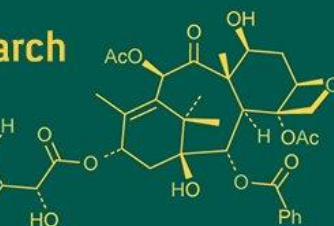
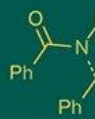


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Genetic architecture and inheritance dynamics governing Mungbean Yellow Mosaic Virus (MYMV) resistance in mungbean (*Vigna radiata* L. Wilczek)

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Abstract

Mungbean Yellow Mosaic Disease (MYMD), caused by the Mungbean Yellow Mosaic Virus (MYMV), is a major biotic constraint causing yield losses ranging from 10% to 100% in mungbean (*Vigna radiata* L. Wilczek). Understanding the nature and magnitude of gene action governing resistance is vital for formulating effective breeding strategies. The present investigation evaluated six basic generations (P_1 , P_2 , F_1 , F_2 , BC_1 , and BC_2) across five distinct crosses evaluated under a Randomized Block Design (RBD) with three replications during Summer 2026 at the Seed Technology Research Unit, Dr. PDKV, Akola. Epidemiological parameters Disease Incidence (%), Percentage Disease Index (PDI), and Area Under Disease Progression Curve (AUDPC) were recorded across five growth intervals. Generation Mean Analysis using Cavalli's joint scaling test and Hayman's six-parameter model revealed that MYMV resistance is quantitative and polygenically controlled. Non-additive gene actions, particularly dominance (h) and epistasis (I , j , l) predominated over additive gene action (d) for PDI and AUDPC across most crosses. Duplicate type non-additive epistasis was prominently observed. High broad-sense heritability (>87%) and high genetic advance as percent of mean (>60%) were recorded for all resistance parameters. Consequently, immediate selection in early segregating generations will be ineffective, and methods like recurrent selection, biparental mating, or delayed selection (F_4/F_5) are recommended to break unwanted linkages and accumulate favorable resistance alleles.

Keywords: Mungbean, Yellow Mosaic Virus, gene action, resistance, heritability

1. Introduction

Mungbean [*Vigna radiata* (L.) Wilczek, $[2n = 2x = 22]$, belonging to the family Fabaceae (subgenus *Ceratotrophis*), is one of the most critical self-pollinating pulse crops cultivated extensively across South and Southeast Asia. Rich in easily digestible protein (~24%) (Kumar *et al.* 2020) [6], essential vitamins, and minerals, mungbean plays a vital role in dietary security and cropping systems owing to its short duration and atmospheric nitrogen-fixing capabilities. In Maharashtra, mungbean represents the second most important pulse crop after pigeonpea. During the 2024-2025 cropping season, Maharashtra cultivated mungbean across 3.85 lakh hectares, producing 1.82 lakh tonnes with an average productivity of 460 kg/ha, which remains significantly lower than the national average productivity (1, 125 kg/ha across 35.27 lakh hectares) (IPR).

A major constraint limiting mungbean productivity is the susceptibility to biotic stresses, chief among which is Mungbean Yellow Mosaic Disease (MYMD). MYMD is caused by the Mungbean Yellow Mosaic Virus (MYMV), a bipartite *begomovirus* in the family *Geminiviridae*, transmitted persistently by the whitefly vector *Bemisia tabaci* Genn. Infection leads to yellowing of leaves, stunting, severe pod distortion, reduced seed size, and yield losses ranging from 10% to complete crop failure (100%), depending on crop growth stage at infection and vector pressure (Khattak *et al.*, 2000; Nay *et al.*, 2008) [5, 8].

Chemical control of whiteflies is economically unviable for smallholders and ecologically hazardous. Consequently, breeding genetically resistant cultivars offers the most sustainable and cost-effective disease management strategy.

However, resistance breeding requires a thorough understanding of the genetic architecture and gene effects (*m, d, h, i, j, l*) governing viral resistance parameters. While classical studies often treated resistance as a simple monogenic trait, recent epidemiological evaluations suggest complex polygenic inheritance involving non-allelic interactions (Kumar *et al.* (2020) [6]. Generation Mean Analysis (GMA), developed by Cavalli (1952) [3] and Hayman (1958) [4], is a powerful biometrical tool designed to resolve genetic variance into additive, dominance, and epistatic (additive × additive [*i*], additive × dominance [*j*], and dominance × dominance [*l*]) components. Alam *et al.* (2014) [1] reported significant additive and dominance effects along with epistatic interactions for MYMV resistance and associated traits, demonstrating the contribution of both additive and non-additive gene effects. Therefore, the primary objective of this investigation was to determine the precise mode of gene action and inheritance governing MYMV resistance parameters—specifically Disease Incidence (%), Percentage Disease Index (PDI), and Area Under Disease Progress Curve (AUDPC)—across five distinct cross combinations in

mungbean.

2. Materials and Methods

2.1 Experimental Site and Agro-Climatic Conditions

The field trial was carried out at the experimental farm of the Seed Technology Research Unit (STRU), Dr. Panjabrao Deshmukh Krishi Vidyapeeth (PDKV), Akola, Maharashtra, India. The site represents the semi-arid tropical region of Central India. Hybridisation to generate *F*₁, backcross (*BC*₁, *BC*₂), and selfed (*F*₂) generations was completed during Kharif 2025. The evaluation of all developed generations along with parents and standard checks was conducted during the Summer 2026 season

2.2 Genetic Materials

The experimental material comprised six basic generations (*P*₁, *P*₂, *F*₁, *F*₂, *BC*₁, *BC*₂) derived from five crosses involving seven parents (3 females, 4 males) with contrasting reactions to MYMV, alongside three commercial checks and their Salient Features of Parental Genotypes and Standard Checks

Table 1: Role Genotype Phenotypic Character MYMV Reaction Group

Role	Genotype	Phenotypic character	MYMV Reaction Group
Female Parent (<i>P</i> ₁)	Kopergaon	Bold seeded, early maturing, wider adaptability	Highly Susceptible (HS)
Female Parent (<i>P</i> ₁)	TAKM 140	High yielding	Resistant (R)
Female Parent (<i>P</i> ₁)	SML 668	High yielding, bold seeded	Resistant (R)
Male Parent (<i>P</i> ₂)	PUSA 9531	High yielding	Highly Resistant (HR)
Male Parent (<i>P</i> ₂)	PDM 139	High yielding, large shiny seeds	Resistant (R)
Male Parent (<i>P</i> ₂)	PANT M-5	Adapted local cultivar	Resistant (R)
Male Parent (<i>P</i> ₂)	BM 2003-2	Bold seeded	Moderately Resistant (MR)

Table 2: Details of Six Generations across Five Crosses.

Generation	Cross I	Cross II	Cross III	Cross IV	Cross V
<i>P</i> ₁	Kopergaon	Kopergaon	TAKM 140	TAKM 140	SML 668
<i>P</i> ₂	PUSA 9531	PDM 139	PANT M 5	PDM 139	BM 2003-2
<i>F</i> ₁	(Kopergaon x PUSA 9531)	(Kopergaon x PDM 139)	(TAKM 140 x PANT M 5)	(TAKM 140 x PDM 139)	(SML 668 x BM 2003-2)
<i>F</i> ₂	Self on <i>F</i> ₁	Self on <i>F</i> ₁	Self on <i>F</i> ₁	Self on <i>F</i> ₁	Self on <i>F</i> ₁
<i>BC</i> ₁	{(H1) x Kopergaon} X kopergaon	[(H2) x Kopergaon] X Kopergaon	[(H3) x TAKM 140] XTAKM-140	[(H4) x TAKM 140] XTAKM-140	[(H5) x SML 668] XSML-668
<i>BC</i> ₂	[(H1)X PUSA 9531]x PUSA 9531	[(H2)x PDM 139]] x PDM 139	[(H3) x PANT M 5]] x PANT M 5	[(H4) x PDM 139]] x PDM 139	[(H5) x BM 2003-2]] x BM 2003-2

2.3 Experimental Design and Layout

The 30 treatments (comprising parents, *F*₁s, *F*₂s, *BC*₁s, *BC*₂s, and 3 standard checks) were laid out in a Randomized Block Design (RBD) with three replications. Planting was performed by hand dibbling with a row-to-row spacing of 45 cm and plant-to-plant spacing of 15 cm. Fertilizer doses and cultural practices were maintained per state recommendations. Susceptible spreader rows (Kopergaon and SML 134) were planted around the experimental block to ensure uniform natural whitefly vector pressure and severe viral exposure.

2.4 Pathological Observations and Disease Scoring

MYMV disease screening was performed visually based on the 0-9 rating scale standardized by Mayee and Datar (1986) [7]

Pathological data were recorded across five crop growth stages: 31, 40, 50, 61, and 73 Days After Sowing (DAS).

Three main epidemiological parameters were computed:

1. Disease Incidence (DI %)

$$\text{Disease Incidence (\%)} = \left(\frac{\text{Number of infected plants per plot}}{\text{Total number of plants evaluated per plot}} \right) \times 100$$

2. Percentage Disease Index (PDI %)

$$\text{PDI (\%)} = \left[\frac{\sum (\text{Scale Score} \times \text{No. of plants in that scale})}{\text{Total plants evaluated} \times \text{Maximum scale score (9)}} \right] \times 100$$

3. Area under Disease Progress Curve (AUDPC)

Calculated across the five time intervals using the formula (Shaner and Finney, 1977) [9]:

$$\text{AUDPC} = \sum_{i=1}^{n-1} \left[\frac{Y_i + Y_{i+1}}{2} \right] \times (t_{i+1} - t_i)$$

2.5 Statistical and Biometrical Analysis

Analysis of Variance (ANOVA) was executed across replications and treatments. Cavalli's (1952) ^[3] Joint Scaling Test was applied using a three-parameter model (m, d, h) evaluated via weighted least squares. Goodness-of-fit was tested using chi-square (χ^2) test with 3 degrees of freedom. Where χ^2 was statistically significant, Hayman's (1958) ^[4] six-parameter model was evaluated to resolve full additive, dominance, and non-allelic epistatic parameters: m = Mean of all generations, d = Additive gene effect, h = Dominance gene effect, i = Additive $\times$ Additive epistatic interaction effect, j = Additive $\times$ Dominance epistatic interaction effect, l = Dominance $\times$ Dominance epistatic

interaction effect.

3. Results and Discussion

3.1 Analysis of Variance (ANOVA)

The ANOVA for MYMV resistance traits demonstrated highly significant differences ($p < 0.01$) among treatments across all parameters. Mean Sum of Squares (MSS) for treatments was 362.20 for Disease Incidence, 333.32 for PDI, and 1, 448, 632.98 for AUDPC values, against low error variances (9.66, 21.15, and 56.69 respectively). This confirms the existence of high genetic variability across the basic generations and crosses under evaluation.

Table 3: ANOVA (Mean Sum of Squares) for MYMV Resistance Traits

Source of Variation	Degrees of Freedom (df)	Disease Incidence (%)	Percentage Disease Index (PDI)	AUDPC Values
Replications	2	6.85	4.11	81.22
Treatments	29	362.20**	333.32**	1, 448, 632.98**
Error	58	9.66	21.15	56.69

* Significant at $p < 0.05$; **Significant at $p < 0.01$.

3.2 Mean Performance of Generations

Phenotypic disease severity varied markedly depending on cross combination and parental susceptibility:

Disease Incidence (DI %) In susceptible female crosses (Crosses I & II), P_1 (Kopergaon) reached 50.0% incidence. In contrast, resistant parent P_2 (PUSA 9531) showed only 21.11% incidence. Resistant $\times$ Resistant crosses (Cross III & IV) maintained lower incidences (31.66% to 38.20%). Cross V (SML 668 $\times$ BM 2003-2) showed lowest disease incidence, with P_1 registering 10.61%. Percentage Disease Index (PDI) Susceptible parent Kopergaon displayed PDI

values of 46.03% and 48.03% in Crosses I and II. Resistant males like PUSA 9531 registered 15.62%. F_1 hybrids displayed intermediate-to-high PDI values (35.73% in Cross I; 39.15% in Cross II), indicating partial dominance of susceptibility or complex interaction effects. AUDPC values ranged from 150.33 (P_1 of Cross V) to 2361.33 (P_1 of Crosses I & II). The resistant check PUSA 9531 maintained a low AUDPC of 288.33, whereas susceptible checks SML-134 and Kopergaon recorded AUDPC values of 1483.00 and 2255.32, respectively.

Table 4: Mean Performance of Generations for MYMV Epidemiological Traits

Cross/Genotype	Generation	Disease Incidence (%)	Percentage Disease Index (PDI)	AUDPC Values
Cross I (Kopergaon $\times$ PUSA 9531)	P_1	50	46.03	2361.3
	P_2	21.11	15.62	241.3
	F_1	35.73	36.45	564.7
	F_2	38.38	38	802.8
	BC_1	43	45.31	609.64
	BC_2	41.4	40.61	356.66
Cross II (Kopergaon $\times$ PDM 139)	P_1	50	48.03	2361.3
	P_2	20.22	32.71	516.3
	F_1	41.33	39.15	559.4
	F_2	45.44	44.03	1403
	BC_1	49	47.61	783.6
	BC_2	45	41.81	592.2
Cross III (TAKM 140 $\times$ PANT M-5)	P_1	38.24	40.48	451.6
	P_2	18.19	4.25	50.67
	F_1	16.24	6.26	61.8
	F_2	31.33	27.64	439.26
	BC_1	36.83	39.71	449.9
	BC_2	33.53	35.59	478.26
Cross IV (TAKM 140 $\times$ PDM 139)	P_1	38.24	40.83	851.6
	P_2	20.22	23.82	271.6
	F_1	16.62	21.23	371.3
	F_2	39.23	38.13	550.66
	BC_1	33.33	35.49	477.3
	BC_2	19.29	35.8	335.4
Cross V (SML 668 $\times$ BM 2003-2)	P_1	10.61	11.41	150.33
	P_2	50	49.32	1026.3
	F_1	33.73	33.34	418.2
	F_2	19.62	21.52	288.8
	BC_1	18.52	24.66	309.33
	BC_2	33.93	31.25	523.6

Checks	SML-134 (S Check)	38	3.61	483.4
	Kopergaon (S Check)	39.54	3.72	2553.2
	PUSA 9531 (R Check)	11.33	16.62	288.33
Statistical Parameters	SE (m)±	1.72	2.65	51.24
	CD at 5%	5.08	7.52	6.2
	CD at 1%	6.78	10.03	46.2
	CV (%)	9.91	3.8	9.02

3.3 Estimates of Gene Effects and Epistatic Interaction Parameters

Joint scaling tests produced highly significant χ^2 values across all crosses for DI, PDI, and AUDPC, proving that the

additive-dominance model is inadequate and non-allelic epistatic interactions play a significant role.

Table 5: Estimates of Gene Effects (m, d, h, i, j, l) for MYMV Resistance Traits

1. For disease incidence

Trait	Cross I	Cross II	Cross III	Cross IV	Cross V
m	38.267**	35.400**	33.600**	36.200**	34.333**
d	13.167**	11.500**	21.700**	7.367**	'-2.567**
h	3.467	11.200*	11.000*	'-6.020*	1.620*
i	14.734**	1.400**	14.200**	'-6.734**	4.466
j	'-0.667	'-1.884*	'-1.834*	'-4.884*	0.666
l	'-9.080	'-1.250	'-5.600	'-23.410	1.54
$\chi^2(df = 3)$	15.567**	12.450**	14.280**	7.875**	11.827**

2. Percentage Disease Index (PDI)

Trait	Cross I	Cross II	Cross III	Cross IV	Cross V
m	47.933**	49.067**	44.467**	48.233**	38.267**
d	0.700**	1.133	0.200**	0.35	'-1.167**
h	19.850**	12.600*	19.267**	31.200*	13.467**
i	4.867	11.333	20.533**	'-7.967	14.734**
j	'-1.147**	'-3.717	'-0.667**	'-1.650	0.667**
l	'-33.520*	'-22.800*	'-45.733**	'-35.600*	'-19.080*
$\chi^2(df = 3)$	14.820**	17.040**	18.876**	36.931**	15.560**

3. AUDPC Values

Trait	Cross I	Cross II	Cross III	Cross IV	Cross V
m	1297.033**	1358.090**	1217.277**	1350.610**	1273.867**
d	27.447**	'-9.147	22.730**	'-1.287	94.407**
h	'-63.493	'-500.000**	'-534.072**	415.200**	253.084*
i	'-38.773	'-289.520	541.367**	'-25.907**	9.547
j	'-44.530	'-110.190**	25.398**	'-25.218**	161.733**
l	'-74.853	700.000*	1049.903**	'-538.038**	'-455.090**
$\chi^2(df = 3)$	2.688**	11.342*	34.593**	13.105**	33.958**

3.4 Heritability and Genetic Advance

Broad-sense heritability (h_{bs}^2), Genetic Advance (GA), and

Genetic Advance as Percent of Mean (GAM) for all MYMV parameters across all five crosses are summarized below:

Table 6: Heritability (h_{bs}^2 %), Genetic Advance, and GAM (%) across Crosses

Character Metric	Parameter	Cross I	Cross II	Cross III	Cross IV	Cross V
h_{bs}^2 (%)	Disease Incidence	91.08	88.9	87.54	90.03	88.24
	PDI	91.7	93.5	90.3	91.2	92.4
	AUDPC	90.07	91.02	92.97	93.07	95.6
GA	Disease Incidence	21.05	23.4	20.85	25.12	22.68
	PDI	24.73	25.51	22.5	23.7	24.51
	AUDPC	750.61	1664.67	1619.61	1513.15	1858.59
GAM (%)	Disease Incidence	82.5	85.2	81.1	88.4	83.6
	PDI	71.02	60.52	75.57	76.81	95.1
	AUDPC	143.76	110.25	182.64	173.43	215.27

High broad-sense heritability (>87%) paired with extraordinarily high GAM (>60% for PDI and >110% for AUDPC) confirms strong genetic influence over environmental variance. However, because dominance (h)

and epistasis (i, j, l) contribute substantially to broad-sense heritability, high h_{bs}^2 alone does not guarantee rapid selection progress in F_2 .

4. Breeding Implications & Strategies

The synthesis of generation mean parameters, epistasis models, and heritability estimates provides a practical

blueprint for breeding MYMV-resistant mungbean genotypes:

Table 7: Overall View of MYMV Resistance Traits, Gene Actions, and Breeding Strategies

Trait	Major Gene Effect (s)	Inheritance Type	Breeding Implication & Recommended Strategy	References
Disease Incidence (%)	d, i	Additive + Epistasis	Selection in segregating populations (F_2/F_3) can fix additive resistance alleles.	Alam <i>et al.</i> (2014) ^[1] ; Akbhar <i>et al.</i> (2018) ^[2]
Percentage Disease Index (PDI)	h, j, l	Dominance + Epistasis	Duplicate epistasis masks true genetic worth. Delay selection to advanced generations (F_4-F_6).	Alam <i>et al.</i> (2014) ^[1] ; Akbhar <i>et al.</i> (2018) ^[2]
AUDPC Values	h, j, l	Dominance + Epistasis	Non-additive gene action dominates. Employ recurrent selection, biparental mating, or diallel selective mating to accumulate favorable polygenes.	Alam <i>et al.</i> (2014); Akbhar <i>et al.</i> (2018) ^[2]

Discussion of Gene Actions

- Disease Incidence (DI):** Significant positive additive (d) and additive $\times$ additive (i) effects were found in Cross I ($d = 13.16^{**}$, $i = 14.73^{**}$) and Cross III ($d = 21.70^{**}$, $i = 14.2^{**}$). Because additive components are fixable, selection for reduced disease incidence can be initiated in early-to-intermediate segregating generations in these crosses.
- PDI and AUDPC Severity Parameters:** PDI exhibited significant positive dominance effects (h) across all five crosses (ranging from 12.600* to 31.200*). Furthermore, dominance $\times$ dominance (l) effects were uniformly significant and negative (e.g., $l = -45.73^{**}$ in Cross III). Because [h] and [l] carry opposite algebraic signs, resistance reaction to PDI and AUDPC is predominantly governed by duplicate epistasis. Duplicate interactions cancel out additive genetic gains in early generations and mask true line performance.

5. Conclusion

- Polygenic Genetic Architecture:** Inheritance of MYMV resistance in mungbean is quantitative and polygenically regulated.
- Predominance of Non-Additive Epistasis:** While Disease Incidence displays significant fixable additive effects (d, i), disease severity parameters (PDI and AUDPC) are governed by dominance (h) and non-allelic epistatic interactions (j, l), with duplicate epistasis prevailing across major crosses.
- Breeding Recommendations:** Traditional single-seed descent or immediate F_2 pedigree selection will be inefficient for viral severity traits due to masking epistatic effects. Mungbean improvement programs should adopt delayed selection ($F_4 - F_6$), inter-mating among desirable segregants (biparental crossing), or recurrent selection to assemble resistant polygenic complexes.

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