

International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; SP-10(5): 1068-1071
www.biochemjournal.com
Received: 01-03-2026
Accepted: 05-04-2026

Nithinkumar KR
College of Horticulture,
Bengaluru, UHS, Bagalkot,
Karnataka, India

Dhananjaya MV
Division of Vegetable Crops,
ICAR-IIHR, Bengaluru,
Karnataka, India

Anjanappa M
College of Horticulture, Kolar,
UHS, Bagalkot, Karnataka,
India

Varalakshmi B
Division of Vegetable Crops,
ICAR-IIHR, Bengaluru,
Karnataka, India

Sri Ram
Division of Crop Protection,
ICAR-IIHR, Bengaluru,
Karnataka, India

Ravishankar KV
Division of Biotechnology,
ICAR-IIHR, Bengaluru,
Karnataka, India

Aravinda Kumar JS
College of Horticulture, UHS,
Bagalkot, Karnataka, India

Venugopalan R
Division of Basic Science,
ICAR-IIHR, Bengaluru,
Karnataka, India

Corresponding Author:
Nithinkumar KR
College of Horticulture,
Bengaluru, UHS, Bagalkot,
Karnataka, India

Inheritance of gummy stem blight resistance in bottle gourd

Nithinkumar KR, Dhananjaya MV, Anjanappa M, Varalakshmi B, Sri Ram, Ravishankar KV, Aravinda Kumar JS and Venugopalan R

DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i5SI.9314>

Abstract

The study was to investigate the inheritance pattern of resistance to gummy stem blight disease in bottle gourd. The genetics of resistance to gummy stem blight was examined in two segregating populations derived from crosses between the bottle gourd genotypes IIHR-77-6-1 × IIHR-64 and IIHR-95 × IIHR-18. The six generations P₁, P₂, F₁, F₂, BC₁ and BC₂ of two bottle gourd crosses were evaluated in a gummy stem blight-infested plot. Disease incidence and severity were recorded for each individual across all six generations and replications from the day after inoculation until flowering. Initial disease incidence was noted when the first symptoms appeared, with subsequent observations made daily. In six parameter model, all main and interacting crosses showed the significant except additive × additive and additive × dominance. Different signs of dominance ($\hat{h}$) and additive × dominance ($\hat{I}$) components revealed that GSB resistance governed by duplicate epistasis decreasing effect genes between dominance. The experiment suggests that the IIHR-77-6-1 and IIHR-95 genotypes can serve as sources of gummy stem blight resistance for future crop improvement programs in bottle gourd.

Keywords: GSB, genes, inheritance

Introduction

Gummy stem blight caused by *Didymella bryoniae* has emerged as a major limiting factor of bottle gourd cultivation in India because, the disease severity varies from season to season and limited success has been achieved in controlling GSB with chemical methods. Therefore, the best method for overcoming GSB in bottle gourd is to develop more resistant cultivars by molecular breeding. To date, there are no reports on molecular markers and mapping for use in breeding GSB resistance in bottle gourd. However, some works have been reported in melons and cucumber.

GSB resistance reactions and mechanisms of resistance are truly based on location and environmental specific. Many scientists have reported that different types of gene action have been attributed to GSB resistance in cucurbits, including single dominance gene (Joseph *et al.*, 2007) [6], single recessive gene (Hassan *et al.*, 2018) [4] and one study indicated that resistance to GSB was quantitative and mainly governed by three pairs of additive epistatic major genes (Zhang *et al.*, 2017) [11]. A study indicated that the resistance to GSB in cucumber seedlings was quantitative and mainly governed by two pairs of major QTLs and multiple minor QTLs (Liu *et al.* 2017) [8] and one report suggested that resistance to GSB may be under the control of a more complex genetic system (Gusmini *et al.*, 2017) [1]. Nevertheless, the inheritance of GSB resistance in bottle gourd is yet unclear. Hence, an attempt is made to study and to generate information on genetics of gummy stem blight disease resistance of bottle gourd.

Materials and Methods

The cross combination of resistant × susceptible *i.e.*, IIHR-77-6-1 × IIHR-64 and IIHR-95 × IIHR-18 were taken to study the generation mean analysis for gummy stem blight resistance. In season II, the susceptible and resistant parents were crossed to produce F₁'s in April-September, 2020. In season III, The F₁'s was selfed to get F₂ and B₁ & B₂ combinations were generated by back crossing of F₁ to their respective parents in November

to March, 2020-21. In Season IV, the six generations of two crosses were evaluated in the field during May-September, 2021.

The materials used in this experiment to study the genetics of GSB resistance are six generations viz., P_1 , P_2 , F_1 , F_2 , B_1 and B_2 populations were raised in the field during May-September, 2021, to evaluate fruit yield and inheritance of GSB disease.

Data were recorded from 10 plants of parents (P_1 and P_2) and F_1 each, 40 plants of each B_1 and B_2 each and 208 F_2 plants of cross IIHR-77-6-1 $\times$ IIHR-64 and 249 F_2 plants of cross IIHR-95 $\times$ IIHR-18 for GSB incidence. Susceptible line IIHR-24 and Pusa Naveen was raised along the border as well as after every five rows to maintain uniform spread of disease.

Populations were screened under natural epiphytotic condition and disease incidence was recorded based on evaluation of the intensity of the disease symptoms on leaves and stem of the individual plant. The disease scoring was started a month after planting at weekly interval for 8 weeks until plant growth ended. Ratings were done on 0 to 9 scales as described by Gusmini *et al.*, 2002 [3]. The individual plant ratings thus recorded were used to calculate the mean percentage disease index (PDI), Area under disease progression curve (AUDPC).

Statistical analysis for the recorded data was performed using WINDOSTAT Version 9.3 package (Indostat services, Hyderabad).

Results and Discussion

For this study, IIHR-77-6-1 $\times$ IIHR-64 and IIHR-95 $\times$ IIHR-18 crosses were selected. Further, in each cross six generations, viz., P_1 , P_2 , F_1 , F_2 , B_1 and B_2 were produced. Both the segregating (F_2 , B_1 and B_2) and non-segregating (P_1 , P_2 and F_1) generations were screened for gummy stem blight disease reaction under field condition. The mean PDI data were subjected to Mather scale, joint scaling test and six generation mean analysis in order to study inheritance pattern of gummy stem blight resistant genes.

The means for six generations in two crosses (IIHR-77-6-1 $\times$ IIHR-64 and IIHR-95 $\times$ IIHR-18) are given, primarily to compare these population's means with one another (Table 1).

The results of mean PDI indicating that all the generations were recorded mean PDI within the parental range in both the crosses IIHR-77-6-1 $\times$ IIHR-64 and IIHR-95 $\times$ IIHR-18 population (Fig. 1). The mean PDI data obtained from six generations studied were subjected to generation mean analysis to obtain information on additive, dominance and duplicate epistatic interactions (Table 2).

The results of the scaling tests and generation mean analysis for the both combinations are presented in Table 2. The scaling tests conducted on each cross were found to be significant (Hayman, 1958) [5]. The significant values of scaling tests indicated the inadequacy of simple additive-dominance model and the presence of epistasis. These crosses are called interacting crosses.

Cross-I. (IIHR-77-6-1 $\times$ IIHR-64)

Based on mean PDI P_2 , F_2 and B_2 recorded susceptibility to GSB disease. B_1 and F_1 showed moderately resistant reaction and resistant reaction were observed in P_1 . The maximum mean PDI was recorded in P_2 (77.77) followed by F_2 (51.81) and B_2 (46.94). The P_1 recorded least mean PDI

(23.33) among the generations and P_2 recorded high variances of 1700.95. In the cross IIHR-77-6-1 $\times$ IIHR-64, scaling tests (A, C and D) were significant, indicating that inadequacy of simple additive-dominance model and presence of epistasis.

All the main gene components contributed towards susceptibility rather resistance with high dominance effect (-71.15). In interacting crosses, additive $\times$ dominance (57.77) was significant and possessed a positive value suggesting their contribution towards resistance rather than susceptibility to disease. Additive $\times$ additive (36.14) and dominance $\times$ dominance (37.26) components were showed non-significant. Among all the gene effects, additive $\times$ dominance component was of much higher in magnitude for resistance due to its negative value. Similar results were also reported in by Woluka *et al.* (2007) [10]. Opposite signs of $\hat{h}$ and $\hat{l}$ components revealed duplicate epistasis and number of effective gene controlling resistance was 2.1 (Castle Wright's estimation).

Cross II (IIHR-95 $\times$ IIHR-18)

Based on mean PDI P_2 recorded highly susceptible to GSB disease, whereas F_2 and B_2 showed susceptible reaction, F_1 and B_1 exhibited moderate resistance and P_1 exhibits resistance reaction. The maximum mean PDI was recorded in P_2 (90.00) followed by F_2 (50.02) and B_2 (Fig. 2). The P_1 recorded least mean PDI (18.88) among the generations. Highest variance was observed in F_2 segregating population (382.44). In the cross IIHR-95 $\times$ IIHR-18, all scaling tests (A, B, C and D) were significant, indicating that inadequacy of simple additive-dominance model and presence of epistasis. Similar results were observed by Gusmini *et al.*, 2005 [2].

Among the main gene components all are towards susceptibility rather resistance with high dominance ($\hat{h}$) (-52.86) effect. In interacting crosses, dominance $\times$ dominance (25.64) component possessed a negative value suggesting their contribution towards resistance rather than susceptibility to disease and additive $\times$ additive ($\hat{j}$) (23.05) component contributed towards susceptibility. Among all the gene effects, dominance $\times$ dominance ($\hat{l}$) component was of much higher in magnitude for resistance due to its negative value. Opposite signs of ($\hat{h}$) and ($\hat{l}$) components revealed duplicate epistasis and number of effective gene controlling resistance was 2.4 (Castle Wright's estimation). In this study, the crosses IIHR-77-6-1 $\times$ IIHR-64 and IIHR-95 $\times$ IIHR-18 were made to produce six generations: P_1 , P_2 , F_1 , F_2 , B_1 and B_2 . Both segregating (F_2 , B_1 and B_2) and non-segregating (P_1 , P_2 and F_1) generations were assessed for disease resistance, and data on percent disease incidence (PDI) along with various other traits were collected. The evidence suggests that resistance or susceptibility is controlled by nuclear genes, with no cytoplasmic influence. While it is challenging to completely rule out the presence of multiple allelism (Kempthorne, 1957), it has not been deemed significantly impactful. Since F_1 generations were included in this study, the estimates derived from non-segregating progenies are unlikely to be affected by linkage. In the IIHR-77-6-1 $\times$ IIHR-64 and IIHR-95 $\times$ IIHR-18 crosses, the mean values of F_1 and F_2 exhibited significant fluctuations compared to each other, with their means F_2 exhibited Significant fluctuations compared to each other, with their means skewing towards the susceptible parent, indicating dominance for susceptibility in these crosses. The

significance of scaling tests for both crosses suggested that resistance was not governed solely by simple additive or dominant gene effects. Instead, gene interactions appeared to play a role in its inheritance (Malek and Shamsuddin, 1998) [9]. This indicates that the phenotypic expression of genes at one locus may be influenced by genes at other loci, a phenomenon known as non-allelic interaction.

In six parameter model, all main and interacting crosses

showed the significant except additive $\times$ additive and additive $\times$ dominance. Different signs of dominance ($\hat{h}$) and additive $\times$ dominance ($\hat{i}$) components revealed that GSB resistance governed by duplicate epistasis decreasing effect genes between dominance. The study shows that the IIHR-77-6-1 and IIHR-95 genotypes can serve as sources of gummy stem blight resistance for future crop improvement programs in bottle gourd.

Table 1: Mean and variances of six generation for percent disease index (PDI) of GSB in bottle gourd

Crosses		P1	P2	F1	F2	B1	B2
IIHR-77-6-1 X IIHR-64	Mean $\pm$ SE	23.33 $\pm$ 1.99	77.77 $\pm$ 1.48	25.55 $\pm$ 1.69	51.81 $\pm$ 1.48	38.61 $\pm$ 1.25	46.94 $\pm$ 1.40
	Variance	39.78	1700.95	28.80	458.30	63.23	79.06
IIHR-95 X IIHR-18	Mean $\pm$ SE	18.88 $\pm$ 1.69	90.00 $\pm$ 2.59	30.00 $\pm$ 1.69	50.02 $\pm$ 1.23	36.66 $\pm$ 1.13	49.16 $\pm$ 1.25
	Variance	28.80	67.21	28.80	382.44	51.91	62.59

Table 2: Gene action of two crosses for GSB disease intensity in bottle gourd

Crosses	A	B	C	D	$\hat{m}$	$\hat{d}$	$\hat{h}$	$\hat{i}$	$\hat{j}$	$\hat{i}$	Type of epistasis
IIHR-77-6-1 X IIHR-64	-28.33 $\pm$ 3.63**	29.44 $\pm$ 3.60	-35.04 $\pm$ 7.27**	18.07 $\pm$ 3.51**	51.81 $\pm$ 1.48**	-8.33 $\pm$ 1.88**	-71.15 $\pm$ 7.34**	-36.14 $\pm$ 7.03**	57.77 $\pm$ 4.51**	37.26 $\pm$ 0.48**	D
IIHR-95 X IIHR-18	24.44 $\pm$ 3.30**	-21.66 $\pm$ 3.98**	31.20 $\pm$ 6.76**	14.21 $\pm$ 3.00**	50.02 $\pm$ 1.23**	-12.5 $\pm$ 1.69**	-52.86 $\pm$ 6.42**	-28.42 $\pm$ 6.00**	23.05 $\pm$ 2.29**	25.64 $\pm$ 9.56**	D

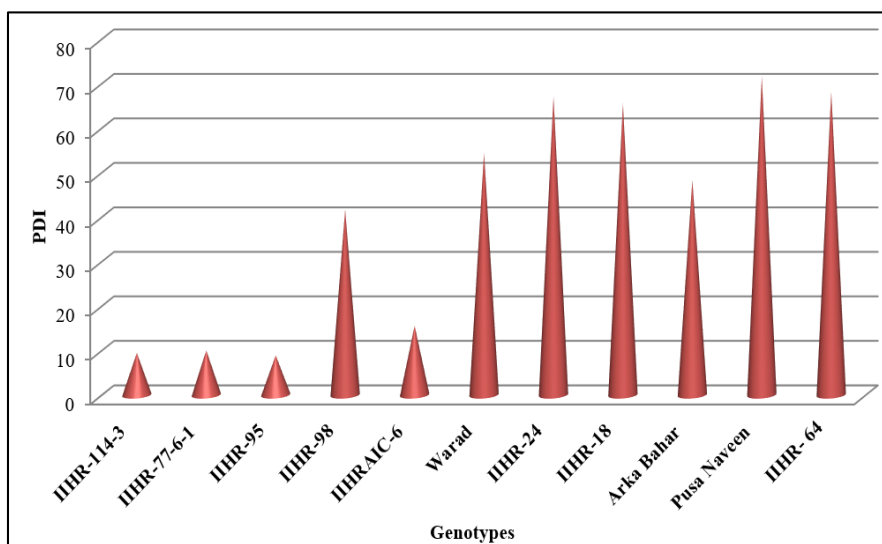


Fig 1: Graphical representation of resistance reaction of bottle gourd genotypes against gummy stem blight

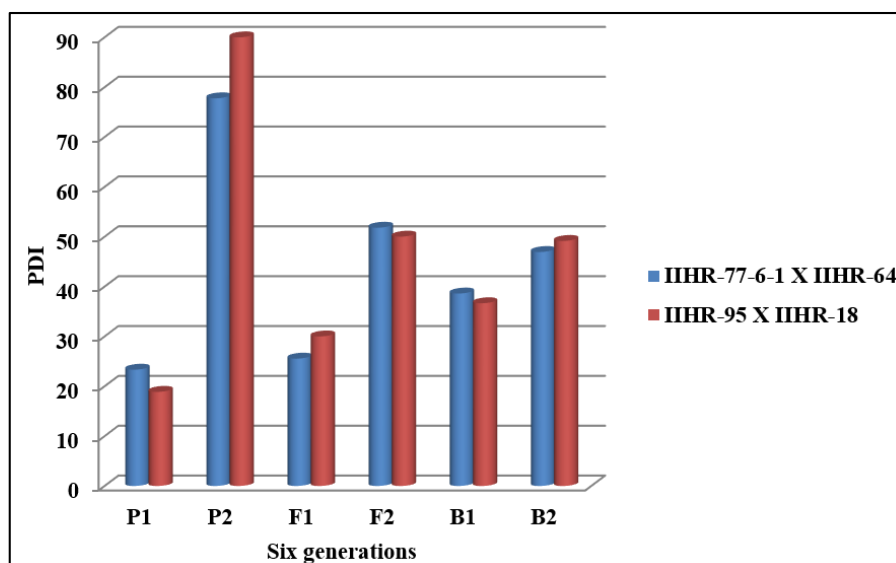


Fig 2: Graphical representation of PDI of six generation against gummy stem blight

Acknowledgement

This research was a part of doctoral programme of first author and financial support from ICAR Indian Institute of Horticulture Research (IIHR).

References

1. Gusmini G, Luis A, Burgos R, Wehner TC. Inheritance of resistance to gummy stem blight in watermelon. *HortScience*. 2017;52(11):1477-1482.
2. Gusmini G, Song R, Wehner TC. New sources of resistance to gummy stem blight in watermelon. *Crop Science*. 2005;45:582-588.
3. Gusmini G, Wehner TC, Holmes GJ. Disease assessment scales for seedling screening and detached leaf assay for gummy stem blight in watermelon. *Cucurbit Genetics Cooperative Report*. 2002;25:36-40.
4. Hassan MZ, Rahim MA, Natarajan S, Robin AHK, Park J, Nou S. Gummy stem blight resistance in melon: inheritance pattern and development of molecular markers. *International Journal of Molecular Sciences*. 2018;19:2914.
5. Hayman BI. The separation of epistatic from additive and dominance variation in generation mean. *Heredity*. 1958;12(3):371-390.
6. Joseph N, Zhou WXH, Li, Zhang YB, Chen JF. Resistance to gummy stem blight in melon (*Cucumis melo* L.) germplasm and inheritance of resistance from plant introductions 157076, 420145, and 323498. *HortScience*. 2007;42:215-221.
7. Kempthorne O. *An Introduction to Genetical Statistics*. New York: John Wiley and Sons Inc.; 1957. p. 88-94.
8. Liu S, Shi Y, Miao H, Wang M, Li B, Gu X, *et al.* Genetic analysis and QTL mapping of resistance to gummy stem blight in *Cucumis sativus* seedling stage. *Plant Disease*. 2017;101:1145-1152.
9. Malek MA, Shamsuddin AKM. Combining ability and heterosis for yield and yield contributing characters in upland cotton (*Gossypium hirsutum* L.).
10. Wolukau JN. Genetic, phylogenetic and molecular studies of melon (*Cucumis melo* L.) resistance to gummy stem blight (*Didymella bryoniae*). [PhD Thesis]. Nanjing: Nanjing Agricultural University; 2007.
11. Zhang S, Liu S, Miao H, Shi Y, Wang M, Wang Y, *et al.* Inheritance and QTL mapping of resistance to gummy stem blight in cucumber stem. *Molecular Breeding*. 2017;37:49.