



# Screening of bitter gourd germplasm and advanced breeding lines against powdery mildew

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## Abstract

Powdery mildew caused by *Podosphaera xanthii* is a devastating fungal disease in bitter gourd growing areas of India inflicting more than 70% losses. Resistance sources were identified among 31 genotypes of *Momordica charantia*, *M. charantia* var. *muricata* and *M. balsamina* based on screening under natural field and artificial epiphytotic condition at seedling stage. The genotypes varied with respect to percent disease index (PDI), area under disease progressive curve (AUDPC) and the apparent rate of disease spread in unit time period ( $r$ ). The mean PDI under natural and artificial screening ranged from 0.0 to 100.0 and 0.0 to 94.4, respectively. The genotypes, IIHR-80-1-2 and IIHR-80-1-3 (*M. balsamina*) were free from disease, whereas, IIHR-144-1 (*M. charantia* var. *muricata*) showing mild symptoms with PDI of 1.8 and 4.3 under natural and artificial screening, respectively at later stage were also resistant. Genotypes IIHR-40-1 and IIHR-49-34-5 were moderately susceptible (PDI < 40) PDI was highly correlated with AUDPC under both natural and artificial screening ( $r=0.99, 0.95$ ;  $P < 0.01$ ). Also PDI under natural field screening was highly correlated with the artificial screening under controlled conditions. The highest apparent infection rate ( $r$ ) was recorded in the genotype, Arka Harit (1.29), whereas, it was the least in IIHR-80-1-2 and IIHR-80-1-3 (0.00). Statistical analysis of disease severity over time using non linear growth model revealed that 97% of variability in disease progression over time was captured by nonlinear logistic model. The rate of disease severity was maximum at  $t=2-3$  weeks in susceptible and  $t=4.24$  in moderately susceptible genotype IIHR-40-1, inferring that management strategies may be undertaken within the period identified to avoid loss. The newly identified resistant sources may be used for developing powdery mildew resistant bitter gourd varieties/hybrids.

**Keywords** Powdery mildew · Bitter gourd · AUDPC · Resistance · SAS programming

## Introduction

Bitter gourd (*Momordica charantia* L.) is one of the most popular vegetables cultivated throughout Asia, especially in India. Each and every part of this plant has nutritive and medicinal significance (Behera 2004; Akram et al. 2010)

and has been used in various herbal medicine systems due to the presence of disease preventing and health promoting phyto-chemical compounds. Bitter gourd fruits are rich in nutrients such as beta-carotene, vitamin C, folic acid, magnesium, phosphorus, and potassium (Dhillon et al. 2016a). Active compounds extracted from bitter gourd seeds inhibited human liver cancer cell (Fang et al. 2012) and breast cancer cell proliferation (Ray et al. 2010).

In India, it is cultivated in an area of 122 thousand hectares with the production of 1204 thousand metric tonnes (Saxena et al. 2016). Among various factors for low productivity of bitter gourd, diseases take a heavy toll of crop. Powdery mildew caused by *Podosphaera xanthii* (Castagne) Braun and Shishkoff) (formerly *Sphaerotheca fuliginea* (Schlecht.) Poll.) is a major production constraint in almost all parts of India and its incidence is observed throughout the year. The losses due to powdery mildew have been reported up to 50% (Dhillon et al. 2016b) and variety Arka Harit

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recorded a damage of 70–80%. The disease can be controlled with fungicides (Urban and Lebeda 2006), however, genetic resistance provides economically sound, practically feasible and environmentally safe approach. Moreover, identification of resistant donors is the prerequisite for any disease resistance breeding program. Hence, the present study was undertaken to identify powdery mildew resistant germplasm under natural field and artificial epiphytotic conditions during *rabi* season.

## Materials and methods

The experiments were conducted at the experimental farm of Division of Vegetable Crops, ICAR-IIHR, Hessaraghatta, Bengaluru, Karnataka, India, during *rabi*-summer season of 2016–2017. The experimental materials comprised 31 germplasm and advanced breeding lines of *Momordica* spp. (maintained as inbreds) including landraces, commercially released varieties and wild types viz., *Momordica charantia* var. *muricata* and *M. balsamina*.

### Screening under natural epiphytotic conditions

The test genotypes were screened under natural epiphytotic conditions during the late kharif season (August–December, 2016), when the climatic conditions were favourable for severe development of the disease. The experiment was conducted in randomized block design with three replications and 10 plants per replication. Seedlings of each genotype were transplanted in raised beds spacing 150 cm apart with plant to plant distance of 50 cm following recommended package of practices (Dinesh 2018). The plants developed natural infection of powdery mildew and five plants were randomly selected in each replication for disease scoring based on per cent leaf area infected and 0–9 rating scale was followed for disease ratings as suggested by Jenkins and Wehner (1983) and percent diseases index (PDI) was calculated (Fanourakis 1990).

### Screening under Artificial epiphytotic conditions

The test genotypes of *Momordica* spp were sown in pro-trays (98 cells) in October 2016. Powdery mildew was mass propagated on the susceptible plants and was used for inoculation. The seedlings (at cotyledonary stage, 9 days after sowing) were dust inoculated on the cotyledons. The spores were transferred from infected leaf to healthy leaves with the help of a thin inoculation brushes described earlier by Neethu and Sriram (2017). For the inoculation of powdery mildew, use of conidial suspension though may give opportunity to know the exact spores per ml of the suspension, did not help in getting good and consistent infection as the pathogen needed dry

and cool condition for infectivity and not moist condition. The dusting of spores was done evenly to cover the surface of each leaflet. Disease was scored on 5th day after inoculation and at 5 days interval thereafter for 35 days.

### Disease assessment

Disease scoring was done based on per cent leaf area infected by, on 0–9 rating scale (Jenkins and Wehner 1983).

Score	% Disease	Description
0	0	No disease
1	0–3	Few small leaf lesions
2	3–6	Few lesions on few leaves with no stem lesions
3	6–12	Few lesions on few leaves or with superficial stem lesions
4	12–25	Few well formed leaf lesions or superficial stem lesions
5	25–50	Few well formed leaf lesions or enlarging stem lesions
6	50–75	Many large leaf lesions or deep stem lesions with abundant sporulation or plant more than 50% defoliated
7	75–87	Many large coalescing leaf or stem lesions, over 75% of plant area affected or defoliated
8	87–100	Plants largely defoliated, leaf or stem with abundant sporulating lesions
9	100	Plants dead

The PDI was calculated using the formula,

$$\text{PDI} = \frac{\text{Sum of numerical values}}{\text{Number of leaves graded} \times \text{Maximum ratings}} \times 100.$$

After calculating PDI, the genotypes in the population was categorized into five categories namely resistant (0–10%), moderately resistant (10–20%), moderately susceptible (20–40%), susceptible (40–60%) and highly susceptible (>60%). The data on percent disease index was analyzed using randomized block design (Gomez and Gomez 1984).

AUDPC was determined using the following formula (Cambell and Madden 1990).

$$\text{Ak} = \sum_{i=1}^{N_i-1} \frac{(y_i + y_{i+1})}{2} (t_{i+1} - t_i).$$

where  $y_i$  is the proportion of disease on the  $i$ th observation,  $t_i$  is the time (days) of observation expressed as days after sowing (DAS) and  $n$  is the total number of disease severity readings (PDI) taken throughout the experimental period.

The apparent rate of disease development ( $r$ ) is a measure of the speed at which an epidemic develops. ‘ $r$ ’ was calculated from the powdery mildew severity recorded at different time intervals (once in a week) of the plant growth using the given formula (Vanderplank 1963):

$$r = 2.3/t_2 - t_1 \{ \log (X_2(1 - X_1)/X_1(1 - X_2)) \},$$

where  $r$  is the apparent infection rate in non-logarithmic phase,  $X_1$  is the disease index at time  $t_1$ ,  $X_2$  is the disease index at subsequent week time  $t_2$ .

### Data analysis

Analysis of variance of the data generated on percent disease index was analyzed using WASP 2.0 (Web Agri Stat Package) software developed by ICAR-Central Coastal Agricultural Research Institute, Goa, India. The data were arcsine transformed to minimize heterogeneity. The mean disease scores of the replicates were used in Pearson’s correlation to compare the qualitative and quantitative indices (Gomez and Gomez 1984).

### Non linear statistical modeling

In order to fit the non-linear growth model for the powdery mildew severity data, Levenberg–Marquardt technique (Ratkowsky 1990) was utilized and programming codes were developed using Statistical Analysis System (SAS) package available at ICAR-IIHR, Bangalore, India (SAS V 9.3). PROC NLIN subroutine was utilized to construct SAS codes (SAS-bitter gourd-PM). Global convergence of the parameter estimates were ensured by trying different sets of initial values.

### Measures of model adequacy

As a measure of goodness of fit, the value of coefficient of determination ( $R^2$ ) (Kvalseth 1985) was calculated as

$$R^2 = 1 - [(\sum Y_t - Y)^2 / (\sum Y_t - \bar{Y})^2]$$

where  $Y_t$  represents the percent disease incidence during the period  $t$ .

### Residual analysis

Residual analysis was also carried out using the one sample run-test, for testing the randomness assumption and the normality assumption of residuals were tested using Shapiro–Wilk test (Siegel and Castellan 1988).

## Results and discussion

It is important that the plant population must be exposed to the pathogen in such a way that resistant and susceptible plants can be distinguished without any ambiguity, which reveals the efficacy of screening. All the susceptible genotypes exhibited typical powdery growth on the leaves which has spread to the entire plant rapidly.

### Screening under natural condition

Field screening revealed that mean PDI values of the 31 genotypes ranged from 0.0 to 100.0 (Table 1). The wild species accessions, IIHR-80-1-2 and IIHR-80-1-3 were free from symptoms whereas, IIHR-144-1 was highly resistant with PDI 1.8. Genotypes, IIHR 40-1 and IIHR 49-34-5 were moderately susceptible with PDI of 31.1 and 34.6 respectively. Rest of the genotypes showed high susceptibility to powdery mildew. Incidentally, commercial varieties of bitter gourd, viz., Arka Harit, Pusa Vishesh, Preethi, Meghana and Hirkani were highly susceptible. As has been observed in the present studies, Dhillon et al. (2014) reported that lines viz., THMC 143, THMC 167, THMC 177 showed resistance to powdery mildew.

### Screening under artificial conditions

Mean percent disease index ranged from 0.0 to 94.4 (Table 1) after 35 days of inoculation. Disease symptoms started to appear as small white powdery growth on the first true leaves of the susceptible genotypes 5 days after inoculation which increased with yellow colouration followed by necrosis which spread to developing leaves. The resistant genotypes viz, IIHR-80-1-2, IIHR-80-1-3 showed no disease development and IIHR-144-1 which showed mild symptoms in later stage of plant growth was highly resistant (PDI 4.3). The genotype, IIHR-40-1 was moderately susceptible with PDI 39.4 and rest of the genotypes were susceptible. Moderate resistance or slow disease development and disease progress is a unique phenomenon observed in various host-powdery mildew pathogen systems (Douglas et al. 1984; Hautea et al. 1987; Raju et al. 1991), and is characterized by delay in the onset and severity of symptom development (Raju and Anilkumar 1990). Such slow mildewing intermediate resistance is often found to be more durable than the complete resistance, as reported in other crops like water melon (Tetteh et al. 2010) and pumpkin (Luitel et al. 2016). Therefore, the slow mildewing genotypes have gained much attention from breeding point of view (Bus et al. 2005; Liang et al. 2006).

**Table 1** Screening of 31 bitter gourd genotypes for resistance against powdery mildew

Sl. no.	Genotype	Natural screening			Yield/plot (kg/7.5 sq m)	Artificial screening			Apparent rate of infection (r)		Disease reaction
		Mean PDI 1 WAI	Mean PDI 10 WAI	Mean PDI* AUDP		Mean PDI 5 DAI	Mean PDI 35 DAI	Mean PDI* AUDP	AUDP		
1	Arka Harit	25.2 (30.1)	99.6 (88.8)	86.7 (68.8)	5629.8	18.5 (25.1)	94.4 (76.2)	63.6 (52.6)	2720.9	1.29	HS
2	IIHR-144-1	0.0 (0.0)	11.1 (19.2)	1.8 (7.7)	88.1	0.0 (0.0)	10.6 (18.1)	4.3 (13.9)	172.3	0.08	R
3	IIHR-80-1-2	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0	0.00	R
4	Pusa Vishesh	17.8 (24.3)	94.8 (78.3)	79.5 (63.1)	5170.5	12.6 (20.7)	91.4 (73.0)	60.2 (50.9)	2583.5	1.26	HS
5	Pusa Do Mausami	14.1 (20.2)	92.8 (75.1)	74.7 (59.8)	4852.5	23.3 (28.6)	85.5 (67.8)	63.2 (52.7)	2719.6	1.24	HS
6	Phule Ujwala	11.0 (19.3)	92.1 (73.9)	72.4 (58.4)	4710.3	15.2 (22.8)	84.1 (66.5)	58.8 (50.0)	2535.6	1.23	HS
7	IIHR-30-4	11.9 (19.8)	94.8 (77.5)	75.2 (60.1)	4890.1	22.9 (28.1)	88.5 (70.2)	62.7 (52.4)	2684.6	1.25	HS
8	IIHR-49-34-5	0.6 (2.6)	73.6 (59.1)	34.6 (35.9)	2160.9	11.1 (19.4)	79.2 (62.9)	46.7 (43.1)	1975.6	0.88	MR
9	IIHR-46-1	10.5 (18.8)	88.4 (70.2)	70.1 (56.9)	4560.8	7.0 (15.3)	84.8 (67.1)	51.3 (45.7)	2195.9	1.22	HS
10	NDBT-9	9.6 (17.7)	92.3 (73.9)	72.6 (58.5)	4728.9	16.7 (23.2)	84.0 (66.6)	60.5 (51.0)	2613.3	1.23	HS
11	NDBT-7	6.0 (13.7)	88.1 (69.9)	67.3 (55.1)	4381.0	9.4 (17.2)	82.5 (65.3)	56.5 (48.7)	2447.9	1.20	HS
12	CO-1	5.6 (12.9)	81.9 (65.0)	66.2 (54.5)	4330.1	13.7 (21.3)	82.2 (65.3)	56.7 (48.8)	2446.1	1.20	HS
13	IIHR-38-2	6.4 (14.4)	89.3 (71.2)	71.4 (57.6)	4659.8	30.0 (33.0)	87.4 (69.2)	63.9 (53.1)	2723.5	1.23	HS
14	IIHR-151-2	22.3 (28.0)	95.4 (79.8)	81.2 (64.3)	5272.5	20.0 (26.4)	86.6 (68.7)	64.9 (53.7)	2806.4	1.27	HS
15	IIHR-40-1	1.1 (6.0)	70.4 (57.1)	31.1 (33.9)	1921.3	8.8 (17.1)	63.7 (52.9)	39.4 (38.9)	1680.9	0.83	MR
16	IIHR-148-7	4.3 (11.9)	85.6 (67.9)	49.5 (44.7)	3149.8	15.8 (23.4)	87.7 (69.6)	57.1 (49.1)	2436.8	1.09	S
17	IIHR-101-1-7	13.9 (21.9)	98.9 (85.3)	80.5 (63.8)	5239.6	26.6 (30.7)	88.1 (69.9)	69.5 (56.5)	3007.4	1.27	HS
18	IIHR-17-1	11.9 (19.9)	86.4 (68.5)	73.6 (59.1)	4808.8	32.2 (34.5)	87.0 (68.9)	67.1 (55.0)	2872.6	1.24	HS
19	IIHR-8-2	7.6 (16.0)	80.6 (63.9)	62.4 (52.2)	4064.7	17.7 (24.8)	87.7 (69.6)	62.3 (52.1)	2684.6	1.19	HS
20	IIHR-86-11-6	13.5 (21.4)	85.9 (69.0)	75.1 (60.1)	4912.5	22.9 (28.4)	86.6 (68.6)	65.4 (54.0)	2823.3	1.24	HS
21	IIHR-147-4	11.1 (18.9)	93.3 (75.7)	74.1 (59.4)	4824.8	24.0 (29.2)	84.4 (66.8)	58.5 (49.9)	2490.2	1.24	HS
22	IIHR-12-6	8.8 (17.1)	88.6 (70.4)	68.7 (55.9)	4467.9	14.4 (22.2)	86.2 (68.3)	59.2 (50.3)	2548.5	1.22	HS
23	IIHR-80-1-3	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0	0.00	R
24	Preethi	13.7 (21.4)	94.5 (77.4)	76.7 (61.2)	4995.5	15.5 (23.1)	82.9 (65.7)	58.1 (49.6)	2504.4	1.25	HS
25	IIHR-44-2	8.2 (15.9)	90.9 (72.6)	73.7 (59.1)	4816.2	27.0 (31.1)	84.4 (66.8)	58.7 (50.0)	2487.6	1.24	HS
26	NABG-1	8.8 (17.0)	95.0 (77.3)	79.2 (62.9)	5185.2	18.8 (25.7)	85.9 (68.3)	61.9 (51.9)	2669.1	1.26	HS
27	IIHR- Sel-5-8	20.6 (26.0)	99.1 (86.9)	83.8 (66.3)	5450.5	26.6 (31.0)	88.8 (70.6)	68.8 (56.1)	2971.1	1.28	HS
28	IIHR-145	10.7 (18.8)	91.1 (72.8)	72.1 (58.1)	4693.8	25.5 (29.8)	82.5 (65.3)	61.4 (51.6)	2634.1	1.23	HS
29	Hirkani	10.2 (18.6)	98.0 (83.3)	81.3 (64.4)	5316.1	20.7 (26.5)	88.8 (70.8)	63.5 (52.8)	2730.0	1.27	HS
30	Meghana	10.8 (18.9)	96.6 (79.5)	77.6 (61.7)	5057.7	23.3 (28.7)	87.7 (69.6)	64.7 (53.5)	2784.4	1.26	HS
31	IIHR-14-4	13.2 (21.2)	97.8 (81.8)	77.0 (61.4)	5006.7	22.5 (28.2)	82.9 (65.7)	63.6 (52.9)	2749.4	1.25	HS
CD at 5%		7.5	7.0	3.2	-	7.8	4.7	2.0	-	-	-
CV (%)		27.8	6.4	3.8	-	20.8	4.6	2.6	-	-	-

\*Mean PDI of all the observations; WAI week after infection, DAI days after inoculation; arecine transformed values in parenthesis, R resistant, MS moderately susceptible, S susceptible, HS highly susceptible

The rate of disease growth occurred over time epoch was recorded and the rate of disease growth at weekly interval for selected highly susceptible, moderately susceptible and highly resistant genotypes is also depicted in Fig. 1. Perusal of the graph showed that disease severity in Arka Harit and IIHR-Sel-5-8 was above 80 percent in second week after the infection started and reached its maximum during fifth week. Whereas, slow disease progress was observed in moderately susceptible genotypes viz., IIHR-40-1 and IIHR-49-34-5. IIHR-144-1 recorded a mild disease incidence at the end of the season and IIHR-80-1-2 and IIHR-80-1-3 were completely free from the disease. Hence, appropriate management strategies for controlling the disease should be oriented within the period identified in the investigation separately as envisaged by the rate of disease growth.

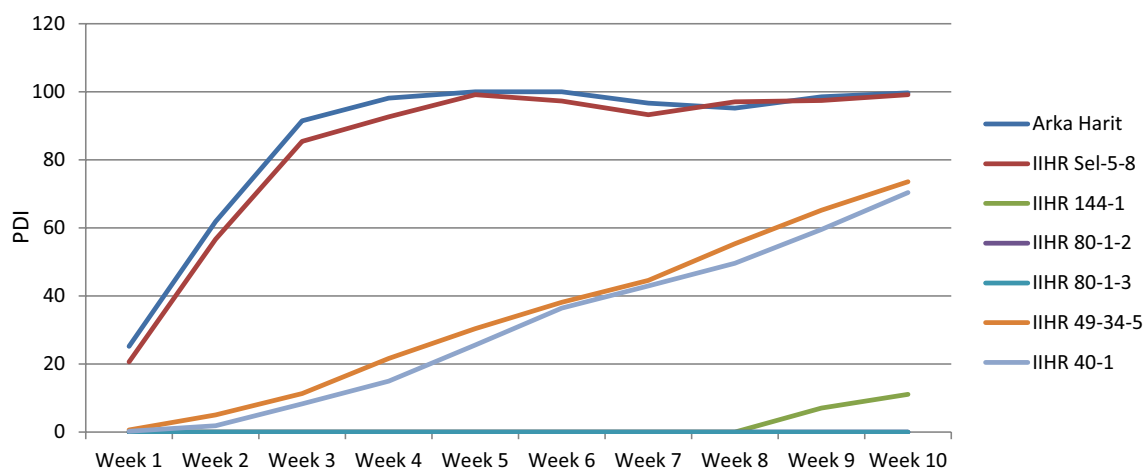
The relationship between per plot (7.5 m<sup>2</sup>) fruit yield and PDI (Table 1) indicates that genotypes IIHR-148-7 (10.36 kg), IIHR-40-1 (10.34 kg) recorded relatively higher yield followed by IIHR-44-2 (9.07 kg) and IIHR-30-4 (8.36 kg) in the presence of disease. Being wild genotypes with smaller sized fruits, IIHR-144-1 (4.17 kg), IIHR-80-1-2 (1.8 kg) and IIHR-80-1-3 (0.97 kg) recorded less yield even in the absence of the disease. The low yielding resistant genotypes may be used as donors to develop varieties with high yield and desirable level of resistance along with good horticultural traits.

Various workers have recorded slow mildewing as a function of disease progress over unit time (Krishna and Mishra 1989; Roderick and Jones 1991; Chaudhary and Banyal 2016). AUDPC values varied greatly from 0.0 (IIHR-80-1-2 and IIHR-80-1-3) to 5629.8 (Arka Harit) under natural epiphytotic conditions (Table 1) and 0.0 (IIHR-80-1-2 and IIHR-80-1-3) to 3007.4 (IIHR-101-1-7) in artificial screening (Table 1) which indicates the importance of measuring disease progress in the germplasm. The

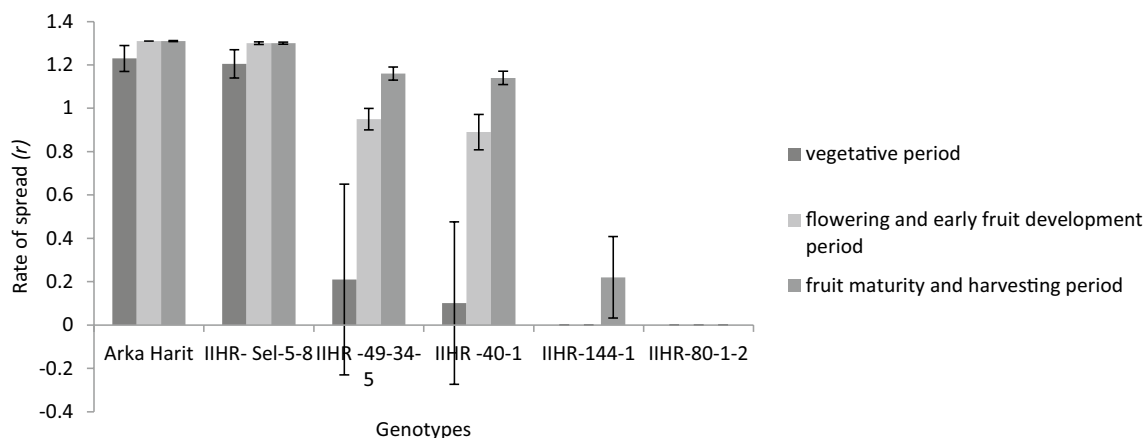
genotypes with lower AUDPC, such as IIHR-80-1-2 and IIHR-80-1-3, and IIHR-144-1 also had lower PDI rating, while most susceptible sources with higher AUDPC values also had higher PDI scores. The apparent infection rate (*r*) of genotypes at various stages showed a wide variation (Table 1). The highest average '*r*' value was observed in the genotype Arka Harit (1.29/day) and the least in IIHR-80-1-2 and IIHR-80-1-3 (0.00/day). Young plants of genotypes, IIHR-40-1 and IIHR-49-34-5 in its vegetative growth period (35 days) showed slow disease development which suggested that with increase in the age, the susceptibility of the crop increases (Fig. 2). This information becomes a useful guide for time of application of the fungicides for its best efficacy in management of the powdery mildew pathogen. Also, the genotype, IIHR-40-1 recorded higher yield in presence of disease (Table 1), hence based on these results, this genotype can be used for cultivation in disease prone areas with the limited application of fungicides at correct stage for better profits.

### Correlation studies between natural screening and artificial screening

Correlation between PDI with AUDPC under natural and artificial screening showed values of 0.99 and 0.82 ( $P < 0.01$ ), respectively (Table 2), indicating a good agreement between the methods tested for disease assessment. The correlation between PDI of natural screening (adult plant level) and PDI of artificial screening (seedling stage) also showed significant positive correlation (0.824) complementing each other. The use of a controlled screening system allows initiating the disease screening trials of powdery mildew at specific time in the season, regardless of the weather.



**Fig. 1** Disease progress curve for powdery mildew in resistant, moderately resistant and susceptible genotypes of bitter melon (weekly basis)



**Fig. 2** Disease progress in selected genotypes of bitter gourd at different stages of crop growth

**Table 2** Correlation coefficient among qualitative and quantitative parameters of disease on natural and artificially screened bitter gourd genotypes against powdery mildew

Parameter	PDI (NS)	AUDPC (NS)	PDI (AS)	AUDPC (AS)
PDI (NS)	1			
AUDPC (NS)	0.999**	1		
PDI (AS)	0.824**	0.826**	1	
AUDPC (AS)	0.954**	0.951**	0.828**	1

*PDI* percent disease index, *AUDPC* area under disease progress curve, *NS* natural screening, *AS* artificial screening

\*\*Indicates significance at  $P < 0.01$

### Nonlinear statistical modeling

Growth models for plant disease play an important role in understanding the dynamics of growth pattern over time. It is generally observed that a plant disease progresses at a slow rate in the beginning; subsequently it picks up and reaches a maximum, beyond which it starts declining. This type of behaviour can be readily captured by the well-known logistic growth model (Cambell and Madden 1990; Jeger 1984). The data pertaining to bitter gourd powdery mildew system are given in Table 3. Parameter estimates of fitted models, measures of goodness of fit of the model ( $R^2$  and MSE) along with the tested, measures of model adequacy were also presented.

Results showed that severity of powdery mildew over time was explained by logistic model fit to the extent of 99% in majority of the genotypes and can be used to provide quantitative information about the progress of the disease. Mean square error values ranges from 2.3 to 36.03. Further, examination of assumptions about residuals showed that errors are randomly distributed as the run test statistics value of all the genotypes are well within

the critical region of 1.96. However, the tests of normality (Shapiro Wilk test), resulted insignificant values for all the genotypes. This further strengthened the statistical adequacy of the fitted model.

For each genotype, weekly growth of powdery mildew was calculated by computing the values of the derivative  $dX/dt$ , where  $X(t)$  denote the powdery mildew severity at time  $t$ . Evidently, a fungicide should be sprayed for controlling the disease when the rate of growth of fungus ( $dX/dt$ ) was maximum and not when the disease severity was maximum. The time ( $t$ ) for which the mildew severity growth was maximum, is given by  $t = \ln(b/a)$  where, ' $b$ ' refers to the incremental relative rate of relative growth rate of the disease and ' $a$ ' is the intrinsic growth rate. It is depicted that the maximum mildew severity growth rate occurred at  $t = 2$  or  $t = 3$  weeks for most of the susceptible genotypes and  $t = 4.24$  for moderately susceptible genotype IIHR-40-1. This type of information will help in optimising the fungicide spray schedule for disease control. Similar growth models using non linear statistic have been established for powdery mildew development in mango (Sinha and Prajneshu 2002) and in downy mildew of grape (Venugopalan and Vijay 2017).

In conclusion, utilization of host plant resistance is the effective method to control powdery mildew disease. The identified resistant genotypes from this experiment viz, IIHR-80-1-2, IIHR-80-1-3, IIHR 144-1 can be used for breeding powdery mildew resistant bitter gourd varieties/hybrids by introgressing gene(s) into elite lines. At the same time, to know that the single or multiple genes responsible for resistance in these sources, needs further genetic analysis. This effort will go a long way in resistance breeding programme in bitter gourd for developing durable resistance to minimize dependency on chemical fungicides.



**Table 3** Fitting of logistic model to powdery mildew severity in bitter gourd (non linear regression analysis)

Sl. no.	Genotypes	a	b	c	R <sup>2</sup> %	MSE	Run test (Z)	SW stat	t
1	Arka Harit	1.71	17.24	98.72	99	4.28	0.21*	0.84*	2.31
2	IIHR-144-1	0.26	6.04	5.82	27	14.39	1.92*	0.75*	3.12
3	IIHR-80-1-2**	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	Pusa Vishesh	1.71	28.22	93.35	97	23.87	0.49*	0.85*	2.80
5	Pusa Do Mausami	2.20	95.66	88.20	97	26.58	0.21*	0.92*	3.77
6	Phule Ujwala	2.31	138.26	86.22	98	18.85	0.33*	0.97*	4.09
7	IIHR-30-4	1.86	58.85	90.41	99	7.32	0.49*	0.92*	3.45
8	IIHR-49-34-5	0.50	25.99	84.19	98	8.88	1.01*	0.94*	3.95
9	IIHR-46-1	1.77	58.12	85.49	97	20.76	0.49*	0.82*	3.49
10	NDBT-9	1.94	79.09	88.01	97	22.88	0.33*	0.91*	3.71
11	NDBT-7	2.51	459.06	83.09	99	8.79	0.49*	0.95*	5.21
12	CO-1	2.31	230.98	81.08	99	10.01	0.49*	0.84*	4.60
13	IIHR-38-2	2.31	198.70	86.71	99	6.29	0.49*	0.93*	4.45
14	IIHR-151-2	1.47	15.62	94.87	99	5.72	0.00*	0.89*	2.36
15	IIHR-40-1	0.56	39.10	76.33	98	9.90	0.91*	0.91*	4.24
16	IIHR-148-7	0.61	17.39	85.67	99	7.75	0.00*	0.92*	3.34
17	IIHR-101-1-7	1.46	22.94	96.93	99	5.15	1.01*	0.92*	2.75
18	IIHR-17-1	1.94	63.69	88.03	99	7.72	0.00*	0.88*	3.49
19	IIHR-8-2	2.11	95.94	74.84	97	17.13	0.21*	0.98*	3.81
20	IIHR-86-11-6	1.83	52.82	90.04	98	16.01	1.01*	0.94*	3.36
21	IIHR-147-4	2.09	103.37	89.18	96	36.03	0.21*	0.95*	3.90
22	IIHR-12-6	1.86	70.97	83.72	98	13.57	0.21*	0.97*	3.64
23	IIHR-80-1-3**	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
24	Preethi	1.73	39.69	91.80	98	13.66	0.33*	0.95*	3.13
25	IIHR-44-2	1.78	47.60	88.92	99	2.89	1.19*	0.94*	3.29
26	NABG-1	2.13	83.41	94.17	99	6.24	1.06*	0.88*	3.66
27	IIHR-Sel-5-8	1.63	18.81	97.04	99	4.22	0.00*	0.89*	2.44
28	IIHR-145	1.96	63.14	86.07	98	18.21	0.33*	0.92*	3.47
29	Hirkani	2.06	74.50	96.84	99	2.34	1.89*	0.77*	3.59
30	Meghana	1.88	58.00	93.11	98	11.74	0.33*	0.91*	3.43
31	IIHR-14-4	1.62	40.05	93.85	99	6.93	0.21*	0.91*	3.20

\*Significant at  $P = 0.05$ ; \*\*There was no disease incidence

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