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DISSECTING GENE EFFECT AND COMBINING RESISTANCE GENE FOR ENHANCED TOLERANCE TO *ALTERNARIA* BLIGHT AND WHITE RUST IN INDIAN MUSTARD (*Brassica juncea* L. CZERN & COSS)

Manidipa Pan, Ayushi Singh*, Anu Singh, Kajal Mehra, Anil Kumar, A.S. Jeena and Usha Pant*

Department of Genetics and Plant Breeding, College of Agriculture, Govind Ballabh University of Agriculture and Technology, Pantnagar, 263145, U.S. Nagar, Uttarakhand, India

*Corresponding authors E-mail: ushapantgpb@gmail.com; singhayushi2000@gmail.com

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ABSTRACT

To understand the genetic basis of resistance to *Alternaria* blight and White rust in *Brassica juncea* and to assess its implications for gene pyramiding, the study was conducted during the *rabi* season of 2021-2022 and 2022-2023 at NEBCRC, GBPUAT, Pantnagar. Six generations (P1, P2, F1, F2, BC1, BC2) from four crosses involving *Alternaria* blight-tolerant (PAB 17-3, PAB 9511, PHR-2, PAB 17-1) and white rust-resistant genotypes (Donskaja and PWR 13-8-7) were evaluated under natural field conditions using a Compact Family Block Design. Analysis of variance revealed significant differences among generations and families for disease and yield traits. Gene effect estimation through scaling and joint scaling tests revealed that both additive [d] and non-additive effects, particularly dominance [h] and dominance × dominance [l], were involved in governing resistance. Duplicate epistasis was frequently observed, suggesting complex inheritance patterns. Furthermore, the natural disease scoring data provided a strong genetic foundation for pyramiding resistance genes. The expression of complementary resistance across backcross generations supports the potential for combining multiple resistance sources. Incorporating molecular markers and epiphytotic screening in future breeding programs will enhance selection accuracy and accelerate the development of cultivars with durable, broad-spectrum disease resistance.

***Key words*:** Disease resistance, gene effects, pyramiding

Introduction

Brassicas, commonly known as rapeseed-mustard, is an important oilseed after soybean and palm, with significant domestic demand for mustard oil and animal feed, this crop plays a pivotal role in world economy. Globally, rapeseed-mustard group of crops ranks first in oil seed production and contributed to 13.16 million tonnes (33.24%) of oilseed production in 2023–24 (DAC&FW 2024). Among the various species of rapeseed-mustard, Indian mustard (*Brassica juncea* L. Czern & Coss.; AABB; 2n = 36) is the principal source of edible oil in the Indian subcontinent. It is currently cultivated on approximately 9.183 million hectares, producing around 13.255 million tonnes, with Rajasthan accounting for about 43% of the total production (DAC&FW 2024).

In recent years, the production of oilseed *Brassica* has increased a lot, owing to the high-yielding varieties, biotechnology, and improved farming practices, still there exist a persistent gap between domestic edible oil production and consumption, leading to costly imports that strain the economy. A major factor behind this shortfall is the devastating impact of fungal diseases, particularly *Alternaria* blight and white rust. *Alternaria* blight, caused by *Alternaria brassicae* and *A. brassicicola*, leads to dark brown concentric spots on leaves and can cause yield losses of 32–38% (Dharmendra *et al.*, 2014), with recent regional surveys confirming its high prevalence

(Babli *et al*., 2022).White rust, caused predominantly by two races of *Albugo candida*, is equally damaging, resulting in white pustules, malformed "stag-head" inflorescences, and yield losses ranging from 17% to 55% (Petrie 1988; Rimmer *et al*., 2000; Singh *et al*., 2022). While resistant varieties exist, their mitigation is often incomplete, as resistance to one disease does not confer resistance to the other. Moreover, reliance on single-gene resistance increases the risk of pathogen adaptation.

Compounding the problem, current resistant varieties often fail under field conditions where both diseases co-occur. This vulnerability underscores the urgent need for more durable solutions. To broaden host resistance against the wider range of pathogen races, gene pyramiding will offer durable resistance and promote the sustainable establishment of crop in the field areas (Watson and Singh, 1953; Servin *et al*., 2004; Malav *et al*., 2016). This research focuses on testing the adequacy of genetic models to understand the inheritance patterns and gene interactions underlying resistance. It also explores future prospects for pyramiding resistance genes for *Alternaria* blight and white rust into elite Indian mustard backgrounds, ultimately contributing to the development of resilient, high-yielding cultivars.

Material and Methods

The experimental material consisted of four *Brassica juncea* genotypes tolerant to *Alternaria* blight (PAB 17-3, PAB 9511, PHR-2, and PAB 17-1) and two genotypes tolerant to white rust (Donskaja and PWR 13-8-7). These were evaluated at NEBCRC, GBPUAT, Pantnagar. During the *rabi* season of 2021–22, four F_1s, initially provided for the study and derived from crosses between *Alternaria* blight-tolerant and white rust-tolerant lines, were used to generate six generations. These F_1s were backcrossed with their respective parents to develop BC_1 and BC_2 generations, while F_2 seeds were obtained through selfing, resulting in four segregating families as shown in Table 1. In the subsequent *rabi* season of 2022-23, each family comprising six generations viz. P_1, P_2, F_1, F_2, BC_1, and BC_2 was evaluated using a Compact Family Block Design with three replications. Generations within families were randomized, and each plot was laid out with standard spacing to ensure uniformity in field conditions.

Further assessment of disease scoring for *Alternaria* blight and white rust was conducted at both the leaf and siliqua stages was carried out following the standard protocols prescribed by the All India Coordinated Research Project (AICRP) on rapeseed-mustard. Ratings were taken based on visual symptom expression, as outlined in Table 2, to ensure consistent screening across generations.

To estimate gene effects and assess the adequacy of genetic models, Simple Scaling Tests were conducted and further validated using the Joint Scaling Test (Cavalli 1952), which is widely used for detecting epistasis and estimating genetic parameters such as [m], [d], [h], [i], [j], and [l]. This method also enables evaluation of model adequacy by comparing observed generation means with expected values derived from the estimated parameters. In cases where the additive-dominance model (three-parameter model) did not provide a satisfactory fit, the six-parameter model approach of Hayman and Mather (1958) was employed. This approach provides a comprehensive understanding of both additive and non-additive genetic effects, including various types of epistasis, thus offering a more robust interpretation of the inheritance pattern in segregating generations.

Result and Discussion

The pooled ANOVA for *Alternaria* blight and white rust showed significant variation among progenies for white rust on leaves ($p < 0.01$), while variations for *Alternaria* blight on leaves and siliqua, and white rust on siliqua were non-significant as shown in the Table 3. Replications, families, and errors showed no significant differences.

The analysis of scaling tests for *Alternaria* blight on leaves, on siliquae and white rust on leaves is displayed in Table 4. The scaling test analysis for *Alternaria* blight

Table 1: Description of Families.

Family A	Family B	Family C	Family D
PAB17-3	PHR-2	PAB17-1	PAB9511
Donskaja	PWR 13-8-7	PWR 13-8-7	Donskaja
PAB17-3 × Donskaja	PHR-2 × PWR 13-8-7	PAB17-1 × PWR 13-8-7	PAB9511 × Donskaja
PAB17-3 × Donskaja	PHR-2 × PWR 13-8-7	PAB17-1 × PWR 13-8-7	PAB9511 × Donskaja
(PAB17-3 × Donskaja) × PAB17-3	(PHR-2 × PWR 13-8-7) × PHR-2	(PAB17-1 × PWR 13-8-7) × PAB17-1	(PAB9511 × Donskaja) × PAB9511
(PAB17-3 × Donskaja) × Donskaja	(PHR-2 × PWR 13-8-7) × PWR 13-8-7	(PAB17-1 × PWR 13-8-7) × PWR 13-8-7	(PAB9511 × Donskaja) × Donskaja

Table 2: Criteria for disease scoring as per AICRP guidelines.

Disease Score	% Disease Incidence	Inference
0	Nil	Immune
1	<5%	Highly resistant
3	5 – 10%	Resistant
5	11 – 25%	Moderately resistant
7	26 – 50%	Susceptible
9	>50%	Highly susceptible

on leaves demonstrated significant values for all four scales (A, B, C, and D) in Families A, C, and D, while only scale B was significant in Family B. In the case of *Alternaria* blight on siliquae, significant scale estimates were detected in all families. Specifically, scales C and D were significant in Family A; scales A, B, and C in Families B and C; and scales A, C, and D in Family D. These results consistently point toward the presence of non-allelic interactions influencing resistance in siliquae. Furthermore, the estimates for scaling test in white rust on leaves showed significance in scale C for Family A, scales B and D for Family B, and scales A, B, and D for Family D. Family C, however, did not show any significant estimates.

The consistent significance of multiple scaling tests across traits and families indicates the involvement of complex gene interactions, particularly epistasis, in the inheritance of resistance to *Alternaria* blight and white rust. For *Alternaria* blight on both leaves and siliquae, non-allelic interactions were evident across most families, making simple additive-dominance models insufficient. The expression of white rust resistance varied by family, with non-allelic interactions prevalent in most, but not all, genetic backgrounds highlighting the need for trait-specific and family-specific breeding strategies.

The estimates of genetic parameters under an adequate genetic model for each family, along with the respective chi-square values and the type of epistasis for *Alternaria* blight on leaves, on siliquae, and white rust on leaves, are shown in Table 5.

Table 3: Pooled Analysis of Variance (Compact Family Block Design) of different families for *Alternaria* Blight and White Rust.

Source of variation	d.f.	Mean sum of squares		
		Alternaria Blight		White Rust
		On Leaves	On Siliqua	On Leaves
Replications	2	0.249	1.486	1.741
Families	3	1.535	0.181	0.471
Error (a)	6	1.028	1.150	0.895
Progenies	20	0.219	1.125	1.446**
Error (b)	40	0.954	1.021	0.683

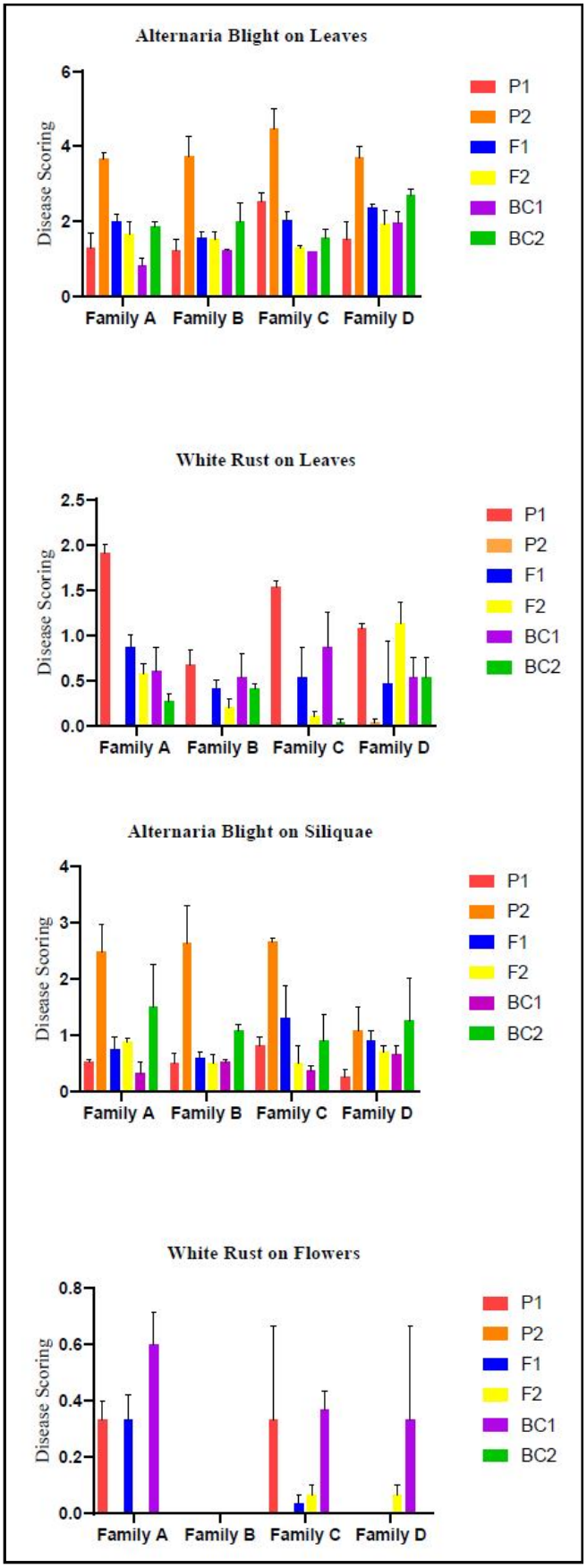


Fig. 1: Family wise disease scoring data for Alternaria blight and white rust.

Table 4: The Estimates of Scaling tests for disease.

Observation	FAMILIES				
	Scales	Family A	Family B	Family C	Family D
***Alternaria* blight on leaves**	A	0.31± 0.56*	0.23± 0.48	0.80± 0.28**	0.13± 0.36**
	B	0.11± 0.33**	0.94± 0.30*	0.10± 0.31**	0.22± 0.47**
	C	0.36± 0.60**	1.17± 1.08	0.14± 0.37**	0.81± 0.90**
	D	0.57± 0.24**	0.26± 0.51	0.38± 0.19**	0.15± 0.39**
***Alternaria* blight on siliquae**	A	1.06± 0.88	0.38± 0.19**	0.38± 0.62*	0.95± 0.30**
	B	2.28± 1.51	0.27± 0.51**	1.16± 1.08*	0.17± 0.41
	C	0.36± 0.60**	0.42± 0.64**	2.98± 1.72*	0.18± 0.43*
	D	0.82± 0.90*	0.14± 0.37	0.63± 0.79	0.83± 0.28**
White rust on leaves	A	0.36± 0.60	0.60± 0.24	0.24± 0.49	0.24± 0.49*
	B	0.31± 0.56	0.14± 0.37**	0.13± 0.36	0.45± 0.21**
	C	0.49± 0.70*	0.88± 0.29	1.33± 1.15	0.44± 0.66
	D	0.20± 0.45	0.65± 0.25*	0.25± 0.50	0.15± 0.39*
****=Significant at 1% probability level; *=Significant at 5% probability level**					

For resistance to *Alternaria* blight on leaves, digenic interaction models proved adequate for all families, supported by non-significant chi-square values (0.28 in Family A, 0.24 in Family B). Family A exhibited significant additive [d] (-1.07**) and dominance [h] (1.55*) effects, along with strong additive × additive [i] (1.99**) and dominance × dominance [j] (2.98**) interactions, indicating both additive and epistatic gene actions. Similarly, Family B demonstrated strong additive [d] effect (-1.49**) and significant [j] interaction (1.43*), suggesting additive effects played a greater role. Meanwhile, Families C and D showed evidence of duplicate epistasis, as seen in high dominance [h] and [l] values with opposite signs ([h] = -21.36**, [l] = 12.33** in Family C). Consequently, additive effects were more prominent in Families A and B, while Families C and D required complex approaches due to dominant and epistatic interactions. Regarding siliquae resistance, parameter estimates further revealed that additive gene effects [d] played a major role in reducing blight symptoms in Families A and D ([d] = -1.21** in A and -0.58* in D), favoring simple breeding methods. Conversely, Families B and C were more influenced by dominance [h] and epistatic interactions, with Family B showing duplicate epistasis ([h] = -5.85**, [l] = 3.75**), indicating complex gene interactions. These contrasting patterns highlight the need to tailor breeding strategies based on the underlying gene action for improving resistance to *Alternaria* blight on siliquae.

For white rust resistance on leaves, parameter estimates showed that dominance [h] was the key factor in improving resistance in Families A and C ([h] = -0.67** in A and -1.00** in C), both fitting additive-dominance models. In contrast, Families B and D were governed by dominance × dominance [l] interaction ([l] = -1.05** in B and -2.24** in D), with Family B also showing duplicate epistasis, indicated by opposite signs of [h] and [l]. All families maintained non-significant chi-square values (0.62 in A, 0.37 in B), confirming model adequacy.

The outcomes of chi-square tests across all eighteen traits confirmed the adequacy of an epistatic genetic model, indicating a significant role of non-allelic interactions in trait expression. The involvement of epistasis in Indian mustard has also been substantiated by earlier studies, including those by Tripathi *et al*., (2005), Pathak (2016), Manjunath *et al*., (2017), Philanim *et al*., (2019), Meena *et al*., (2019), Dahiya *et al*., (2019), and Kandpal (2020), even under diverse genetic backgrounds and cross combinations. In the absence of epistasis, simple breeding strategies such as direct selection may suffice. However, the presence of significant epistatic interactions necessitates the adoption of more complex approaches like the pedigree or bulk method, which are more suited for capturing non-additive genetic variation and exploiting heterosis effectively.

Moreover, these results highlight the predominance of both heritable ([d], [i]) and non-heritable ([h], [l]) gene effects, with duplicate epistasis observed for disease resistance traits. This genetic complexity underscores the need for breeding strategies that utilize both additive and non-additive gene actions. Approaches such as reciprocal recurrent selection, biparental mating, and selective diallel mating are effective for exploiting additive × additive interactions (Raliya *et al*., 2018; Meena *et al*., 2019), while non-fixable epistasis, such as additive × dominance [j] and dominance × dominance [l], can be effectively harnessed through hybrid development. Additionally, the diallel selective mating system enables the development of phenotypically stable yet genetically diverse Indian mustard varieties. Overall, the data demonstrate that the inheritance of resistance to *Alternaria* blight in these

Table 5: Estimates of genetic parameter under the adequate genetic model with respective χ^2 value and type of epistasis involved for *Alternaria* blight on leaves, *Alternaria* blight on siliquae and white rust on leaves

Observation	Family	Model	[m]	[d]	[h]	[i]	[j]	[l]	χ^2 Value	Type of Epistasis
Alternaria blight on leaves	Family A	DI (5PM)	0.64±0.20*	-1.07±0.21**	1.55±0.35*	1.99±0.27**	2.98±0.54**		0.28	
	Family B	DI (4PM)	2.69±0.14**	-1.49±0.18**	-1.14±0.24**		1.43±0.47*		0.24	
	Family C	DI (5PM)	10.90±0.41**	-1.23±0.13**	-21.36±1.13**	-7.13±0.39**		12.33±0.74**	0.17	Duplicate epistasis
	Family D	DI (5PM)		-0.89±0.17**	5.18±0.41**	2.85±0.17**	5.05±0.49**	-3.38±0.57**	0.95	Duplicate epistasis
Alternaria blight on siliquae	Family A	DI (3PM)	0.31±0.86*	-1.21±0.74**		1.43±0.11**			0.70	
	Family B	DI (5PM)	2.53±0.12**	-1.03±0.12**	-5.85±0.53**		2.02±0.45**	3.75±0.47**	0.62	Duplicate epistasis
	Family C	AD (3PM)	1.60±0.88**	-0.95±0.87**	-1.67±0.27**				0.73	
	Family D	DI (3PM)	2.78±0.58**	-0.58±0.11*	3.59±0.57**				0.34	
White rust on leaves	Family A	AD (3PM)	0.70±0.10**	0.57±0.10**	-0.67±0.20**				0.62	
	Family B	DI (4PM)		0.18±0.32*	1.35±0.22**	0.31±0.33**		-1.05±0.24**	0.37	Duplicate epistasis
	Family C	AD (3PM)	1.12±0.65**	1.06±0.62**	-1.00±0.15**				0.18	
	Family D	DI (4PM)	0.50±0.58**	0.43±0.57**	1.93±0.33**			-2.24±0.37**	0.10	

AD=Additive-Dominance model; DI=Digenic-interaction model; PM=Parameter model;
*=significant at 5% probability level; **=significant at 1% probability level

genotypes is governed by more than just simple additive or dominance gene action. Epistatic interactions, especially duplicate epistasis, are evident and suggest that breeding strategies must account for these complexities to effectively improve disease resistance.

Pyramiding of *Alternaria* Blight and White Rust Resistance

Following the estimation of genetic parameters and identification of gene effects, efforts are made in establishing a robust genetic basis for pyramiding resistance to *Alternaria blight* and *white rust.* For this purpose, four families (A,B,C and D) were generated by crossing *Alternaria* blight-tolerant and white rust-resistant lines, with successive generations (F_1, F_2, BC_1, BC_2) evaluated under natural field conditions for disease expression on leaves, siliquae, and flowers. The presence of additive and dominance gene effects, along with duplicate-type epistasis, confirms the polygenic nature of disease resistance and the feasibility of combining favorable alleles from both parents. The F_1 hybrids exhibited intermediate disease scores, suggesting partial dominance of resistance traits. F_2 populations showed segregation for both traits, with generally lower disease scores than F_1, indicating potential for selection. For example, in Family A, BC_1 showed improved tolerance to *Alternaria* blight (1.4) and white rust (0.6), while BC_2 showed enhanced white rust resistance (0.2). Similar trends were observed in other families, with BC_1 often closer to the *Alternaria* blight tolerant parent and BC_2 to the white rust resistant parent. In BC_1 and BC_2 generations, disease scores for *Alternaria* blight and white rust were further reduced, reflecting successful introgression of resistance alleles from respective tolerant parents. The natural disease scoring data supported, presented in Table 6 and displayed in Fig. 1, showed that the trait expression is influenced by the parental genotype combination, and disease resistance could be improved through backcross breeding. However, minor inconsistencies due to environmental variation in natural screening were noted, suggesting the need for epiphytotic screening and molecular tools to ensure stable selection. Integration of molecular tools like marker-assisted selection (MAS) and epiphytotic screening can further refine this process, enabling early and accurate identification of resistant genotypes. Such advancements will facilitate cultivar development and strengthen long-term disease management in Indian mustard.

The present investigation concludes that the genetic architecture underlying resistance to *Alternaria blight* and *white rust* in Indian mustard through pooled ANOVA,

Table 6: Family wise disease scoring data for *Alternaria* blight and white rust.

Family	Generation	DISEASE SCORING															
		ALTERNARIA BLIGHT								WHITE RUST							
		On leaves				On siliqua				On leaves				On flowers (Staghead)			
		R_1	R_2	R_3	Average	R_1	R_2	R_3	Average	R_1	R_2	R_3	Average	R_1	R_2	R_3	Average
A	P_1	0.6	1.3	2	1.3	0.5	0.6	0.5	0.5	2.1	1.8	1.8	1.8	0.4	0.4	0.2	0.3
	P_2	3.6	4	3.4	3.3	3.2	2.7	1.5	2.5	0	0	0	0	0	0	0	0
	F_1	1.8	2.4	1.8	2.4	0.6	1.2	0.4	0.7	1	0.6	1	0.9	0.2	0.5	0.3	0.3
	F_2	1.4	1.3	2.3	2	0.8	0.8	1	0.8	0.4	0.5	0.8	0.3	0	0	0	0
	BC_1	0.7	0.6	1.2	1.4	0.7	0.1	0.2	1	0.5	0.2	1.1	0.6	0.4	0.6	0.8	0
	BC_2	2.6	1.6	2	2.1	2.3	2.2	0	1.5	0.4	0.3	0.1	0.2	0	0	0	0
B	P_1	1.1	0.8	1.8	1.2	0.8	0.2	0.5	0.5	0.4	0.6	1	1.3	0	0	0	0
	P_2	2.8	3.8	4.6	3.7	3.7	1.4	2.8	2.6	0	0	0	0	0	0	0	0
	F_1	1.4	1.4	1.9	1.6	0.5	0.8	0.5	0.9	0.2	0.5	0.5	0.4	0	0	0	0
	F_2	1.2	1.8	1.6	1.5	0.4	0.8	0.3	0.5	0.3	0	0.3	0.2	0	0	0	0
	BC_1	1.2	1.3	1.2	1.2	0.6	0.5	0.5	0.5	0.8	0	0.8	0.6	0	0	0	0
	BC_2	1	2.4	2.6	2	0.9	1.3	1	1.07	0.3	0.5	0.4	0.4	0	0	0	0
C	P_1	3	2.2	2.4	2.5	0.5	1.1	0.8	0.6	1.4	1.6	1.6	1.5	1	0	0	0.3
	P_2	5.2	4.8	3.4	4.5	2.8	2.6	2.6	2	0	0	0	0	0	0	0	0
	F_1	1.8	1.8	2.5	2.03	0.5	2.4	1	1.3	0.2	1.2	0.2	0.5	0	0	0.1	0.3
	F_2	1.3	1.2	1.4	1.3	0.4	1.1	0	0.5	0.2	0.1	0	0.1	0.1	0	0.1	0.7
	BC_1	1.2	1.2	1.2	1.2	0.4	0.5	0.2	0.3	0.1	1.1	1.4	0.9	0.3	0.3	0.5	0
	BC_2	1.5	2	1.2	1.6	1.8	0.6	0.3	0.9	0.1	0	0	0	0	0	0	0
D	P_1	2.3	1.6	0.7	1.5	0.4	0	0.4	0.3	1.2	1	1	1.07	0	0	0	0
	P_2	3.7	3.2	4.2	3.7	0.3	1.8	1.1	1.07	0.1	0	0	0	0	0	0	0
	F_1	2.4	2.2	2.5	2.4	0.9	1.2	0.6	0.9	0	0	1.4	0.5	0	0	0	0
	F_2	1.3	2.6	1.9	1.9	0.5	0.9	0.7	0.7	1.5	0.7	1.2	1.13	0.1	0.1	0	0.1
	BC_1	2.4	1.4	2.1	1.8	0.9	0.4	0.7	0.7	0.1	0.7	0.8	0.5	1	0	0	0.3
	BC_2	2.8	2.4	2.9	2.75	2.6	0	1.2	1.3	0.1	0.7	0.8	0.5	0	0	0	0

scaling tests, and genetic parameter estimation across four biparental families (A, B, C and D). Significant non-allelic interactions, particularly duplicate epistasis, were detected for most traits, affirming the complexity of gene action beyond simple additive-dominance models. Additive effects predominated in Families A and B, while Families C and D were influenced by dominant and epistatic interactions. The adequacy of digenic interaction models was confirmed by non-significant chi-square values, and consistent significance of multiple scaling tests suggested the involvement of epistasis in trait inheritance. Backcross generations (BC_1, BC_2) showed improved resistance scores, validating the successful introgression and pyramiding of resistance alleles. The polygenic nature of disease resistance and segregation patterns in F_2 populations support the feasibility of genetic enhancement through backcross breeding. However, genotype-by-environment interactions necessitate the incorporation of epiphytotic screening and molecular tools such as marker-assisted selection (MAS) for precise and stable selection. The integration of classical and molecular breeding approaches is imperative to exploit both additive and non-additive genetic variance effectively. Collectively, these findings provide a robust genetic framework for pyramiding disease resistance and form the basis for developing durable, disease-resistant Indian mustard cultivars.

Authorship Contribution Statement

- Mandipa Pan executed the experiments and performed primary data recording.
- Anu Singh contributed to data recording.
- Ayushi Singh executed figurative data representation and co-wrote the manuscript with Kajal Mehra.
- Usha Pant conceptualized and designed the study, supervised the experimental work, and conducted data analysis.
- Anil Kumar and A. S. Jeena assisted in data analysis and contributed to manuscript preparation.

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