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# Genetic analysis of resistance to watermelon bud necrosis orthotospovirus in watermelon [*Citrullus lanatus* (Thunb.) Matsum & Nakai]

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## Abstract

An experiment was conducted to study the genetics and nature of gene action of resistance to watermelon bud necrosis orthotospovirus (WBNV) in watermelon. The experimental materials comprised of two resistant (BIL-53 and IIHR-19) and one susceptible (IIHR-140) parents. Each of the resistant parents was crossed with the susceptible parent to develop six generations (P1, P2, $F_1$, $F_2$, BC1 and BC2) to study genetics. The results of segregation in $F_2$ and backcross progenies suggested that resistance is governed by a major dominant gene along with other background minor genes in both the crosses. BIL-53 was found to possess higher degree of resistance with simple inheritance and hence may be of interest to breeders. Simple selection can be effective for improving the trait in the cross BIL-53 × IIHR-140 as additive gene action is prevalent.

**KEYWORDS**
genetics, orthotospovirus, resistance, watermelon, WBNV

## 1 | INTRODUCTION

Watermelon [*Citrullus lanatus* (Thunb.) Matsum & Nakai] is an important specialty crop accounting for seven per cent of the agricultural area devoted to vegetable crops. The world watermelon harvested area is about 3.50 million hectares with a production of 111 million tonnes. China is the largest producer and consumer with an annual production of about 79.2 million tonnes (FAO, 2016). In India, watermelon is a major cucurbit cultivated in an area of 82 thousand hectares with a production of 2.038 million tonnes (NHB, 2015). The productivity levels are constrained by the occurrence of various diseases. Important among them is thrips-transmitted watermelon bud necrosis orthotospovirus (WBNV) (Family: *Tospoviridae*, *Bunyavirales*) that belongs to the watermelon silver mottle orthotospovirus (WSMoV) serogroup (Adams et al., 2017; Jain, Mandal, Pappu, & Holkar, 2015; Jain, Pappu, Pappu, Krishnareddy, & Vani, 1998). It was first recorded during 1991 infecting watermelon at Indian Institute of Horticultural Research (IIHR), Bangalore, India (Singh & Krishnareddy, 1996) and later found to infect several other cucurbits, such as cucumber, ridge gourd and muskmelon (Jain, Bag, Umamaheswaran, & Mandal, 2007; Jain et al., 1998; Kumar, Mandal, Geetanjali, Jain, & Jaiwal, 2010; Mandal, Jain, Chaudhary, & Varma, 2003). Recently, WBNV was also detected in chilli pepper, tomato (Kunkalikar et al., 2011) and chrysanthemum (Holkar et al., 2017) in northern India. WBNV is widely distributed and endemic in many states of India, such as Andhra Pradesh, Gujarat, Haryana, Himachal Pradesh, Karnataka, Kerala, Madhya Pradesh, Maharashtra, Orissa, Punjab, Rajasthan, Tamil Nadu, Uttar Pradesh and West Bengal (Mandal et al., 2012). It has a tripartite genome made of three single-stranded RNA molecules that are each bound by a nucleocapsid protein (Jain et al., 1998). The phylogenetic analysis of its proteins revealed that WBNV is closely related to WSMoV, groundnut bud necrosis

orthotospovirus (GBNV) and capsicum chlorosis orthotospovirus (CaCV) (Li et al., 2011). Natural infection of WSMoV on watermelon has been reported in Japan, Indonesia (Kameya-Iwaki, Hanada, Honda, & Tochihara, 1988) and China (Rao, Liu, Wu, & Li, 2011), while no such report is available from India yet. CaCV which is a serious pathogen of capsicum and chilli was also reported to infect tomato and groundnut (Chen, Xu, Yan, & Wang, 2007; Persley, Thomas, & Sharman, 2006 and Premachandra, Borgemeister, Maiss, Knierim, & Poehling, 2005), but not yet reported to infect watermelon. GBNV was found not to infect watermelon (Holkar et al., 2017). In India, WBNV is naturally transmitted by a melon thrips, *Thrips palmi* Karny (Rajasekharam, 2010; Rebijith, Asokan, Hande, & Kumar, 2016; Rebijith, Asokan, Krishna Kumar, Krishna, & Ramamurthy, 2012). Disease incidence from 39% to 100% (Krishnareddy & Singh, 1993) with a yield loss up to 100% (Jain et al., 1998, 2007; Kunkalikar et al., 2011; Singh & Krishnareddy, 1996) has been reported. The field symptoms of WBNV in watermelon initially develop as shortened internodes, upright growth of younger shoots and necrosis on apical bud, stem, petiole and fruit stalk. Infected plants produce unmarketable small, deformed fruits with uneven surface and necrotic or chlorotic rings, depending on the cultivar (Mandal et al., 2012).

Although several control measures (cultural, chemical and biological) have been suggested for vector management, practically no effective control has been achieved so far for the management of WBNV (Krishna Kumar, Venkatesh, Kalleshwaraswamy, & Ranganath, 2006). Hence, host plant resistance has been suggested as the most feasible management option for the control of this disease (Riley & Pappu, 2000). However, not many efforts have been made in this direction; Pandey and Pandey (2001) reported Durgapura Selection, RHRWH-2 and EC-393243 as resistant to WBNV, while Holkar, Basavaraj, Mandal, and Jain (2018) reported *Citrullus colocynthis* as resistant to this disease. However, no further progress has been reported using these resistant sources.

There are no studies on genetics of resistance to WBNV in watermelon to date. However, the reports of resistance to orthotospoviruses affecting other vegetable crops range from monogenic to polygenic inheritance. Resistance to tomato spotted wilt orthotospovirus (TSWV) in tomato is controlled by a single dominant gene (Rosello, Ricarte, Diez, & Nuez, 2001 and Stevens, Scott, & Gergerich, 1992) and in some cases few recessive genes, apparently by more than four genes (Kumar & Irulappan, 1992; and Maluf Toma-Braghini, & Corte, 1991). In tomato, GBNV resistance is inherited by a single dominant gene (Ramana et al., 2011). Resistance to TSWV (Black, Hobbs, & Kammerlohr, 1996; Boiteux & de Avila, 1994; Moury, Palloix, Selassie, & Marchoux, 1997) and CaCV (Persley, Sharman, Mcgrath, & Garland, 2005) in pepper is governed by single dominant genes and is expressed as hypersensitive response. Inheritance to tomato necrotic ring orthotospovirus resistance (TNRV) in pepper is controlled by single recessive gene (Puangmalai, Potapohn, Akarapisarn, & Pascha, 2013). Some of the genes identified to confer resistance against orthotospoviruses are as follows: *Swa1*, *Swb1* (Finlay, 1953), *Sw-5* (Stevens et al., 1992), *Sw-6* (Rosello et al., 1999) and *Sw-7* (Stevens et al., 2006) are dominant genes, while *sw2*, *sw3* and *sw4* (Finlay, 1953) are recessive genes conferring resistance to TSWV in tomato. The *Tsw* gene confers dominantly inherited resistance to TSWV in *Capsicum* spp. (Jahn et al., 2000).

As WBNV is prevalent in major watermelon-growing areas of India, there is an immediate need to develop varieties/hybrids possessing resistance to this disease. In this direction, efforts are underway at IIHR, Bengaluru, India, to develop varieties resistant to WBNV. During the period 2010–2014, the protocol to screen for WBNV resistance under natural epiphytotic conditions was standardized and a total of 128 germplasm and advanced breeding lines were evaluated. Among them, the genotypes BIL-53 and IIHR-19 have been identified as resistant to WBNV. The current experiment was taken up to understand the genetics of resistance in these lines.

## 2 | MATERIALS AND METHODS

### 2.1 | Plant material

The experimental materials for the study comprised of two WBNV-resistant inbred lines, *viz*., BIL-53 and IIHR-19. BIL-53 is a backcross inbred line ($BC_1F_6$) derived from an intraspecific cross between IIHR-82 (*Citrullus lanatus* var. *citroides*) and 'Arka Manik' (recurrent parent), which is a very popular variety in Indian subcontinent. IIHR-19 is a canary yellow-fleshed inbred line derived from segregating progeny of a Taiwanese introduction. These lines were crossed to a WBNV-susceptible, red-fleshed icebox inbred, IIHR-140 to generate $F_1$, $F_2$, BC1 (backcrossed to resistant parent) and BC2 (backcrossed to susceptible parent) progeny during 2015–2016.

The four progeny ($F_1$, $F_2$, BC1 & BC2) along with parental lines involving BIL-53 were evaluated for disease reaction during summer, 2016, and those involving IIHR-19 during summer, 2017. The evaluation trial was laid out in a randomized complete block design with three replications. Each replication of P1, P2 and $F_1$ consisted of 10 plants; BC1 and BC2 consisted of 15 plants; and $F_2$ consisted of 50 plants. All package of practices except insecticidal sprays were followed to raise the crop. Data were recorded for disease severity, plant survival (%) and vine length (cm) at 10 days interval, starting from 35 to 65 days after sowing (DAS).

### 2.2 | Disease screening method

Natural epiphytotic screening for reaction to WBNV was carried out during summer season, when natural vector population is high favouring natural disease occurrence. Paired row spot planting technique under unmulched condition along with infector genotypes and yellow ribbons were used to attract thrips (vector of WBNV). Popular commercial varieties, *viz*., NS-295 and 'Arka Manik' that are susceptible to WBNV were used as infector lines. These were planted 10 days prior to planting of test progeny to build up the field inoculum.

The disease severity was scored visually for symptoms on a scale of 0–3 as suggested by Sugiyama, Okuda, and Sakata (2009) with slight modifications, where 0 = no symptom, 1 = slight crinkling of leaves, 2 = crinkling with yellowing or silver mottling of leaves and 3 = dieback or severe bud necrosis. The disease severity scores of individual plants thus recorded were used to calculate per cent disease index (PDI) using the following formula:

$$\text{PDI} = \frac{\text{Sum of all ratings}}{\text{Total number of observations} \times \text{Maximum rating}} \times 100$$

The area under disease progress curve (AUDPC) (Campbell & Madden, 1990) was calculated using the formula:

$$\text{AUDPC} = \sum_{i=1}^{n=1}\left[\left\{\frac{X_i + X_{i+1}}{2}\right\} \times \{t_{i+1} - t_i\}\right]$$

where $X_i$ = disease index at $i^{th}$ observation, $X_{i+1}$ = disease index at $i$ + 1st observation $t_{i+1} - t_i$ = number of days between two observations and $n$ = total number of observations.

The interval when the checks attained 80% threshold PDI (45 DAS in BIL-53 × IIHR-140 and 65 DAS in IIHR-19 × IIHR-140) was used for interpretation of the results for genetics and gene action.

### 2.3 | Disease diagnosis

To confirm WBNV infection in the test entries during screening, direct antigen-coated enzyme-linked immunosorbent assay (DAC-ELISA) and reverse transcription–polymerase chain reaction (RT-PCR) techniques were used. DAC-ELISA was performed using general orthotospovirus antiserum as per the protocol described by Hobbs, Reddy, Rajeshwari, and Reddy (1987). To specifically identify WBNV, RT-PCR was performed using primers specific to the nucleocapsid (N) gene of WBNV as described by Holkar et al. (2017).

### 2.4 | Classical Mendelian segregation pattern of resistance

In this experiment, different Mendelian ratios were tested to fit observed segregation of WBNV resistance into a classical Mendelian model. The disease reaction of individual plants in $F_2$ and backcross progeny were classified based on the PDI ± *SE* of resistant and susceptible parents into two major phenotypic classes, *viz*., resistant and susceptible as suggested by Thirthamallappa and Lohithaswa (2000) (Supporting information Table S1). The total number of plants falling into different classes was counted and subjected to chi-square analysis for goodness of fit to various classical Mendelian ratios as suggested by Panse and Sukhatme (1985).

### 2.5 | Nature and magnitude of gene effects for WBNV resistance

The individual plant PDI values were used for generation mean analysis. Scaling test as suggested by Mather (1949) and Hayman and Mather (1955) was conducted. A six-parameter model as suggested by Hayman (1958) and Jinks and Jones (1958) was employed for estimation of various genetic components. Statistical analysis was performed using Windostat Version 9.2 from Indostat services, Hyderabad, India.

## 3 | RESULTS

### 3.1 | Symptoms of WBNV during field screening

The initial symptoms of the disease on leaves appeared as chlorotic spots, mild mosaic mottling, crinkling, yellowing, narrowing of leaf lamina, silvering of leaves, dark brown- or black-coloured necrotic spots and rugosity of young leaves. Further mid-veins and lateral veins of leaves turned black and became thick and distorted (Supporting information Figure S1).

Affected plants were severely stunted, had shortened internodes and became very brittle with upright growth of younger branches. Other predominant symptoms were the presence of longitudinal brown necrotic streaks on vines, tendrils and petioles. As the disease progressed, the necrotic streaks on stem and the growing branches started drying from the tip leading to bud necrosis and dieback.

### 3.2 | Serological and molecular confirmation of WBNV infection

Samples from different types of disease symptoms observed in field were selected for the DAC-ELISA using general orthotospovirus antiserum. The DAC-ELISA absorbance values (405 nm) ranged from 1.14 to 2.70 (Supporting information Table S2). This was comparatively higher than the buffer (0.43) and healthy control (0.45) confirming infection.

To specifically identify WBNV infection, RT-PCR was performed using primers specific to the nucleocapsid (N) gene of WBNV. RNA was isolated from the healthy plant and from plants showing three major symptoms (leaf crinkling, silvering and brittleness of leaves and bud necrosis) observed in field to perform RT-PCR. The diseased plant samples recorded an amplicon of ~750 bp size, while it was absent in healthy plant sample (Supporting information Figure S2).

### 3.3 | Mean performance of different generations

The infector rows were planted 10 days in advance to the test entries to build up inoculum and avoid escapes. The check varieties served as a reference to decide the escapes, wherein 96.7 (IIHR-19 × IIHR-140) to 98.7 (BIL-53 × IIHR-140) PDI was recorded by 65 DAS confirming that escapes were less than 5%.

Analysis of variance revealed significant differences among the generations for both the crosses. The means for different generations of the two crosses primarily provide an idea of their disease response (Table 1). The interval when the checks attained 80% threshold PDI, *that is*, at 45 and 65 DAS in the crosses BIL-53 × IIHR-140 and IIHR-19 × IIHR-140, respectively, were used for the genetic analysis and interpretation of PDI, plant survival and vine length.

**TABLE 1** Per cent disease index (PDI) and area under disease progress curve (AUDPC) values in different generations of the crosses BIL-53 × IIHR-140 and IIHR-19 × IIHR-140 under natural epiphytotic conditions of watermelon bud necrosis orthotospovirus

| | BIL-53 × IIHR-140 | | | | | IIHR-19 × IIHR-140 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Generations** | **35 DAS** | **45 DAS[a]** | **55 DAS** | **65 DAS** | **AUDPC** | **35 DAS** | **45 DAS** | **55 DAS** | **65 DAS[a]** | **AUDPC** |
| P1 | 4.4 (10.9) c | 42.2 (40.5) d | 72.2 (58.3) d | 87.8 (69.6) cd | 1,594.5 d | 6.5 (13.1) bc | 22.8 (28.5) b | 43.6 (41.3) d | 62.4 (52.2) d | 1,009.5 c |
| P2 | 14.4 (21.0) bc | 75.6 (60.7) abc | 91.11 (72.7) bc | 97.8 (82.9) ab | 2,277.8 abc | 18.9 (25.4) a | 44.5 (41.7) a | 60.0 (50.8) bc | 85.6 (68.8) b | 1,566.7 ab |
| $F_1$ | 12.2 (18.9) c | 50.0 (44.9) cd | 73.33 (59.0) d | 81.1 (64.4) d | 1,715.0 d | 4.4 (12.0) c | 23.3 (28.7) b | 43.3 (41.2) d | 56.7 (48.8) d | 972.2 c |
| $F_2$ | 8.7 (17.0) c | 54.9 (47.80)cd | 79.6 (63.2) d | 87.1 (69.0) cd | 1,842.2 cd | 7.7 (16.0) abc | 27.2 (31.3) b | 47.4 (43.5) cd | 68.0 (55.7) cd | 1,124.7 c |
| BC1 | 11.1 (16.3.) c | 51.1 (45.8) cd | 71.9 (58.0) d | 80.7 (64.3) d | 1,705.9 d | 4.7 (11.1) c | 25.7 (30.3) b | 49.7 (44.8) cd | 69.1 (56.4) cd | 1,122.8 c |
| BC2 | 22.2 (25.6) bc | 63.7 (54.8) bcd | 80.74 (64.2) cd | 88.9 (70.6) cd | 2,034.5 bcd | 15.1 (22.8) a | 35.4 (36.4) ab | 65.1 (53.9) ab | 80.8 (64.2) bc | 1,484.7 b |
| Arka Manik | 36.9 (37.4) ab | 80.82 (64.1) ab | 90.5 (73.9) b | 92.1 (75.3) bc | 2,426.4 ab | 14.4 (22.0) ab | 51.4 (45.8) a | 72.8 (58.7) ab | 88.6 (70.3) b | 1,756.9 ab |
| NS-295 | 49.5 (44.7) a | 88.5 (70.8) a | 98.4 (82.9) a | 98.7 (84.6) a | 2,687.8 a | 16.7 (24.0) a | 51.1 (45.7) a | 75.5 (60.5) a | 96.7 (81.4) a | 1,833.3 a |
| *F* value | 4.2* | 4.1* | 9.3** | 7.3** | 6.5** | 3.0* | 5.1** | 9.1** | 17.0** | 10.5 ** |
| *SE* (*m*) | 7.6 | 8.1 | 3.2 | 2.5 | 154.0 | 3.5 | 5.4 | 4.3 | 3.4 | 106.1 |
| CD@ 5% | 23.1 | 24.9 | 10.0 | 7.6 | 467.4 | 9.5 | 16.5 | 13.1 | 10.4 | 322.0 |
| CV (%) | 65.7 | 22.2 | 6.9 | 4.8 | 13.1 | 29.7 | 26.5 | 13.0 | 7.7 | 13.5 |

*Notes*. Values in parentheses are arc sine transformed, P1: resistant parent, P2: susceptible parent and DAS, days after sowing.
Means followed by different lowercase letters are significantly different ($p$ = 0.05: DMRT).
[a]Interval when the checks attained 80% threshold PDI.
*Significant at $p \leq 0.05$. **Significant at $p \leq 0.01$ and.

In general, PDI values for all the generations increased over different intervals of observation in both the crosses (Table 1). Performance of different generations based on mean PDI at 45 DAS for the cross BIL-53 × IIHR-140 revealed that $F_1$ (50.0), $F_2$ (54.9), BC1 (51.1) and BC2 (63.7) exhibited an intermediate reaction compared to P1 (42.2) and P2 (75.6). In the cross IIHR-19 × IIHR-140, performance of different generations based on mean PDI at 65 DAS revealed that $F_1$ (56.7) exhibited a higher resistance compared to P1 (62.4), while $F_2$ (68.0), BC1 (69.1) and BC2 (80.8) exhibited intermediate reaction compared to P1 (62.4) and P2 (85.6). Disease progress curves (Table 1) revealed that the progress of the disease was slow in P1, $F_1$, $F_2$ and BC1, whereas rapid progress of the disease was noticed in case of P2 and BC2, which was almost nearly equal to that of susceptible checks. The AUDPC values are presented in Table 1. The AUDPC ranged from 1,594.5 to 2,277.8 in different generations of the cross BIL-53 × IIHR-140 while checks ranged from 2,426.4 to 2,687.8. The P1 recorded lowest AUDPC (1,594.5), while $F_1$ (1,715.0), $F_2$ (1,842.2) and BC1 (1,705.9) were statistically on par with P1. P2 exhibited highest AUDPC (2,277.8) followed by BC2 (2,034.5). For the cross IIHR-19 × IIHR-140, the AUDPC values ranged from 972.2 to 1,566.7 in different generations (Table 1). The lowest AUDPC was observed in $F_1$ (972.2), while P1 (1,009.5), $F_2$ (1,124.7) and BC1 (1,122.8) recorded AUDPC values at par with $F_1$. However, P2 (1,566.7) recorded highest AUDPC value, which is followed by BC2 (1,484.7). The checks ranged from 1,756.9 to 1,833.3.

The results of the mean plant survival (%) and vine length (cm) for different generations of the two crosses along with susceptible checks are presented in Table 2. Analysis of variance revealed significant differences among the various generations for plant survival in both crosses, whereas only BIL-53 × IIHR-140 exhibited significant difference for vine length.

The maximum plant survival for the cross BIL-53 × IIHR-140 was observed in P1 (100.0%) and BC1 (100.0%). However, $F_1$ (93.3%), $F_2$ (93.3%), BC2 (95.7%) and P2 (83.3%) recorded lower plant survival than P1 and BC1, but they were not significantly different. In the cross, IIHR-19 × IIHR-140, $F_1$ (100.0%) recorded higher plant survival than P1 (83.8%). The survival rate in $F_2$ (78.9%) and BC1 (67.2) were in between P1 and P2; survival in BC2 (63.6%) and P2 (60.0%) was at par. In contrast to the different generations of the two crosses, the checks recorded a lower survival rate ranging from 30.3% to 52.1%.

In the cross BIL-53 × IIHR-140, $F_1$ (58.9 cm) and BC1 (51.4 cm) recorded higher vine length compared to P1 (48.1 cm), while $F_2$ (45.5 cm) was on par with P1 (48.1 cm). A drastic reduction in vine length was observed in P2 (18.5 cm), BC2 (36.8 cm) and checks (17.3 to 24.3 cm). The various generations of the cross, IIHR-19 × IIHR-140, did not show significant difference for vine length. However, $F_1$ (101.9 cm), $F_2$ (105.4 cm), BC1 (95.0 cm) and BC2 (92.0 cm) generations recorded higher vine length than P1 (84.4 cm) and P2 (78.9 cm), while check varieties (77.5 to 78.6 cm) recorded the least vine length.

### 3.4 | Classical Mendelian segregation pattern of resistance

The segregation in $F_2$ and backcross progeny of the two crosses was subjected to chi-square analysis for assessing the goodness of fit to various classical Mendelian ratios.

For the cross BIL-53 × IIHR-140, the resistant and susceptible plants observed in $F_2$ were 109 and 41, respectively. Of various Mendelian ratios tested $F_2$ population segregated in a 3:1 ratio (resistant: susceptible) ($\chi^2$ = 0.57; $p$ = 0.45), indicating that single-dominant gene confers resistance to WBNV in this cross. However in

**TABLE 2** Plant survival and vine length in different generations of the crosses BIL-53 × IIHR-140 and IIHR-19 × IIHR-140 under natural epiphytotic conditions of watermelon bud necrosis orthotospovirus

| | BIL-53 × IIHR-140 | | IIHR-19 × IIHR-140 | |
|---|---|---|---|---|
| Generations | Plant survival (%) @ 45 DAS | Vine length (cm) @ 45 DAS[a] | Plant survival (%) @ 65 DAS | Vine length (cm) @ 65 DAS[a] |
| P1 | 100.0 (89.7) | 48.1 ± 21.9 | 83.8 (66.9) | 84.4 ± 47.5 |
| P2 | 83.3 (66.1) | 18.5 ± 13.0 | 60.0 (50.9) | 78.9 ± 39.6 |
| $F_1$ | 93.3 (77.6) | 58.9 ± 44.4 | 100.0 (89.6) | 101.9 ± 40.5 |
| $F_2$ | 93.3 (75.2) | 45.5 ± 24.7 | 78.9 (63.1) | 105.4 ± 49.1 |
| BC1 | 100.0 (89.7) | 51.4 ± 27.4 | 67.2 (55.3) | 95.0 ± 48.2 |
| BC2 | 95.7 (79.9) | 36.8 ± 23.4 | 63.6 (53.6) | 92.0 ± 43.0 |
| Arka Manik | 52.1 (46.2) | 17.3 ± 13.6 | 40.7 (39.6) | 78.6 ± 35.4 |
| NS-295 | 30.3 (33.2) | 24.3 ± 10.0 | 33.3 (34.9) | 77.5 ± 36.0 |
| *F* value | 80.8** | 10.4** | 7.9** | 7.3 |
| *SE* (*m*) | 2.9 | 4.9 | 7.8 | 7.3 |
| CD@ 5% | 8.8 | 15.0 | 23.9 | - |
| CV (%) | 6.1 | 23.0 | 20.5 | 14.2 |

*Notes*. Values in parentheses are arc sine transformed, [a]mean vine length and their standard deviation, P1: resistant Parent, P2: susceptible parent and DAS, days after sowing.
**Significant at $p \leq 0.01$.

BC1, the segregation ratio had goodness of fit with 1:0 ($\chi^2$ = 0.00; $p$ = 1.00), whereas in BC2 both the ratios tested (1:1 and 1:0) had goodness of fit. Among them, 1:1 ratio recorded the highest probability (0.07). The results thus suggest that resistance in this cross is governed by a major dominant gene along with other background minor genes. For the cross IIHR-19 × IIHR-140, the number of resistant and susceptible plants in $F_2$ generation was 102 and 45, respectively. Of the several ratios tested $F_2$ population had best fit with 3:1 ($\chi^2$ = 2.98; $p$ = 0.08), indicating single-dominant gene inheritance. However, segregation in BC1 has goodness of fit with both the ratios tested. The $\chi^2$ and probability values are $\chi^2$ = 3.97; $p$ = 0.04 and $\chi^2$ = 0.36; $p$ = 0.55 for 1:1 and 1:0 ratios, respectively, whereas in BC2 the segregation was fitting 1:1 ($\chi^2$ = 0.23; $p$ = 0.63) and 1:0 ($\chi^2$ = 3.20; $p$ = 0.07) ratios. The results are therefore inconclusive of simple Mendelian segregation in this cross but tend to suggest the involvement of a major dominant gene along with minor background genes. This needs to be further studied for confirmation with a larger population size.

### 3.5 | Nature and magnitude of gene effects for WBNV resistance

Analysis of variance revealed significant differences in PDI at all the intervals, suggesting that there exists a sufficient variation among different generations of the two crosses studied. The results of scaling tests are presented in Table 3. The nonsignificance of scaling test indicated adequacy of simple additive-dominance model and the absence of nonallelic interaction for the cross BIL-53 × IIHR-140. However, the scaling test was significant for the cross IIHR-19 × IIHR-140 revealing the presence of epistasis.

The estimates of different genetic components, *viz*., mean (*m*), additive (*d*), dominance (*h*), additive × additive (*i*), additive × dominance (*j*) and dominance × dominance (*l*) effects for both the crosses are presented in Table 3. Jinks and Jones (1958) model was used to explain the allelic interaction for the cross BIL-53 × IIHR-140 in the absence of nonallelic interaction. In the cross, BIL-53 × IIHR-140, additive component was significant and it is negative in direction, contributing towards resistance. Hayman (1958) approach was used to interpret the results of nonallelic interaction for the cross IIHR-19 × IIHR-140. In this cross, the additive and dominance × dominance components were found to be significant and negative in direction, contributing towards resistance. Opposite signs of h and l revealed duplicate type of epistasis. The results suggest that both additive and nonadditive components are prevailing and are equally important in this cross.

**TABLE 3** Scaling test and gene effects for watermelon bud necrosis orthotospovirus resistance in watermelon using six-parameter model

| Parameter | BIL-53 × IIHR-140 | IIHR-19 × IIHR-140 |
|---|---|---|
| *A* | 10.00 (13.99) | 16.75 (10.10) |
| *B* | 1.85 (13.44) | 19.93* (9.08) |
| *C* | 1.78 (20.01) | 10.38 (13.29) |
| *D* | −5.03 (9.45) | −13.15 (6.77) |
| *m* | 48.82* (19.29) | 68.02** (2.08) |
| *d* | −16.67** (3.82) | −12.69* (5.34) |
| *h* | 23.11 (51.96) | 9.02 (14.50) |
| *i* | 10.07 (18.91) | 26.30 (13.54) |
| *j* | 4.07 (8.30) | −1.58 (6.07) |
| *l* | 21.93 (18.91) | −62.98* (25.16) |
| Type of epistasis | Absent | Duplicate |

*Notes*. Standard errors in parenthesis. mean (*m*), additive (*d*), dominance (*h*), additive × additive (*i*), additive × dominance (*j*) and dominance × dominance (*l*)

DAS, days after sowing.

*$p < 0.05$, **$p < 0.01$.

## 4 | DISCUSSION

The results of DAC-ELISA of different samples tested using orthotospovirus antiserum and RT-PCR with WBNV nucleocapsid gene-specific primers confirmed that the symptoms observed in the experiment were due to WBNV infection. Further, the value of absorbance for DAC-ELISA of different samples varied with the type of symptoms. This variation may be due to the differences in concentration of the virus, which needs to be further confirmed. Generally, it was observed that crinkling symptoms recorded lower absorbance, compared to leaf silvering and bud necrosis. This suggests that leaf crinkling is a symptom of initial infection while leaf silvering and bud necrosis are symptoms of severe infection.

The performance of the various parental lines and their progeny showing resistance/susceptibility to WBNV suggested that it is an inherited character. The $F_1$, $F_2$, BC1 and BC2 generations in both the crosses fell within the parental range. Although the mean of $F_1$ and $F_2$ showed a fluctuation relative to each other, they were in most cases nearer to but higher than resistant parental value. The mean disease ratings of backcrosses were nearer to but higher than resistant parent in BC1 and lesser than susceptible parent in BC2. Slow disease progress, lower AUDPC (Table 1) and lower PDI (%) were observed in the generations P1, $F_1$, $F_2$ and BC1 compared to P2, BC2 and checks. Such a slow disease build-up observed in P1, $F_1$, $F_2$ and BC1 (Table 1) is practically important, which can hold up during an epidemic without being affected by disease for a longer time, thus providing longer window for adoption of other disease control strategies. Further, they can tolerate a delayed chemical spray interval and may contribute to significant reduction in chemical inputs. Such a slower disease progress was earlier observed in peanut against GBNV (Kesmala, 2003 and Kesmala et al., 2004) and TSWV (Nascimento et al., 2006), in cucumber against cucumber mosaic virus (Munshi et al., 2008) and in okra against okra yellow vein mosaic virus (Seth, Chattopadhyay, Dutta, Hazra, & Singh, 2017). Further, in the experiment it was also observed that the P1, $F_1$, $F_2$ and BC1 showed a higher plant survival and vine length compared to P2, BC2 and checks for both the crosses. Cebolla-Cornejo, Soler, Gomar, Soria, and Nuez (2003) also reported lower mortality

in resistant genotypes compared to susceptible genotypes in capsicum against TSWV. Vine length is important criteria for WBNV resistance, as apical growth is arrested due to meristem necrosis in WBNV-affected plants. A lower reduction in plant height in the resistant genotypes, compared to control plants, was observed against TSWV in peanut (Al-Saleh, Melouk, & Mulder, 2007) and against iris yellow spot orthotospovirus (IYSV) in onion (Diaz-Montano, Fuchs, Nault, & Shelton, 2010).

The estimates of mean and additive effects were significant, and additive effects were negative in both the crosses. Dominance effects were nonsignificant in both the crosses, whereas dominance × dominance component was significant in the cross IIHR-19 × IIHR-140. As observed in the current experiment, Pensuk, Wongkaew, Jogloy, and Patanothai (2002), Buiel (1996) and Pensuk, Jogloy, Wongkaew, and Patanothai (2004) also reported the occurrence of both additive gene action and nonadditive gene action for GBNV resistance in peanut.

Chi-square analysis suggested that the resistance in both crosses is governed by a major dominant gene along with background minor genes. Such major gene resistance was also reported for several other orthotospoviruses. The genes such as *Swa1*, *Swb1* (Finlay, 1953), *Sw-5* (Stevens et al., 1992), *Sw-6* (Rosello et al., 1999) and *Sw-7* (Stevens et al., 2006) in tomato and *Tsw* gene in *Capsicum* spp. (Black et al., 1996; Boiteux & de Avila, 1994; Jahn et al., 2000 & Moury et al., 1997) are dominant genes conferring resistance to TSWV. The dominant gene, *Sws*, confers resistance to melon yellow spot orthotospovirus (MYSV) in cucumber (Sugiyama et al., 2015).

Among the two resistant sources (BIL-53 and IIHR-19) used in current study, BIL-53 seems to possess higher degree of resistance with simple inheritance and hence may be of interest to breeders. Simple selection can be followed for improving the trait in the cross BIL-53 × IIHR-140 as additive gene action is prevalent. This is the first report on genetics of resistance to WBNV in watermelon. Currently, efforts are being made to map this resistance so as to enable marker-assisted selection.

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## CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

## AUTHOR CONTRIBUTIONS

ESR, PM and VB conceived and designed the experiments; NGC performed the experiments with assistance from ESR and MKR for phenotyping; RA for genetic analysis; LRDC for RT-PCR; DKS and MKR for DAC-ELISA. ESR and NGC wrote the manuscript. All the authors have discussed the results and commented on the manuscript.

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## SUPPORTING INFORMATION

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