution of parents, F_1 and F_2 generations of cross showing segregation ratio of 15R : 1S and 3R : 1S for white rust resistance at different locations.

Cross No./ Parents	Generation	Genotype	Phenotype	Location
5. (S-II-9xS-X-2)	P_1	$Ac_1\ Ac_1\ ac_4\ ac_4$	Resistant	Delhi
	P_2	$ac_1\ ac_1\ Ac_4\ Ac_4$	Resistant	Delhi
	F_1	$Ac_1\ ac_1\ Ac_4\ ac_4$	Resistant	Delhi and Wellington
	F_2	$Ac_1 - - -$ / $- Ac_4 - -$	Resistant	Delhi
		$ac_1\ ac_1\ ac_4\ ac_4$	Susceptible	
	F_2	$Ac_1 - - -$	Resistant	Wellington
		$ac_1\ ac_1\ Ac_4 -$ / $ac_1\ ac_1\ ac_4\ ac_4$	Susceptible	and Kukumseri

Table 49: Proposed genetic constitution of parents, F_1 and F_2 generations of cross showing segregation ratio of 3R : 1S and 1R : 3S for white rust resistance at different locations.

Cross No./ Parents	Generation	Genotype	Phenotype	Location
10. (S-IV-9xS-X-4)	P_1	$ac_1\ ac_1\ ac_4\ ac_4$	Susceptible	Delhi
	P_2	$ac_1\ ac_1\ Ac_4\ Ac_4$	Resistant	Delhi
	F_1	$ac_1\ ac_1\ Ac_4\ ac_4$	Resistant	Delhi and Wellington
	F_2	$ac_1\ ac_1\ Ac_4 -$	Resistant	Delhi
		$ac_1\ ac_1\ ac_4\ ac_4$	Susceptible	
	F_2	$ac_1\ ac_1\ Ac_4\ Ac_4$	Resistant	Wellington
		$ac_1\ ac_1\ Ac_4\ ac_4$ / $ac_1\ ac_1\ ac_4\ ac_4$	Susceptible	and Kukumseri

comparatively low resistance or more susceptible grade. Agroclimatic conditions are most favourable at Wellington and Kukumsari making them 'hotspots' for disease development having appropriate humidity, temperature, and different virulent races of pathogen, whereas at Delhi due to less congenial agroclimatic conditions and limited pathogen flora infection was less except in bad years like 1997-98 rabi. Therefore, at Wellington and Kukumsari the plants which were under the resistant grade **0** at Delhi, might be changed to grade **1** or **2** and plants under grade **2** might be changed to grade **3**, **4** or **5** increasing the susceptibility. Change of grade **2** to **3** sometimes even alters the ratio from 3R:1S to 1R :3S.

5.2 Association of leaf waxiness with white rust resistance

Association of leaf waxiness with white rust resistance was observed in five crosses (Table 20 to 24) which is in agreement with Fan *et al.* (1983), Subudhi and Raut (1994), and Jambhulkar and Raut (1995). In all the segregating generations viz., F_2, F_3, B_1 and B_2 for over five crosses, frequency of resistant plants were high under waxy category. χ^2 test of independence in F_2 populations of these crosses shows that white rust resistance was dependent on leaf waxiness (Table 44). Because waxy leaf does not allow the moisture to stick and the spore of pathogen fail to germinate on dry leaf surface. It may also be providing some chemical barrier for the infection to occur. However, there is no linkage of genes governing leaf waxiness and disease resistance.

Independence of white rust resistance from leaf waxiness was noticed in seven crosses (Table 25 to 31). It indicates that white rust is not dependent on leaf waxiness (Table 50) in these crosses because of low intensity of waxiness and nature of resistant gene involved in non-waxy plants which shows resistance even without being associated with waxiness.

Table 50: Test of independence for leaf waxiness and white rust resistance for F_2 population of different crosses.

Cross No./ Parents	Waxy Frequency R	Waxy Frequency S	Non-Waxy Frequency R	Non-Waxy Frequency S	χ^2	P-range
1. (S-II-2xS-III-2)	152	22	38	9	1.30	0.20-0.30
2. (S-II-3xS-IV-2)	89	18	33	11	1.34	0.20-0.30
3. (S-II-6xS-VII-2)	160	20	42	9	1.55	0.20-0.30
4. (S-II-7xS-VIII-2)	128	42	42	25	3.77	0.05-0.10
5. (S-II-9xS-X-2)	50	-	28	8	10.60	0.001-0.01
6. (S-III-3xS-IV-3)	166	27	61	22	6.23	0.01-0.02
7. (S-III-9xS-X-3)	31	42	14	75	14.29	
8. (S-IV-6xS-VII-4)	58	17	60	24	0.72	0.30-0.50
9. (S-IV-8xS-IX-4)	97	19	56	25	5.77	0.01-0.02
10. (S-IV-9xS-X-4)	128	31	54	23	3.16	0.05-0.10
11. (S-IV-10xPusaBold)	101	21	62	28	5.63	0.01-0.02
12. (S-VI-8xS-IX-6)	94	35	33	19	1.57	0.20-0.30

Table 51: Test of independence for yellow seed colour and white rust resistance for F_2 population of some crosses involving a yellow seeded parent.

Cross No./ Parents	Seed colour	Brown Frequency R	Brown Frequency S	Yellow Frequency R	Yellow Frequency S	χ^2	P-range
7.	(S-III-9xS-X-3)	40	105	5	12	0.0250	0.80-0.90
10.	(S-IV-9xS-X-4)	161	48	21	6	0.0008	0.95-0.98
12.	(S-VI-8xS-IX-6)	89	32	38	16	0.1900	0.50-0.70

Low intensity of waxiness retains some moisture on leaf surface which allows spores of pathogen to germinate and develop the disease. However, the main reason for failure of association of leaf waxiness and resistance is due to high frequency of non-waxy resistant plants.

5.3 Association of yellow seed colour with white rust resistance

Association of yellow seed colour with white rust resistance could not be observed in the three crosses (Table 32), involving one yellow seeded parent. In F_2 there is digenic inheritance of seed colour where brown colour dominates on the yellow seed colour, yielding only few yellow seeded plants and many of intermediate colour. On the other hand white rust resistance in these crosses were monogenic dominant. The χ^2 analysis showed independent segregation of these two characters (Table 51). The association of these two characters reported in literature (Sridhar, 1996) might be spurious.

5.4 Estimation of Gene Effects

The gene effects were estimated for different plant characters including yield components using six generations P_1, P_2, F_1, F_2, B_1 and B_2 in ten selected crosses and were presented in Section 4.2 (Tables 33 to 42). These gene effects have been discussed here briefly.

All types of gene effects i.e., additive (**d**), dominance (**h**), additive x additive (**i**), additive x dominance (**j**)and dominance x dominance (**l**) were observed to be significant in variable proportions depending on cross and traits. Duplicate epistasis was predominant in all ten crosses except some crosses where complementary epistasis was observed. Negative values in many cases may be an hindrance if not compensated by the positive effects of other parameters but in case of DF, DM and PH negative values to some

extent are desirable. The characters which show additive or additive x additive components of genetic variance, selection for these characters are likely to be easy and effective because these components of genetic variance are highly heritable and easily fixable.

For days to flowering, additive (**d**), dominance (**h**), additive x additive (**i**) and dominance x dominance (**l**) epistasis effects were found to be positive in cross No.2,6 and 8 (Table 33). In cross No.6, dominance x dominance (**l**) effects was negative. It indicates that cross No.6 is desirable for this trait and early maturing strains can be isolated. Additive and additive x additive effects offers easy selection for this trait by any simple selection procedure but non-additive and dominance x dominance epistasis components of genetic variance can be improved by recurrent or reciprocal recurrent selection for this trait. Additive and non-additive gene effects were reported by Labana *et al.*(1978) and Pradhan *et al.* (1993) for days to flowering.

For days to maturity dominance (**h**) and additive x additive (**i**) effects were found in cross No.1 and 12 (Table 34). Negative and higher magnitude of **h** and **i** in cross No. 12 suggests that this cross is desirable for fixation of this trait by easy selection and to breed early maturing strains. Additive and non-additive effects were reported for this character by Labana *et al.* (1978) and Jindal and Labana (1986).

All types of gene effects, additive, dominance, additive x additive, additive x dominance and dominance x dominance were observed for plant height depending on the cross such as cross No.1,3,10,2,4,6,7,11 and 12 (Tabe 35). Additive x additive and dominance effects were negative with higher magnitude and are desirable in most of these crosses. Cross No.2 showed additive, dominance and additive x additive effects positive with lower magnitude and other effects in these crosses which were positive and

not desirable for this trait. Additive x dominance epistasis was negative with higher magnitude in cross No.11 and is desirable. Due to the presence of all type gene effects, reciprocal recurrent selection is better procedure for improvement of this trait simultaneously with yield. Additive and non-additive gene effects were reported by Anand and Reddy (1987), Yadava *et al.* (1993), Kumar *et al.* (1994) and dominance and epistasis was reported by Kumar *et al.* (1998) for plant height.

For number of primary branches, dominance, additive x additive and dominance x dominance components of genetic variance were observed in different crosses (Table 36). These effects were positive and highly significant in most of the crosses indicating that the effects are fixable by reciprocal recurrent selection. The effects with negative magnitude are not desirable. For this character, additive and non-additive nature of gene action were observed by Singh *et al.* (1982), Yadav *et al.* (1992), Yadava *et al.* (1993), Diwakar and Singh (1993), Kumar *et al.* (1994) and dominance and epistasis by Chaudhary and Sharma (1982), Verma *et al.* (1992) and Kumar *et al.* (1998).

Dominance, additive x additive and dominance x dominance effects were observed both positive and negative with higher magnitude in different crosses, for number of secondary branches (Table 37). Cross No. 1,3,4,8,10,11 and 12 were desirable which shows dominance and additive x additive effects positive and high magnitude. This trait with additive and nonadditive gene effects can be improved by recurrent or reciprocal recurrent selection because this method can improve the character in a population through accumulation of favourable additive genes and also simultaneously maintaining the heterozygosity in population for manifestation of non-additive gene effects. Additive and non-additive gene effects was reported for this trait by Ram Dhari and Yadava (1983), Labana *et al.* (1984),

Yadava *et al.* (1993), Kumar *et al.* (1994); epistasis by Verma *et al.* (1992), Verma and Singh (1998); dominance and epistasis by Kumar *et al.* (1998).

For length of main fruiting axis, additive, dominance, additive x additive, additive x dominance and dominance x dominance effects were observed in different crosses (Table 38). In most of the crosses, additive x additive, dominance were positive with higher magnitude than other gene effects. These effects were also desirable for improvement of this character. Singh *et al.* (1981), Rawat (1992), Singh and Mital (1993) have reported additive and non-additive effects and Kumar *et al.* (1998) have reported dominance and epistasis for this character. Reciprocal recurrent selection is best method for improvement of this character.

All types of gene effects were observed for number of siliqua on main fruiting axis in different crosses. Dominance and additive x additive effects were desirable in cross No.2, 3 and 8 with higher magnitude but is undesirable in cross No.7.Additive, additive x dominance and dominance x dominance epistasis were desirable in cross No.7. Other effects were undesirable due to negative and lower magnitude in other crosses (Table 39). Additive and non-additive effects were reported by Labana *et al.* (1984), Verma *et al.* (1992), Singh and Mital (1993); non-additive effects were reported by Anand and Reddy (1987) and Gupta *et al.* (1987) and dominance and epistasis by Kumar *et al.* (1998) for this character.

For seed yield per plant, all types of gene effects were observed in different crosses (Table 40). Additive x additive and dominance gene effects were found desirable in most of the crosses with both positive high and low magnitude effects. Although additive effects were desirable in cross No.3,11 and 12 and dominance x dominance gene effects was desirable in cross No.2 where as additive x dominance epistasis was found desirable in cross No.3 and 11. Reciprocal recurrent selection is the best method for

improvement of this trait. Ram Dhari and Yadava (1983), Jindal and Labana (1986), Jain *et al.* (1988) and Singh and Mital (1993) have reported additive and non-additive effects; Gupta *et al.* (1987), Anand and Reddy (1987) reported non-additive; epistasis for this character was reported by Verma *et al.* (1992) and Verma and Singh (1998).

All types of gene effects were observed for 1000-seed weight in different crosses (Table 41). Dominance and additive x additive effects were found desirable in cross No.8 and 11 with high magnitude in cross No. 11. Dominance x additive effect was found desirable in cross No.3 and 12 with high magnitude, additive effect was desirable in cross No. 12 with high magnitude and cross No. 1 showed dominance x dominance gene effect as desirable with low magnitude. Other effects were found undesirable for this character due to their negative nature. This character can also be improved by recurrent selection. Additive and non-additive gene effect were reported by Anand and Reddy (1987), Badwal and Labana (1987), Singh et al. (1989), Rishipal et al. (1993) and epistasis by Verma and Singh (1998) for this character.

Additive, dominance, additive x additive and dominance x dominance type of gene actions were observed for oil content in different crosses (Table 42). Dominance effect was found desirable and fixable in cross No.6, and dominance and additive x additive effects in cross No.7 because of their positive nature and high magnitude in cross No.7 and low magnitude in cross No.6. Other effects were not found desirable due to their negative nature of gene action. Improvement of this character can be done with both additive and non-additive effects by reciprocal recurrent selection. Additive and non-additive effects were reported by Yadava *et al.* (1981), Govil *et al.* (1983) and Dominance effect by Thakral and Singh (1995); additive and dominance by Singh *et al.* (1986, 1989); Pahuja *et al.* (1996) and additive,

dominance and epistasis were reported by Verma *et al.* (1992), Chauhan *et al.* (1996) for this character.

5.5 Identification of desirable crosses and selection of high yielding resistant recombinants

A prerequisite for any successful selection and improvement programme is the existence of genetic variability. The genetic variability available within cultivated varieties of *B. juncea* in India is limited. In recent years enormous variability has been generated in *B. juncea* by their artificial synthesis and hybridization between digenomic brassicas (Raut and Prakash, 1985; Subudhi, 1991). However, their successful utilization in commercial variety breeding is limited. It is possible to use these genetic stocks in different hybridiziation programme to further increase the variability and make effective selection of high yielding genotypes from desirable crosses. After generation of variability, the next immediate step is selection of crosses for, as many as, desirable characters (Table 43). From the ten crosses on which data for quantitative characters were available cross No.3 and No.7 were identified as desirable for many characters. Other crosses were desirable for specific characters. The significant observation to note from this study was the isolation of several high yielding resistant recombinants in F_2 generation itself some of which maintained high yield potentiality in F_3 along with white rust resistance. To further improve the efficiency of selection and isolation of desirable genotypes breeding and selection procedures for specific characters have been suggested.

In conclusion it can be stated that white rust resistance in Brassica is simply inherited governed by one or two pairs of genes mostly dominant. The expression is influenced by the presence of other genes for resistance and the ecological condition of a location favouring disease development.

Dominant genes conferring complete resistance to white rust or a hypersensitive reaction and stable expression which can be easily incorporated to high yielding desirable genetic background appear most desirable. During limited time frame available for this study some such desirable recombinants have been isolated. Results achieved from this study may go a long way in developing suitable cultivars of Indian mustard for successful disease free cultivation under Indian agro-climatic conditions.

SUMMARY

White rust of mustard (*Brassica juncea)* a fungal disease caused by *Albugo candida* is most widespread in India and results in extensive damage to the crop. Genetical resistance is the most economical and preferable method of reducing yield losses due to white rust. Resistance can be most effectively utilized by knowledge of the identity of resistant genes, their mode of inheritance and stability under differing agro-climatic conditions. So far no commercial cultivar is available in India having absolute resistance to white rust. Eventhough resistant gene/genes are available in *B. juncea*, it is necessary to understand their mode of inheritance, association with any morphological trait such as leaf waxiness and seed colour, and also to combine resistance with yield and yield contributing characters for commercial utilization as varieties.

The present study utilized ten different strains of *B. juncea*, all showing resistance to white rust mostly derived from interspecific crosses and stabilised as *B. juncea* and a susceptible standard variety Pusa Bold. Using off-season facility at Wellington (T.N.) 12 resistant F_1s were selected and backcrossed to both the parents to obtain B_1 and B_2 seeds during rabi 1996-97 at Delhi. F_2 populations of selected 12 crosses were raised during summer 1997 (June-Sept.) in off-season nursery at Wellington and Kukumsari (H.P.) and during 1997-98 rabi season, at Delhi on which observations were recorded on disease score. Parents, F_1, B_1 and B_2 generations of selected 10 crosses were grown in randomized block design with 3 replications during rabi 1997-98 at New Delhi. Data for yield and its components and leaf waxiness and seed colour along with white rust

resistance were recorded using 0-5 disease scoring scale after creating artificial epiphytotic condition.

The findings of the present study are summarised as follows:

1. Two crosses (No.1 and 6) were showing digenic control of white rust resistance having 13R:3S segregation ratio in F_2 at all locations, suggesting involvement of two different genes with dominant and recessive interaction.

2. Two crosses (No.2 and 3) were showing monogenic inheritance of white rust resistance having 3R:1S segregation ratio in F_2 involving both resistant parents (one heterozygous) at all locations. It indicates that both parents were having same gene for white rust resistance.

3. Five crosses (No.4,8,9,11,12) were showing monogenic inheritance of white rust giving 3R:1S segregation ratio in F_2 involving one resistant and one susceptible parent, depending on source of white rust resistance gene.

4. One cross (No.7) was showing susceptibility in F_1 and 1R:3S ratio in F_2 involving one resistant and one susceptible parent. It indicated that susceptibility was dominant over resistance showing monogenic recessive inheritance of white rust resistance. Another interpretation could be inability of heterozygotes to confer resistance requiring two dose of dominant gene to confer resistance.

5. One cross (No.5) was showing 15R:1S ratio at Delhi and 3R:1S ratio at Wellington and Kukumsari in F_2 involving both resistant parents. It indicated the possible prevelence of different race of pathogen at Wellington and Kukumsari, which knocked down the resistant gene of one parent.

6. One cross (No.10) was showing 3R:1S ratio at Delhi and 1R:3S at Wellington and Kukumsari in F_2 involving one resistant and one susceptible parent. It indicated that due to the prevelence of different races of pathogen and congenial agro-climatic conditions at Wellington and Kukumsari, the heterozygotes became susceptible altering the ratio.

7. Variation was found in expression of white rust resistance in different crosses which were involving one common female parent in some crosses, depending on the source of white rust resistance gene of male parent and either it was resistant or susceptible. Therefore, these crosses were giving different segregation ratio in F_2 at different locations.

8. Variation was also found in expression of white rust resistance gene under 0-5 disease scoring scale at different locations in the crosses which were showing same segregation ratio in F_2. It was because of different agro-climatic conditions at different locations.

9. Association of leaf waxiness with white rust resistance was found only in five crosses (No.5,6,7,9 and 11) out of 12 crosses studied. It indicated that other remaining seven crosses were showing independence of leaf waxiness with white rust resistance because of the low intensity of waxiness and/or nature of gene governing resistance to white rust in non-waxy parent.

10. An attempt was made to study the association of yellow seed colour with white rust resistance, in our study involving only three crosses, no association was found between yellow seed colour and white rust resistance.

11. All types of gene effects, additive, dominance, additive x additive, additive x dominance and dominance x dominance were found significant contributor for different yield contributing characters of different crosses both in positive and negative direction suggesting reciprocal recurrent selection would be best for improvement of yield and its components in these crosses.

12. Cross No. 3 and 7 were found to have better performing recombinants/segregants for yield and yield contributing characters alongwith white rust resistance. Several other crosses were also found desirable for specific characters.

13. In early generation of F_2 and F_3 some high yielding single plants with white rust resistance were isolated by visual selection.

14. It was concluded that in view of the availability of stable simply inherited genes for white rust resistance high yielding mustard varieties can be evolved for commercial cultivation.

REFERENCES

Abraham, V. and Bhatia, C.R. 1994. Testing for tolerance to aphids in Indian mustard *Brassica juncea* (L.) Czern & Coss. *Plant Breed.*,**112**: 260-263.

Abraham, V. and Kotwal, S.A. 1995. Yellow seeded Trombay mustard varieties with increased oil and protein content. *Cruciferae Newslett.*, **17**: 58-59.

Anand, I.J.; Reddy, W.R. and Rawat, D.S. 1985. Inheritance of seed coat colour in mustard. *Indian J. Genet.*,**45**: 34-37.

Anand, I.J. and Reddy, W.R. 1987. Estimates of gene effects for seed yield and its components in Indian x exotic mustard. *J. Oilseeds Res.*, **4**: 1-8.

Angadi, S.P.; Singh, J.P. and Anand, I.J. 1987. Inheritance of non-waxiness and tolerance to aphids in Indian mustard. *J. Oilseeds Res.*,**4**: 265-267.

Anonymous. 1997. Annual progress report of the AICRP on rapeseed-mustard. NRC-Rapeseed-mustard, Bharatpur, Rajasthan.

Anonymous. 1998. Annual progress report of the AICRP on rapeseed-mustard. NRC-Rapeseed-mustared Bharatpur, Rajasthan.

Ansari, N.A.; Khan, M.W. and Muheet, A. 1988. Effect of *Alternaria* blight on oil content of rapeseed-mustard. *Curr. Sci.*, **57**: 1023-1024.

Asthana, A.N.; Dube, S.D., and Singh, C.B. 1975. Breeding for improved yellow seeded Indian mustard. *Indian J. Genet.*, **35**: 49-54.

Badwal, S.S. and Labana, K.S. 1987. Diallel analysis for some metric traits in Indian mustard. *Crop Improv.*, **14**: 191-194.

Bains, S.S. and Jhooty, J.S. 1979. Mixed infections by *Albugo candida* and *Peronospora parasitica* on *Brassica juncea* inflorescence and their control. *Indian Phytopathol.*, **32**:268-271.

Bhadouria, S.S.; Jain, G., and Tiwari, A.S. 1991. Combining ability analysis in Indian mustard [*Brassica juncea* (L.) *Czern & Coss*]. Golden Jubilee Symposium on Genetic Research and Education: Current Trends and Next Fifty Years, Feb. 12-15, 1991, New Delhi, Abst. Vol. II: 443.

Bisht, I.S.; Agrawal, R.C. and Singh, R. 1994. White rust (*Albugo candida*) severity in mustard (*B. juncea*) varieties and its effect on seed yield. *Plant Varieties and Seeds.*, **7**: 85-89.

Chaudhary, B.D.; Thukral, S.K.; Singh, D.P.; Singh, P. and Kumar, A. 1987. Combining abilities and components of variation in *Brassica compestris. Res. Develop. Reporter.* **4**: 125-129.

Chaudhary, S.K. and Sharma, S.K. 1982. Note on the inheritance of some quantitative characters in a cross of Indian mustard. *Indian J. Agric. Sci.*, **52**: 23-25.

Chauhan, S.S.; Srivastava, R.K.; Kumar, K. and Chauhan, Y.S. 1996. Genetics of oil content in yellow seeded Indian mustard (*Brassica juncea*). *Cruciferae Newslett.* **18**: 80-81.

Chauhan, Y.S. and Kumar, K. 1987. Genetics of seed colour in mustard [*Brassica juncea* (L.) Czern & Coss.]. *Cruciferae Newslett.*, **12**: 22-23.

Chauhan, Y.S.; Kumar, K.; Bhajan, R. and Maurya, K.N. 1995. Inheritance of seed coat colour and its use in determining natural cross pollination in yellow sarson. *Cruciferae Newslett.*, **17**: 60-61.

Chauhan, Y.S. and Singh, D.P. 1980. Inheritance of seed weight in Indian mustard. *Indian J. Genet.*,**40**: 597-599.

Cockerham, C.C. 1954. An extension of the concept of partitioning hereditary variance from analysis of covariances among relatives when epistasis is present. *Genetics*, **39**: 859-876.

Cockerham, C.C. 1963. Estimation of genetic variances. *In*: Statistical Genetics and Plant Breeding. Eds. (W.D.Hanson and H.F. Robinson). NAS-NRC Publ. No. **982**, pp. 53-94.

Cole, C.; Teutonico, R.; Mengistu, A.; Williams, P.H. and Osborn, T.C. 1996. Molecular mapping of a locus controlling resistance to *Albugo candida* in *Brassica rapa. Phytopathology*, **86**: 367-369.

Conn, K.L. and Tiwari, J.P. 1989. Interaction of *Alternaria brassicae* with leaf epicuticular wax of canola. *Mycol. Res.*, **93**: 240-242.

Delwiche, P.A. and Williams, P.H. 1977. Genetic studies in *Brassica nigra* (L.) Koch. *Cruciferae Newslett.* **2**: 39.

Diwakar, M.C. and Singh, A.K. 1993. Combining ability for oil content and yield attributes in yellow seeded Indian mustard [*Brassica juncea* (L.) Czern & Coss.]. *Ann. Agric. Res.*, **14**: 194-198.

Downey, R.K. and Chopra, V.L. 1998. Emerging trends in *Oleiferous brassica. In:* Crop productivity and sustainability-shaping the future. *Proc. 2nd Int. Crop Sci. Cong.* (V.L. Chopra, R.B. Singh, Anupam Verma ed.), pp. 167-179.

Fan, Z.; Rimmer, S.R. and Stefansson, B.R. 1983. Inheritance of resistance to *Albugo candida* in rape (*Brassica napus* L.). *Canadian J. Genet. Cytol.*, **25**:420-424.

Ferreira, M.E.; Williams, P.H. and Osborn, T.C. 1995. Mapping of a locus controlling resistance to *Albugo candida* in *Brassica napus* using molecular markers. *Phytopathology.* **85**:218-220.

*Fisher, R.A. 1918. The correlation between relation on the basis of Mendelian inheritance. *Trans. Roy. Soc. Edinb.*, **52**: 399-433.

Gadewadikar, P.N.; Bhadouria, S.S. and Bartaria, A.M. 1993. Inheritance of resistance to white rust (*Albugo candida*) disease of Indian mustard [*B. juncea*(L.) Czern.]. *In:* Natn. Semin. Oilseeds Res. and Dev. in India: Status and Strategies, Aug.2-5, 1993, 87.

Getinet, A.; Rakow, G. and Downey, R.K. 1987. Seed colour inheritance in *Brassica carinata* A. Braun. Cultivar S-67. *Plant Breed.*, **99**: 80-82.

Gomez, K.A. and Gomez, A.A. 1984. Statistical Procedures for Agricultural Research. John Wiley and Sons, Inc. New York, USA, pp. 680.

Govil, S.K.; Chaubey, C.N. and Chauhan, Y.S. 1981. Combining ability studies in Indian mustard.*Indian J. Agric. Sci.*, **51**: 623-626.

Govil, S.K.; Srivastava, A.N. and Chauhan, Y.S. 1983. Gene action for oil content in Indian mustard. *Indian J. Agric. Sci.*, **53**: 404-406.

Gupta, M.L.; Labana, K.S. and Badwal, S.S. 1987. Combining ability analysis of yield and its components in Indian mustard. 7th Int. Rapeseed. Cong., Poznan, Poland, 11-14 May, 1987, pp. 110.

Gupta, M.L.; Gupta, V.P. and Labana,K.S. 1987. Combining ability analysis of seed yield and its components in Indian mustard. *Crop Improv.*, **14**: 160-174.

Hayman, B.I. 1958. The separation of epistatic from additive and dominance variation in generation means. *Heredity* **11**: 69-82.

Hill, C.B.; Crute, I.R.; Sherriff, C.; and Williams, P.H. 1988. Specificity of *Albugo candida* and *Peronospora parasitica* pathotypes towards rapid-cycling crucifers. *Cruciferae Newslett.*, **13**: 112-113.

*Hiura, M. 1930. Biologic forms of *Albugo candida* (Pers.) Kuntze on some cruciferous plants. *Japanese J. Bot.*, **5**: 1-20.

Jain, A.K.; Tiwari, A.S.; Kushwah, V.S. and Hirre, C.D. 1988. Genetics of quantitative traits in Indian mustard. *Indian J. Genet.*,**48**: 117-119.

Jambhulkar, S.J. and Raut, R.N. 1995. Inheritance of flower colour and leaf waxiness in *Brassica carinata* A. Br., *Cruciferae Newslett.*,17: 66-67.

Jianguo, M.; Wanqu, L.; Qin, Y. and Bodnaryk, R.P. 1995. Inheritance of waxless character of *Brassica napus* Nilla glossy. *Canadian. J. Plant Sci.*, **75**: 893-894.

Jindal, S.K. and Labana, K.S. 1986. Combining ability in a complete diallel cross of Indian mustard. *Indian J. Agric. Sci.*, **56**: 75-79.

Jones, S.S.; Murray, T.D. and Allan, R.E. 1995. Use of alien genes for the development of disease resistance in wheat. *Annu. Rev. Phytopathol.*, **33**: 429-443.

*Jonsson, R. and Bengtsson, L. 1970. Yellow seeded rape and turnip rape.I. Influence of breeding for yellow seeds upon yield and quality properties. *Sveriges Utsadesforen Tidskr.*, **80**: 149-155. (In Swedish with English Summary).

Knott, D.R. and McIntosh, R.A. 1978. The inheritance of stem rust resistance in common wheat cultivar 'Webster'.*Crop Sci.*, **18**: 365-369.

Kolte, S.J. 1985. Diseases of Annual Edible Oilseed crops. Volume II Rapeseed-Mustard and Sesame diseases. CRC Press, Boca Raton Florida, pp. 135.

Kumar, P.R. 1998. Sustaining yellow revolution in Rapeseed-mustard production: New paradigms. Natl. Semin. Rapeseed-Mustard Res. in 21st Century, B.A.U., Kanke, Ranchi, Aug. 6-8.

Kumar, P.; Thakral, N.K.; Yadava, T.P.; Lekh Raj and Naveen Chandra 1998. Inheritance of yield attributing traits in Indian mustard (*Brassica juncea* L.). *Cruciferae Newslett.*, **20:** 73-74.

Kumar, V.; Singh, D.; Pundir, S.R.; Kamboj, M.C.; Chandra, N. and Chandra, N. 1994. Genetics of yield and its components in Indian mustard [*Brassica juncea* (L.) Czern & Coss]. *Crop Res. Hissar,* **7** : 243-246.

Labana, K.S.; Banga, S.S. and Banga, S.K. 1992. Breeding Oilseed Brassicas, Narosa Publishing House, New Delhi.

Labana, K.S.; Chaurasia, B.D. and Sangha, A.S. 1984. Inheritance studies for some yield components in Indian mustard. *Crop Improv.*, 11: 58-60.

Labana, K.S.; Jindal, S.K., and Mehan, D.K. 1978. Heterosis and combining ability in yellow sarson (*Brassica campestris* L. Var. yellow sarson). *Crop Improv.*, **5**: 50-56.

*Lakra, B.S. 1988. Variability, Epidemiology and control of white rust of Rapeseed and Mustard caused by *Albugo candida* (Lev) Kuntze. Ph.D. Thesis submitted to Haryana Agril. University, Hissar.

Lakra, B.S. and Saharan, G.S. 1988. Morphological and pathological variations in *Albugo candida* associated with *Brassica* species. *Indian J. Mycol. Plant Pathol.*,**18**: 149-156.

*Lelivelt, C.L.C. Van; Frederiks, H.J. and Dijk, N. Van. 1988. Transfer of resistance to beet cyst nematode to Swede rape via interspecific crosses with related species. *Prophyta*, **42**: 153.

Liu, J.Q.; Parks, P. and Rimmer, S.R. 1996. Development of monogenic lines for resistance to *Albugo candida* from a Canadian *Brassica napus* cultivar. *Phytopathology*, **86**: 1000-1004.

Liu, Q. and Rimmer, S.R. 1991. Inheritance of resistance in *Brassica napus* to an Ethiopian isolate of *Albugo candida* from *Brassica carinata*. *Canadian. J. Plant Pathol.*, **13**: 197-201.

Liu, Q.; Rimmer, S.R.; Scarth, R. and McVetty, P.B.E. 1987. Inheritance of resistance to *Albugo candida* in *Brassica napus*. *In*: 7th Int. Rapeseed Cong. Poznan, Poland. pp.244.

. Liu, Q.; Rimmer, S.R., Scarth, R. and McVetty, P.B.E. 1987. Confirmation of digenic model of inheritance of resistance to *Albugo candida* race 7 in *Brassica napus*. *Cruciferae Newslett.*, **12**: 1204-1209

Namai, H. 1978. Aspect of transfer of economic characters by means of interspecific and intergeneric crossing in Cruciferous crops. Proc. 5th Int. Rapeseed Conf., June12-16, p. 127-130.

*Napper, M.E. 1933. Observations on spore germination and specialization of parasitism in *Cystopus candidus*. *J. Pomol. Hortic. Sci.*, **11**: 81-100.

Narain, A. 1979. Interspecific utilization of productive and resistant factors in the different species of *Brassica* for the improvement of the Indian digenomic *Brassica* cultivar. *Cruciferae Newslett.*, 4: 12-13.

Nayar, G.G. and George, K.P. 1970. Inheritance of pod arrangement and seed colour in *B. juncea*. *Indian J. Genet.*,**30**: 579-580.

Pahuja, S.K.; Sangwan, R.S.; Arora, R.N., and Jindal, Y. 1996. Gene effects for oil content in Indian mustard [*Brassica juncea* (L.) Czern & Coss]. H.A.U. J. Res., **26**: 163-167.

Paladhi, M.M.; Prasad, R.C.; Bhagwan-Dass and Dass, B. 1993. Inheritance of field reaction to white rust in Indian mustard. *Indian J. Genet.* **53**: 327-328.

Panse, V.G. and Sukhatme, P.V. 1985. Statistical methods for agricultural workers. Indain Council of Agricultural Research, New Delhi.

*Pawlowski, S.H. 1970. Commercial potential of interspecific crosses among several *Brassica* species. Proc. Int. Conf. Sci. Technol. Mark. Rapeseed and Rapeseed Prod., 472-475.

Petrie, G.A. 1988. Races of *Albugo candida* (white rust and staghead) on cultivated cruciferae in Saskatchewan. *Canadian. J. Plant Pathol.*, **10**: 142-150.

Pidskalny, R.S. and Rimmer, S.R. 1985. Virulence of *Albugo candida* from turnip rape (*Brassica compestris*) and mustard (*Brassica juncea*) on various crucifers. *Canadian. J. Plant Pathol.*, **7**: 283-286.

Pound, G.S., and Williams, P.H. 1963. Biological races of *Albugo candida*. *Phytopathology*, **53**: 1146-1149.

Pradhan, A.K.; Sodhi, Y.S.; Mukhopadhyaya, A. and Pental, D. 1993. Heterosis breeding in Indian mustard [*Brassica juncea* (L.) Czern & Coss]:Analysis of component characters contributing to heterosis for yield. *Euphytica*, **69**: 219-229.

Prakash, S. and Chopra, V.L. 1988. Introgression of resistance to shattering in *Brassica napus* from *Brassica juncea* through non-homologous recombination. *Plant Breed.*, **101**: 167-168.

Prakash, S. and Hinata, K. 1980. Taxonomy, cytogenetics and origin of crop *Brassica*, a review. *Opera. Bot.*, **55**: 1-57.

Ram-Dhari and Yadava, T.P. 1983. Estimation of gene-effects for yield and its component traits in Indian mustard. *Indian J. Agric. Sci.*, **53**: 258-260.

Rao, M.V.B. and Raut, R.N. 1994. Inheritance of white rust (*Albugo candida*) in an interspecific cross between Indian mustard (*Brassica juncea*) and rapeseed (*B. napus*). *Indian J. Agric. Sci.* **64**: 249-251.

Raut, R.N. and Prakash, S. 1985. Synthetic Brassicas-New oilseeds for greater production. *In*: V.L. Chopra ed. *Genetic Manipulation For Crop Improvement.* Oxford & IBH, New Delhi. pp. 205-228.

Rawat, D.S. 1989. Inheritance of seed colour in mustard.*Acta Agronomica Hungarica.*, **38**: 127-130.

Rawat, D.S. 1992. Analysis of reciprocal differences in Indian mustard. *Acta Agronomica Hungarica*, **41**: 227-233.

Rishipal; Prakash Kumar and Kumar, P. 1993. Generation mean analysis of yield and its attributes in Indian mustard (*Brassica juncea*). *Indian J. Agric. Sci.*, **63**: 807-813.

Roy, N.N. 1984. Interspecific transfer of *Brassica juncea* high blackleg resistance to *Brassica napus*. *Euphytica*, **33**:295-303.

Roy, N.N. and Tarr, A.W. 1985. IXLIN- an inter-specific source for high linoleic and low linolenic acid content in rapeseed (*B. napus* **L.**). *Zeitschrift fur Pflanzenzuchtung.*, **95**: 201-209.

Sachan, J.N. and Singh, B. 1988. Genetic analysis of quantitative characters in a cross of Indian mustard (*Brassica juncea*). *Indian J. Agric. Sci.*,**58**: 176-179.

Sacristan, M.D. and Gerdemann, M. 1986. Different behaviour of *Brassica juncea* and *B. carinata* as sources of *Phomalingam* resistance in experiments of interspecific transfer to *B. napus*. *Plant Breed.*, **97**: 304-314.

Saharan, G.S.; Kaushik, C.D.; Gupta, P.P. and Tripathi, N.N. 1984. Assessment of losses and control of white rust of mustard. *Indian Phytopathol.* (Abstract).**37**: 397.

Sengupta, K. , and Mukhopadhyaya, S. 1981. Inheritance of glossy stem character in *Brassica juncea* L. Czern. & Coss. *Curr. Sci.*, **50**: 32.

Shirzadegan, M. 1986. Inheritance of seed colour in *Brassica napus* L.*Z. Pflanzenzuchtung.*,**89**: 278-288.

*Shirzadegan, M. and Robbelen, G. 1985. Influence of seed colour and hull proportion on quality properties of seed in *Brassica napus* L. *Fette Seifen Anstrich Mittel.*, **6**: 235-237.

*Shpota, V.I. and Podkolzina, V.E. 1980. Effectiveness of interspecific hybridization in breeding cruciferous oil crops for oil quality. Byul, NTI, Pomaslich Kul'turam., No.**4**: 18-22.

Singh, A.B.; Chauhan, Y.S. and Singh, P. 1981. Genetics of yield in Indian mustard. *Indian J. Genet.*, **41**: 130-136.

Singh, A.B.; Chauhan, Y.S., and Singh, P. 1982. Components of genetic variation in Indian mustard. *Indian J. Agric. Sci.*, **52**: 634-638.

Singh, B.M. and Bhardwaj, C.L. 1984. Physiologic races of *Albugo candida* on crucifers in Himachal Pradesh. *Indian J. Plant Pathol.*, **14**: 25.

Singh, D. and Singh, H. 1987. Genetic analysis of resistance to white rust in Indian mustard. In: 7th Int. Rapeseed Cong. Poznan, Poland, 11-14 May, 1987, pp. 126.

Singh, D. and Singh, H. 1988. Inheritance of white rust resistance in interspecific crosses of *Brassica juncea* x *B. carinata* L. *Crop Res.*, **1**: 189-193.

Singh, D. and Singh, H. 1988. Inheritance studies on white rust (*Albugo candida*) resistance in five interspecific crosses of *B. juncea x B. carinata*. XVI Int. Congr. Genetics, Toronto, Canada: Abstract No. 32: 566.

Singh, D.; Singh, H. and Yadava, T.P. 1988. Performance of white rust (*Albugo candida*) resistance genotypes developed from interspecific crosses of *B. juncea* L. x *B. carinata* L. *Cruciferae Newslett.* **13**: 110-111.

Singh, D.; Sachan, J.N.; Ram Bhajan; Singh, B. and Singh, S.P. 1986. Combining ability in Indian mustard. *J. Oilseeds Res.*, **3**: 1-7.

Singh, G.P. and Mital, R.K. 1993. Combining ability analysis for yield and yield contributing traits in Indian mustard [*Brassica juncea* (L.) Czern & Coss]. *Ann. Agric. Res.*, **14**: 205-210.

Singh, H. and Singh, D. 1987. A note on tansfer of resistance to white rust from Ethiopian mustard to Indian mustard. *Cruciferae Newslett.*, **12**: 95.

Singh, J.N.; Yadav, M.C. and Sheik, I.A. 1996. Genetic studies for yield and oil content in *Brassica juncea* (L.) Czern& Coss. *Indian J. Genet.*,**56**: 299-304.

Singh, O.N. and Chauhan, Y.S. 1987. Triple test cross analysis for seed and oil yield and its components in Indian mustard [*Brassica juncea* (L.) Czern & Coss]. *In* : 7th Int. Rapeseed Cong., Poznan, Poland.

Singh, R.N. and Srivastava, A.N. 1974. Note on breeding behaviour of a yellow seeded rai (*Brassica juncea* L.). *Science and Culture*, **40**: 407.

Singh, R.P. and McIntosh, R.A. 1986. Cytogenetical studies in wheat. XIV. Sr8b for resistance to *Puccinia graminis tritici. Canadian Genet. Cytol.*, **28**: 189-197.

Singh, V.S.; Srivastava, A.N. and Ahmad, Z. 1989. Combining ability in F_1 and F_2 generations of a diallel cross in Indian mustard. *Crop Improv.*, **16**: 164-167.

Sridhar, K. 1996. Genetical and biochemical analysis of fatty acid composition, disease resistance and yield attributes in *Brassica juncea*. Ph.D. Thesis submitted to P.G. School, Indian Agricultural Research Institute, New Delhi.

Stringham, G.R.; McGregor, D.I. and Pawlowsky, S.H. 1974. Chemical and morphological characteristics associated with seed coat colour in rapeseed. Proc. 4th Int. Rapeseed Conf. Giessen, West Germany, p. 99-108.

Subudhi, P.K. 1991. Widening gene pool for resistance to biotic stress and yield components in *Brassica juncea* by interspecific hybridization. Ph.D. Thesis submitted to P.G. School, IARI, New Delhi.

Subudhi, P.K. and Raut, R.N. 1994. White rust resistance and its association with parental species type and leaf waxiness in *Brassica juncea* (L.) Czern & Coss x *B. napus* (L.) crosses under the action of EDTA and gamma-rays. *Euphytica*, **74**: 1-7.

Thakral, N.K. and Singh, H. 1995. Genetic components of seed yield and oil content under normal and saline environments in Indian mustard. *Cruciferae Newslett.*, **17**: 70-71.

Thukral, S.K. and Singh, H. 1986. Inheritance of white rust resistance in *Brassica juncea.Plant Breed.*, **97**: 75-77.

Thukral, S.K.; Yadava, A.K.; Yadava, T.P. and Kumar, P. 1986. Genetics of seed yield and some important attributes in Indian mustard. *Cruciferae Newslett.*, **11**: 31-33.

Tiwari, A.S.; Petrie, G.A. and Downey, R.K. 1988. Inheritance of resistance to *Albugo candida* race 2 in mustard [*Brassica juncea* (L.) Czern]. *Canadian J. Plant Sci.*, **68**: 297-300

Vasanthi, R.P. 1993. Cytogenetical evaluation of interspecific hybrid derivatives of Brassica for resistance, yield and quality. Ph.D. Thesis submitted to P.G. School, IARI, New Delhi.

Vera, C.L.; Woods, D.L., and Downey, R.K. 1979. Inheritance of seed coat in *Brassica juncea. Canadian. J. Plant Sci.*, **59**: 635-637.

Verma, O.P. and Singh, P.V. 1998. Triple test cross analysis in Indian mustard [*Brassica juncea* L. Czern & Coss]. *Cruciferae Newslett.*, **20**: 77-78.

Verma, O.P.; Singh, R.K.; Karan Singh; Singh, P.V. and Singh, K. 1992. Gene effect for six metric traits in Indian mustard (*Brassica juncea*). *Indian J. Agric. Sci.*, **62**: 827-829.

Verma, P.R.; Harding, H.; Petrie, G.A. and Williams, P.H. 1975. Infection and temporal development of mycelium of *Albugo candida* in cotyledons of four *Brassica* spp. *Canadian J. Bot.*, **53**: 1016-1020.

Verma, U. and Bhowmik, T.P. 1989. Inheritance of resistance to a *Brassica juncea* pathotype of *Albugo candida* in *B. napus*. *Canadian J. Plant Pathol.*, **11**: 356-366.

Williams, P.H. and Pound, G.S. 1963. Nature and inheritance of resistance to *Albugo candida* in radish. *Phytopathology*, **53**: 1150-1154.

Woods, D.L. 1980. Association of yellow seed coat with other characteristics in mustard *B. juncea*. *Cruciferae Newslett.*, **5**: 23-24.

Wright, S. 1921. Systems of mating. *Genetics*, **6**: 111-178.

Wright, S. 1935. The analysis of variance and the correlation between relatives with respect to deviation from an optimum. *J. Genetics*, **30**: 243-256.

Yadava, A.K.; Hari Singh and Yadava, T.P. 1985a. Inheritance of non-waxy trait in Indian mustard and its reaction to the aphids. *J. Oilseeds. Res.*, **2**: 120-123.

Yadava, A.K.; Yadava, T.P. and Kumar, P. 1979. Combining ability studies by line x tester analysis in Indian mustard. *Crop Improv.*, **6**: 141-147.

Yadava, C.K. 1983. Studies on genetics of yield and its components in Indian mustard. Thesis Abstract, H.A.U, **9**: 186-187.

Yadav, M.C. 1992. Genetics of seed yield and oil content in *Brassica juncea* (L.) Czern & Coss. M.Sc. Thesis submitted to P.G. School, IARI, New Delhi.

Yadav, O.P.; Yadava, T.P. and Prakash Kumar. 1992. Combining ability studies for seed yield, its component characters and oil content in Indian mustard [*Brassica juncea* (L.) Coss]. *J. Oilseeds Res.*, **9**: 14-20.

Yadav, O.P.; Yadava, T.P. and Kumar, P. 1994. Inheritance of white rust resistance in Indian mustard. *Indian Phytopathol.*, **47**: 159-163.

Yadava, T.P.; Kumar, P., and Yadav, A.K. 1981. Genetic architecture for yield and its component traits in Indian mustard. *Indian J. Agric. Sci.*, **51**: 374-378.

Yadava, T.P.; Prakash Kumar; Thakral, N.K.; Pundir, S.R. and Naveen Chandra. 1993. Genetic analysis for yield components in Indian mustard. *Annals of Biology*. **9**: 52-55.

Yashpal; Hari-Singh and Singh, H. 1991. Genetic components of white rust resistance and seed yield in Indian mustard (*Brassica juncea*). *J. Oilseeds Res.*, **8**: 259-262.

*Yousuf, M.A. 1982. Interspecific hybridization for the breeding and utilization of yellow seeded oilseed *Brassicas*. Ph.D. Thesis, University of Prague, Czechoslovakia (English Abstract).

*Xi Rong, Z.; Zhi Jiang, Z. and Shu Lin, L. 1995. Inheritance of waxless character in rapeseed (*B. napus* L.). *Acta Agric. Shanghai*, **11**: 87-89.

Zaman, M.W. 1989. Introgression in *Brassica napus* for adaptation to the growing condition in Bangladesh. *Theor. Appl. Genet.*, **77**: 721-728.

Zhang, G.; Angeles, E.R.; Abenes, M.L.P.; Khush, G.S. and Huang, N. 1996. RAPD and RFLP mapping the bacterial blight resistance gene Xa-13 in rice. *Theor. Appl. Genet.*, **93**: 65-70.

* Original not seen.

Appendix 1 : Distribution of rainfall, temperature, relative humidity and sunshine hours during crop season 1997-98 in New Delhi.

Month	Rainfall (m m)	Rainy days	Temperature (°C)		Relative humidity (%)	Sun-shine (hr)
			Max.	Min.		
October	65.8	5	33.0	14.0	63.5	6.7
November	17.2	2	28.4	6.8	66.0	5.5
December	6.8	4	23.2	3.8	65.0	4.6
January	0.0	0	23.2	1.5	79.0	1.6
February	29.7	4	27.2	3.8	65.0	7.2
March	40.4	4	31.6	7.8	63.0	5.6

* Data from Annual Progress Report of AICRP on Rapeseed - mustard - 1998.

Appendix 2 : Seed yield (kg/ha) of standard varieties of mustard during 1997 and 1998 in coordinated trials.

Variety	Zone / Year	II			III		
		1997	1998	Reduction over 1997 (%)	1997	1998	Reduction over 1997 (%)
Varuna		2221	1226	44.80	1438	872	39.36
Kranti		2470	1355	45.14	1585	981	38.11
Zonal check		2004	1267	36.78	1402	849	39.44

* Data from Annual Progress Report of AICRP on Rapessed - mustard 1997, 1998.