Gene effects for phenols content in three crosses of Indian mustard (Brassica juncea L. Czern & Coss.)

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Abstract

Higher phenol content, in general, is considered as an important factor to impart disease resistance in *Brassica* species. Therefore, gene effects involved in governing total phenols were studied in six generations i.e. P_1, P_2, F_1, F_2, BC_1 , and BC_2 , of three crosses in Indian mustard. Both, additive as well as non-additive gene effects figured important. Also, epistatic effects were prominent in most of the crosses studied. Based upon the findings of the present study it is advocated that inter-mating in segregating generations would help to accumulate the favorable alleles responsible for the genetic control of phenol content. Selection of desirable types in advance segregating generations would be useful in improving phenol content.

Key words: indian mustard, Brassica juncea, phenols, gene effects, white rust resistance, Albugo candida

Introduction

Indian mustard (*Brassica juncea* (L.) Czern & Coss.) is the most important oilseed *Brassica* species in Indian Subcontinent. This is vulnerable to number of diseases such as *Alternaria blight*, white rust, downy mildew and powdery mildew. Among these white rust has been found to cause severe losses (up to 54.5 per cent) in yield (Saharan *et al.*, 1984) under late sown conditions. All the high yielding cultivars/varieties of Indian mustard are susceptible to this disease. Studies on disease resistance mechanisms at biochemical level have revealed the importance of phenolic compounds, protein, reducing as well as total sugars in different crops (Chand and Verma, 1980; Dhawan *et al.*, 1981 and Gupta *et al.*, 1984). The biochemical basis of resistance, clearly indicated that higher amount of phenols is important for enhancing the level of resistance (Dhawan *et al.*, 1981). Yadav *et al.*, 1996 observed in Indian mustard that white rust resistant genotypes possess higher content of phenols as compared to the susceptible ones. However, there is scanty information in the literature on the mode of inheritance of this trait in Indian mustard but the Knowledge of gene effects governing phenol content would be a prerequisite to initiate a sound breeding programme to develop white rust resistant cultivars in this crop. Therefore, the present investigation was undertaken to study the gene effects for phenol content in three crosses of Indian mustard involving genetically diverse parents.

Material and Methods

Genetically diverse Indian mustard genotypes differing in their response to white rust namely; RH 8113, RC781, UDN69 (resistant), RH9624, Varuna and Sarita (susceptible) were involved in crosses to develop six Generations (P_1 , P_2 , F_1 , F_2 , BC₁ and BC₂) in respect of three crosses viz; RH 8113×RC781 (R×R), UDN69×RH9624 (R×S) and Varuna×Sarita (S×S). Six generations in each of the three crosses were developed during 1999. The experiment to study gene effects for phenol content was conducted in a compact family block design replicated thrice under two environments i.e. normal (E_1 -21st Oct., 2000) and late (E_2 -23rd Nov., 2000) sown at research area of Department of Plant Breeding CCS HAU, Hisar.

The second environment was created by delayed sowing because under normal sown conditions the temperature remain high with low humidity so the chances of inoculum build up of this disease are very less whereas, under late sown conditions low temperature accompanied with high humidity provide better chances for the growth of fungus. Seeds of each of the nonsegregating generations(P_1, P_2, F_1), backcrosses(BC₁, BC₂) and F_2 's were sown in one, two and eight row plots of four meter length in each replications, respectively, The row to row and plant to plant spacings were maintained as 30 cm and 15cm, respectively. The random leaf samples were collected at vegetative (35DAS) and siliquae formation stage (65 DAS) having diseased and healthy leaves and analyzed for total phenols as per Swain and Hillis, (1959). The data was subjected to generation mean analysis to estimate the gene effects following the methods suggested by Cavalli (1952) and Jinks and Jones (1958) to judge that how best the model fit well in each cross.

Results and Discussion

The results obtained in present studies revealed that phenol content was, in general higher in E_1 as compared to E_2 at 35 DAS as well as 65 DAS irrespective of resistance or susceptibility of parents to white rust. The reduction in phenolic compounds was more at 65 DAS than at 35 DAS. Guleria and Kumar, 2003) also observed that the phenolic constituents were generally higher in resistant cultivars than susceptible ones. More reduction in the phenolic constituents was observed in susceptible cultivars as compared to resistant genotypes. Kumar et al. (2002) also reported more reduction in phenolic compounds after infection of white rust in susceptible than resistant genotypes in Indian mustard. It was found in the

experiment undertaken that the disease incidence was more in E_2 at 65 DAS as compared to E_1 and at 35 DAS. Considering E_2 as most favourable environment and 65 DAS as most appropriate plant growth stage for the disease development. A comparative evaluation for phenolic content among parents revealed interestingly that phenolic content in most of the parents was at par with each other at 65 DAS in E_2 . However, after infection susceptible parents RH 9624, Varuna and Sarita exhibited 31.07 to 38.03% reduction in phenol content incidentally these genotypes were also at par with each other for phenol content at 65 DAS post infection. The resistant parents, however, revealed much lower reduction in phenol content (4.73 to 13.36%) at 65 DAS post infection. Similar results were reported by Gupta *et al.* (1990) in case of *Alternaria blight* of mustard. They also reported that magnitude of post infection reduction in phenolic compounds was more in susceptible cultivars as against resistant ones. Therefore, the genotypes which have the ability to maintain the values of phenolic compounds after the incidence of disease are more important for white rust resistance. The F_1 ^{*s} exhibited in general, either intermediate values of parental genotypes for phenol content or tended towards the parent possess low phenol contents. However, the crosses involving both or at least one of the resistant genotypes, the F_1 ^{*s} at 65 DAS showed considerably higher phenol content.

The results of the present studies revealed that the additive –dominance model holds adequacy for the crosses RH $8113 \times RC$ 781 at vegetative stage and UDN 69x RH 9624 at siliqua formation stage under healthy conditions (E₂). Whereas, for other crosses additive-dominance model was found inadequate, indicating the presence of non-allelic interactions. Therefore, the data was subjected to work out the digenic non-allelic interactions.

The additive as well as non-additive gene effects were found significant in all three crosses under most of the situations. The dominance gene effects had higher magnitude than the additive gene effects. The additive xadditive type of interactions were significant in crosses RH 8113×RC 781 in E1 and E2 under both the stages; UDN 69×RH 9624 under diseased (D) conditions only and in the cross Varuna×Sarita across the environments and stages except at siliqua formation stage in healthy leaves under late sown conditions. Additive x additive kind of interactions being fixable in nature and can be exploited for further improvement through simple selection. The additive×dominance type of interactions were found significant in crosses RH 8113×RC 781 at siliqua formation stage in both the environments; UDN 69×RH 9624 at vegetative stage under late sown conditions and in E₁ at siliqua formation stage; Varuna×Sarita across the environments and stages except at siliqua formation stage under late sown conditions. The dominance x dominance types of interactions were found to be significant for crosses RH 8113×RC 781 at siliqua formation stage under late sown conditions (healthy and diseased); UDN 69×RH 9624 under diseased conditions and Varuna×Sarita in both the environments at vegetative stage and at siligua formation stage under late sown conditions (healthy leaves). Duplicate type of epistasis was also found in crosses RH 8113×RC 781 at siliqua formation stage under late sown conditions, UDN 69×RH 9624 under diseased conditions whereas, Varuna×Sarita under late sown conditions at vegetative stage. Similar results were reported by Yadav et al. (1996) in Indian mustard and Singh (1989) in guar. However, a supplementary study on a larger number of genotypes and these crosses is needed to establish a correlation between phenol content and disease resistance over time and space so as to chalk out a coherent strategy for breeding white rust resistance in Brassica. Considerable proportion of additive as well as additive×additive gene effect for phenol content in the crosses RH 8113×RC 781 in both stages and environments whereas, UDN 69×RH 9624 under diseased condition only, warrant use of simple pedigree selection for further improvement. On the other hand intermating in advance segregating generations followed by delayed selection will in general, be useful to improve any trait, when additive and non-additive gene effects with epistatic effects are significant. This kind of breeding approach will be helpful in accumulating favorable alleles, responsible for the genetic control of phenol content.

Conclusion

The higher amount of phenol content as well as its stability across the environments of utmost interest to plant breeders engaged in breeding for white rust resistant genotypes in oilseed *Brassica* species. Therefore, the efforts should be made to further enhance phenol content to a desired level with its stability under heavy disease pressure. Based upon, the genetic information generated on the breeding material studied in the present study, it is advocated that intermating in segregating generations of selected stable plant progenies followed by selecting in advance generation would help to enhance the level of phenolic constituents and their stability with resistance to white rust.

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| Cross | Stage | Environment | Gene effects | | | | | | Type of | Joint | |
|--------------|----------------------------------|----------------|---------------|-------------|----------------|----------------|------------------|--------------|----------|-------------------------|--|
| | | | m | (d) | (h) | (i) | (j) | (1) | Epitasis | scaling test $(\chi 2)$ | |
| | | | Total Phenols | | | | | | | | |
| RH8113 | Vegetative (35 DAS) | E_1 | 28.83±6.82 | **9.46±0.21 | 21.87±14.95 | **18.28±6.81 | -0.36±2.76 | 3.58±8.32 | - | **54.42 | |
| ×RC781 | Siliqua | E ₂ | | - | - | _ | — | - | - | 5.14 | |
| | formation (65 DAS) | E_1 | 41.89±5.05 | **4.20±0.22 | **-28.18±10.70 | **-18.32±5.05 | **11.34±1.59 | 8.50±5.74 | - | **118.24 | |
| | | $E_2(H)$ | 36.55±4.52 | **0.96±0.12 | **-44.86±9.85 | **-16.37±4.52 | **7.96±1.76 | **24.75±5.44 | D | **33.15 | |
| | | $E_2(D)$ | 12.56±2.33 | 0.01±0.06 | **16.12±4.92 | *5.76±2.32 | **4.25±0.71 | **-7.47±2.64 | D | **53.72 | |
| UDN 69 × | Vegetative (35 DAS) | E_1 | 25.44±4.27 | **5.09±0.25 | -3.42±9.12 | 3.41±4.27 | 1.80±1.47 | 2.62±4.94 | _ | **21.84 | |
| RH9624 | | E_2 | 22.75±3.70 | **1.95±0.11 | -1.48 ± 1.02 | -2.23 ± 3.70 | *2.81±1.38 | -2.84±4.41 | - | **30.89 | |
| | Siliqua formation (65 DAS) | E_1 | 20.18±5.66 | **4.46±0.25 | 6.13±12.55 | 1.82±5.65 | **7.86±2.45 | -8.82±7.05 | - | **21.84 | |
| | | $E_2(H)$ | - | - | - | _ | - | - | - | 3.86 | |
| | | $E_2(D)$ | 22.83±2.00 | **0.96±0.07 | **-10.22±4.39 | *4.23±2.00 | -0.83 ± 0.80 | **7.17±2.49 | D | *11.10 | |
| Varuna | Vegetative (35 DAS) | E_1 | 21.29±4.96 | **2.46±0.46 | 18.11±11.10 | *10.49±4.94 | **10.07±2.36 | *14.16±6.32 | - | **23.77 | |
| ×Sarita | | E_2 | 52.04±4.18 | **1.85±0.27 | **-49.65±8.90 | **-21.92±4.17 | *3.04±1.43 | **26.34±4.80 | D | **33.11 | |
| | Siliqua formation (65 DAS) | E_1 | 10.93±3.25 | **2.22±0.23 | *14.16±7.21 | **8.72±3.24 | **-8.89±1.45 | -6.50±4.06 | - | **53.06 | |
| | ``` | $E_2(H)$ | 28.02±4.43 | *0.31±0.12 | -17.59±9.42 | -7.68±4.42 | -2.18±1.44 | *10.33±5.07 | — | **11.42 | |
| + ++ 0' - '0 | 50/ . 1.10/ | $E_2(D)$ | 16.54±10.24 | 0.11±0.12 | *-7.72±3.61 | *-3.66±1.61 | -0.84±0.75 | 2.61±2.05 | - | **11.64 | |

*,** Significant at 5% and 1% level, respectively, ► - Denotes Additive-Dominance model