Inheritance of Resistance to Cucurbit Powdery Mildew in Bitter Gourd

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Abstract. Cucurbit powdery mildew (CPM) caused by *Podosphaera xanthii* (Px) is an economically important disease of bitter gourd (BG; *Momordica charantia*) in Asia. High-level resistance to CPM is known in various BG accessions that have been used to develop BG breeding lines that originated in different countries. Breeding lines THMC 113 (Belize), THMC 143 (India), THMC 153 (Thailand), THMC 167 (India), and THMC 170 (Taiwan) possess high-level resistance to BG Px race (BG-CPM), designated Mc-1 from a field at Kamphaeng Saen, Thailand, whereas THMC 144 (India) is susceptible. Our objective was to determine the inheritance of resistance to BG-CPM race Mc-1. To that end, THMC 144 (India) was crossed with the five resistant lines. The parents and their respective F₁, F₂, backcross progenies were evaluated for BG-CPM disease severity in inoculated field and growth chamber tests. Resistance to BG-CPM race Mc-1 in the five resistant lines was controlled by at least two independent, recessive genes. Intercrosses of the BG-CPM–resistant lines revealed allelic resistances in four of the breeding lines: THMC 113, THMC 153, THMC 167, and THMC 170. Resistance in THMC 143 was clearly non-allelic for resistance to BG-CPM with the other four BG-CPM–resistant lines.

CPM, caused by *Px*, has become one of the most serious diseases of field and greenhouse cucurbits worldwide (Kristóková et al., 2009; McGrath, 2017; Sitterly, 1978). It is a serious fungal disease of BG (*Momordica charantia*), which is an economically and nutritionally important crop that is mainly cultivated by smallholder farmers in Asia, where it is grown on ~340,000 ha (Arvind Kapur, Ascen HyVeg Private Limited, personal communication). Powdery mildew infection of BG can lead to diminished plant canopy, early foliage loss, poor fruit quality, and consequently reduction in yield. Yield losses of up to 50% have been observed in China and India (J. Fu, Enza Zaden, and V. Chawda, VNR Seeds Private Limited, personal communication). Powdery mildew control often is, however, difficult and tedious because the spray must cover the underside of the leaves, unless systemic fungicides are used. Increased production costs, environmental pollution, and development of fungicide-insensitive mildew races are the major concerns for chemical control of CPM (McGrath, 2001). Adequate disease control often is, however, difficult and tedious because the spray must cover the underside of the leaves, unless systemic fungicides are used. Increased production costs, environmental pollution, and development of fungicide-insensitive mildew races are the major concerns for chemical control of CPM (Lebeda et al., 2010; McGrath et al., 1996). The most cost-effective, environmentally friendly, and sustainable management of CPM can be achieved through genetic host plant resistance.

One hundred fifty BG accessions of the World Vegetable Center Genebank were screened for resistance to a local isolate of *Px* at Kamphaeng Saen, Thailand. A single resistant plant was identified in each of five heterogeneous populations derived from five of the accessions that originated from India, Thailand, Taiwan, and Belize. Multiple cycles of inbreeding and selection led to the development of five Px-resistant inbred lines: THMC 113, THMC 143, THMC 153, THMC 167, and THMC 170. These lines were uniformly resistant to the local isolate of *Px* in Kamphaeng Saen, Thailand, in field tests over three seasons (Dhillon et al., 2018). The objective of this study was to understand the mode of inheritance of resistance to CPM in the five inbred lines.

**Materials and Methods**

Field trials and inoculations. The BG breeding lines used in this study included the BG-CPM–susceptible line THMC 144 and five BG-CPM–resistant lines: THMC 113, THMC 143, THMC 153, THMC 167, and THMC 170. The resistant lines were used as pollinators in crosses with THMC 144 to produce the F₁ generation for each inbred line, from which the respective F₂, and reciprocal backcrosses were produced. The resistant lines were intercrossed and their F₁ progenies tested to determine allelic relationships of their resistance genes.

BG-CPM incited by *Px* is observed annually in the BG fields at the World Vegetable Center Research and Training Station, East and Southeast Asia, Kamphaeng Saen, Thailand, during November to January. The susceptible check variety THMC 144 exhibits 100% susceptibility when planted in the field in this period, and the five other lines are resistant, indicating that this strain belongs to race Mc-1 (Dhillon et al., 2018). Twenty plants of each parent and each F₁ generation, 100 plants of each backcross generation, and 200 plants of each F₂ generation were evaluated for resistance to BG-CPM (no replicates). Seeds were sown and plants transplanted 13 Nov. and 23 Nov. 2016, respectively. Plants were arranged in single rows on raised beds with a row spacing of 1.6-m and 1-m within-row spacing, and staked. BG-CPM–susceptible THMC 144 was transplanted on the boundary of the test field 2 weeks before establishment of the genetic study to increase the BG-CPM inoculum level. Additional spreader rows of THMC 144 were transplanted around the field at the time the genetic study was transplanted. Seedlings were inoculated with inoculum collected from the leaves of BG-CPM–susceptible THMC 144 plants 4 days before transplanting at the three to four true leaf stage and at 20 days posttransplanting. Each plant was inoculated to runoff, using a 1-L hand sprayer, with a spore suspension of *Px* prepared as follows. Freshly prepared spores were washed from detached, heavily infected leaves, unless systemic fungicides are used. Increased production costs, environmental pollution, and development of fungicide-insensitive mildew races are the major concerns for chemical control of CPM (Lebeda et al., 2010; McGrath et al., 1996). The most cost-effective, environmentally friendly, and sustainable management of CPM can be achieved through genetic host plant resistance.

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leaves with a spray of 100 mL of water and filtered through a double layer of cheesecloth and diluted to a concentration of 4 × 10^4 conidia/mL, as determined by a hemocytometer. Disease severity 30 days’ postinoculation was rated on the first five true leaves of individual 50-day-old plants using a 0 to 5 visual rating scale, where 0 = free from fungal infection; and 1 = 1% to 10%, 2 = 11% to 25%, 3 = 26% to 50%, 4 = 51% to 75%, and 5 = >75% leaf surface covered with BG-CPM. BG-CPM–susceptible THMC 144 was uniformly rated 5 at that time.

Growth chamber tests. THMC 144, THMC 113, THMC 143, THMC 153, and THMC 167, and their respective F₁, F₂, and backcross generations to THMC 144 were included in this test; numbers of plants in the F₂ populations ranged from 84 to 120 (Table 1). Seeds were sown and plants were raised until the second-leaf stage in a glasshouse, at which time they were inoculated as described previously, spraying until runoff with a spore suspension in water at a concentration of 5 × 10^4 conidia/mL. A single-spore CPM strain isolated from M. charantia was grown in an open field in Kamphaeng Saen, Thailand, was used as source of inoculum; it was identified as race Mc-1 (Dhillon et al., 2018). A single spore was taken under a binocular with an eye lens and deposited on a Lagenaria (bottle gourd) cotyledon maintained in axenic conditions. This operation was conducted twice. The single-spore isolate was then maintained and multiplied in vitro on Lagenaria cotyledons for long-term storage in liquid nitrogen or in a freezer at −80 °C. The inoculated plants were incubated after inoculation in a growth chamber (16-h day 26 °C/8-h night 20 °C). Disease severity at 14 days postinoculation was rated on the second leaf using a 0 to 3 visual rating scale, where 0 = no visible symptoms, 1 = chlorotic local lesions with very light sporulation observed upon close examination, 2 = clear sporulation but not abundant, and 3 = abundant sporulation corresponding to susceptible control.

Greenhouse test. Allelic relationships for resistance to BG-CPM race Mc-1 were investigated in a single greenhouse test. The five BG-CPM-resistant lines were intercrossed in a half diallel. Their F₁ progenies (10 plants of each progeny) were transplanted into pots (10 × 15 cm) and inoculated at the 10-leaf stage, as described previously for field tests. Disease severity at 14 days postinoculation was rated using the 0 to 5 visual scale described for field test.

For both field and growth chamber tests, χ² was used to test goodness-of-fit of observed to expected ratios using SAS-STAT statistical package (SAS Institute, Cary, NC).

Results

Inheritance study. THMC 113, THMC 143, THMC 153, THMC 167, and THMC 170 were uniformly resistant to infection, and, as expected, THMC 144 was uniformly observed highly susceptible with disease ratings (DR) of 5 and 3 in the field and growth chamber tests, respectively (Table 1). The five F₁ progenies from crosses of susceptible THMC 144 with the resistant lines also were uniformly rated 5 for disease severity in the field test. The F₁ progenies with THMC 113, THMC 143, and THMC 167 were susceptible (ratings of 2 and 3) in the growth chamber with symptoms typical of CPM, whereas five plants of the F₁ THMC

| Table 1. Cucurbit powdery mildew disease reactions of five resistant and one susceptible (THMC 144) bitter gourd breeding lines and their respective F₁, F₂, backcross progenies from crosses of the susceptible THMC 144 with the five resistant lines in field (0–5 rating scale) and growth chamber (0–3 rating scale) tests. |
|---|---|---|---|---|---|---|---|---|---|---|
| THMC line or progeny | Field² | | | | | | | | | |
| | 0 | 1 | 2 | 3 | 4 | 5 | 0 | 1 | 2 | 3 |
| Parents | | | | | | | | | | |
| 113 | 20 | 0 | 0 | 0 | 0 | 0 | 9 | 0 | 0 | 0 |
| 143 | 20 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 26 |
| 144 | 0 | 0 | 0 | 0 | 20 | 0 | 0 | 0 | 0 | 0 |
| 153 | 20 | 0 | 0 | 0 | 0 | 0 | 8 | 0 | 0 | 0 |
| 167 | 20 | 0 | 0 | 0 | 0 | 0 | 11 | 0 | 0 | 0 |
| 170 | 20 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Crosses | | | | | | | | | | |
| 144 × 113 | | | | | | | | | | |
| F₁ | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 0 | 0 | 0 |
| F₂ | 29 | 24 | 21 | 71 | 47 | 8 | 23 | 24 | 25 | 48 |
| BC₁₃ | 7 | 33 | 28 | 21 | 9 | 2 | 25 | 0 | 13 | 46 |
| BC₁₄ | 0 | 0 | 0 | 0 | 0 | 100 | 0 | 0 | 0 | 0 |
| 144 × 143 | | | | | | | | | | |
| F₁ | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 0 | 0 | 15 |
| F₂ | 0 | 19 | 23 | 102 | 55 | 1 | 25 | 0 | 13 | 46 |
| BC₁₃ | 0 | 8 | 29 | 63 | 0 | 0 | 100 | 0 | 0 | 0 |
| BC₁₄ | 0 | 0 | 0 | 0 | 0 | 100 | 0 | 0 | 0 | 0 |
| 144 × 153 | | | | | | | | | | |
| F₁ | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 5 | 0 | 9 |
| F₂ | 0 | 29 | 10 | 98 | 63 | 0 | 34 | 29 | 0 | 23 |
| BC₁₃ | 0 | 19 | 22 | 54 | 5 | 0 | 6 | 5 | 0 | 23 |
| BC₁₄ | 0 | 0 | 0 | 0 | 0 | 100 | 0 | 0 | 0 | 0 |
| 144 × 167 | | | | | | | | | | |
| F₁ | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 0 | 0 | 10 |
| F₂ | 0 | 13 | 42 | 80 | 64 | 1 | 24 | 0 | 29 | 46 |
| BC₁₃ | 0 | 46 | 6 | 41 | 7 | 0 | 0 | 0 | 17 | 25 |
| BC₁₄ | 0 | 0 | 0 | 0 | 0 | 100 | 0 | 0 | 0 | 0 |
| 144 × 170 | | | | | | | | | | |
| F₁ | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 0 | 0 | 0 |
| F₂ | 0 | 5 | 36 | 109 | 48 | 2 | 0 | 0 | 0 | 0 |
| BC₁₃ | 0 | 23 | 21 | 43 | 13 | 0 | 100 | 0 | 0 | 0 |
| BC₁₄ | 0 | 0 | 0 | 0 | 0 | 100 | 0 | 0 | 0 | 0 |

²Inoculated to runoff with an aqueous spore suspension (4 × 10^4 conidia/mL) before transplanting at the three to four true leaf stage and at 20 days’ posttransplanting. Disease severity rated on the leaves of individual 50-day-old plants using a 0–5 visual rating scale, where 0 = no visible infection, 1 = 1% to 10%, 2 = 11% to 25%, 3 = 26% to 50%, 4 = 51% to 75%, and 5 = >75% leaf surface covered with CPM.

³Inoculated to runoff with an aqueous spore suspension (5 × 10^4 conidia/mL) at the two-leaf stage. Disease severity rated at the second leaf stage using a 0–3 visual rating scale, where 0 = no visible symptoms, 1 = very light sporulation, 2 = clear sporulation but not abundant, and 3 = abundant sporulation corresponding to susceptible control.
144 × THMC 153 were rated 1 and nine plants were rated 3 (Table 1). These data clearly indicate recessive inheritance of this trait in the five resistant lines.

The F2 THMC 144 × THMC 113 segregate in the field test in an acceptable fit to a 3 resistant (DR = 0): 13 susceptible (DR = 1 to 5) ($\chi^2 = 2.37, P = 0.13$), which suggests two recessive genes with epistasis (Table 1). This model was not confirmed by the backcross of the F1 to the resistant parent when considering the class DR = 0 as resistant and the DR = 1 to 5 classes as susceptible. When DR classes 0 and 1 are, however, combined, the segregations of the two generations support a single recessive gene model, where the F2 segregated 53 resistant: 147 susceptible ($\chi^2 = 0.24, P = 0.63$), and the backcross of the F1 to the resistant parent segregated 40 resistant: 60 susceptible ($\chi^2 = 4.0, P = 0.046$). The single recessive gene model was supported by growth chamber results (Table 1), where the F2 THMC 144 × THMC 113 segregated in an acceptable fit to a 1 resistant (DR = 0): 3 susceptible (DR = 1 to 3) ($\chi^2 = 2.178, P = 0.14$). This single recessive model is, however, challenged by the conflict in classifying plants in the field test with as much as 10% infected foliage as resistant, and then applying a greater threshold in the growth chamber tests where plants with very light sporulation are classified susceptible.

In contrast to the THMC 144 × THMC 113 cross, there were no resistant (DR = 0) segregants in any of the other F2 progenies in the field test, which suggests additional recessive, perhaps epistatic, loci for resistance to BG-CPM race Mc-1; combining DR classes 0 and 1 did not produce acceptable single or digenic segregations. Their respective backcross progenies to THMC 144 (susceptible) were highly susceptible. There were no resistant (DR = 0) segregants in the respective backcrosses to the four resistant parents, although when DR classes 0 and 1 were combined, two (THMC 153 and THMC 170) segregated in acceptable fits to 1 resistant: 3 susceptible expected in a digenic model: $\chi^2$ values (and probabilities) were, respectively, 1.92 (0.17) and 0.21 (0.66).

In the growth chamber tests, ≈25% of the plants in F2 progenies with THMC 143 and THMC 167 were resistant; $\chi^2$ values (and probabilities) were, respectively, 1.016 (0.31), and 0.03 (0.86). Only two backcrosses (F1 × THMC 144) were included in the growth chamber tests; they were both susceptible. The growth chamber data suggest single recessive genes for resistance to BG-CPM race Mc-1 in THMC 113, THMC 143, and THMC 167. In contrast, THMC 153 produced distinctive results in the growth chamber. Some individuals in the F1 and F2 generations exhibited very light symptoms with chlorotic lesions (DR = 1). The F1 was not as susceptible as those with the other breeding lines, and the F2 segregated a greater number of resistant (growth chamber DR = 0) plants than acceptable in a single recessive gene model ($\chi^2 = 9.69, P = 0.002$). In the BC with THMC 144, six plants were observed without symptoms and five with chlorotic lesions. Resistance in accession THMC 153 seems to be controlled by gene(s) with intermediate (not fully recessive) resistance.

Allelic relationships. All offspring from crosses among four inbred lines THMC 113, THMC 153, THMC 167, and THMC 170 were resistant, which suggests that the same locus is involved in the resistance of these four BG inbred lines against BG-CPM race Mc-1 (Table 2). In contrast, the F1 progenies between THMC 143 and the four other resistant lines were fully susceptible, indicating a different major locus for resistance in this accession.

Discussion

BG is an important cash crop and its current seed market in Asia is worth ≈16 million Euros (Dhillon et al., 2016). It is mainly cultivated by smallholder farmers in Asia, where CPM species is a major cucurbit production constraint in the field as well as glasshouse production systems (Lebeda et al., 2010); the adoption of CPM-resistant BG cultivars will enhance the profitability of the farmers while reducing numbers and frequency of chemical control and potential adverse environmental effects. The five BG-CPM-resistant breeding lines used in this study have been observed resistant to many local isolates of $P_{x}$ during multilocation testing in Thailand, Vietnam, Philippines, Myanmar, China, India, and Bangladesh (Dhillon et al., 2018).

Race-specific resistance has been used to breed CPM-resistant cultivars of cucurbit species such as muskmelon, Cucumis melo L. (Dhillon et al., 2012), watermelon, Citrullus lanatus (Thunb.) Matsum. & Nakai (Ben-Naim and Cohen, 2015), squash and pumpkin, Cucurbita sp. (Jahn et al., 2002). There are currently no available cultivars of BG resistant to CPM.

CPM species $P_{x}$ has a high evolutionary potential (McDonald and Linde, 2002) and is, therefore, likely to overcome such kind of race-specific resistance. This has been borne out in melon, for which the number of races has increased to such a large number that an international powdery race initiative was proposed based on a triple-septet set of melon cultivars, breeding lines, and accessions to objectively and systematically designate melon CPM races (Lebeda et al., 2016).

The discrepancy in reactions of the F2 progenies between the field and growth chamber tests was likely due to several factors: 1) the use of a single-spore–derived strain in the growth chamber vs. an unknown mixture of strains in the field; 2) plant age, i.e., young plants in the growth chamber vs. older plants in the field; 3) more severe infection in the field due to multiple inoculum sources and cycles of multiplication of CPM over the 30-day period of disease development in the field tests vs. 14-day period of disease development following a single inoculation in the growth chamber test; and 4) differences in the disease reaction rating scales. The scale used for field evaluations is proportional to the surface of the leaf covered with powdery mildew, which resulted from several cycles of multiplication and infection by powdery mildew. The severity scale used for the growth chamber tests is proportional to the intensity of the sporulation because there is only one cycle of

Table 2. Allelic relationships between five bitter gourd lines carrying recessive genes for resistance against $P_{x}$. Resistant lines were intercrossed; 10 plants of each $F_{1}$ were evaluated for disease reaction to CPM; artificial inoculation test in a greenhouse.

| $F_{1}$ progeny | Disease reaction (number of plants) $^{a}$ | 0 | 1 | 2 | 3 | 4 | 5 |
|----------------|---------------------------------------------|---|---|---|---|---|---|
| THMC 113 × THMC 143 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 113 × THMC 153 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 113 × THMC 167 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 113 × THMC 170 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 143 × THMC 153 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 143 × THMC 167 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 143 × THMC 170 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 153 × THMC 167 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 153 × THMC 170 | 10 | 0 | 10 | 0 | 0 | 0 |
| THMC 167 × THMC 170 | 10 | 0 | 10 | 0 | 0 | 0 |

*Inoculated to runoff with an aqueous spore suspension (4 × 10$^{4}$ conidia/mL) at the 10-leaf stage. Disease severity rated on the leaves of individual 50-day-old plants using a 0–5 visual rating scale, where 0 = no visible infection; 1 = 1% to 10%; 2 = 11% to 25%; 3 = 26% to 50%; 4 = 51% to 75%, and 5 = ≥75% leaf surface covered with CPM.
infection/multiplication, and thus the number of mildew colonies is proportional to the concentration of the inoculum. Nevertheless, the main conclusions are that resistance to BG-CPM in these five breeding lines is recessive and that at least two loci are involved.

Four BG-CPM races are known. This study was conducted with race Mc-1, to which all five breeding lines are resistant (Dhillon et al., 2018). THMC 153 and THMC 167 were resistant to all four races (Mc-1, Mc-2, Mc-3, Mc-4) in that study, whereas THMC 113 and THMC 143 were susceptible to one race (Mc-3 and Mc-4, respectively), and THMC 170 was susceptible to two races (Mc-2 and Mc-3). BG-CPM races Mc-2 and Mc-4 were each compatible on one line, THMC 170 and THMC 143, respectively, and Mc-3 was compatible on two lines, THMC 113 and THMC 170. We found two independent, recessive genes for resistance to race Mc-1 in this study. Inheritance of resistance to the other three races remains to be determined; such studies will reveal new genes, perhaps some dominant, for resistance to these four races and give us a complete characterization of the of BG-CPM resistance gene combinations in these breeding lines. This information will inform efforts to combine BG-CPM resistance genes in the next generation of BG breeding lines and cultivars that will potentially be more stable in terms of race durability. Knowledge of BG-CPM races is relatively new, and it remains to be determined whether BG–Px CPM interactions will be similar to the experience in melon, with many races, watermelon with two races, or be more like cucumber and melon, with many races, watermelon with the main conclusions are that resistance to powdery mildew is of multiple virus resistance, p. 167–195. In: M.M. Kyle (ed.). Resistance to viral diseases of vegetables. Timber Press, Portland, OR.

Sitterly, W.R. 1978. Powdery mildews of cucurbits, p. 359–379. In: D.M. Spencer (ed.). The powdery mildews. Academic Press, New York. Van Der Plank, J.E. 1968. Disease resistance in plants. Academic Press, New York.

Literature Cited
Ben-Naim, Y. and Y. Cohen. 2015. Inheritance of resistance to powdery mildew race 1W in watermelon. Phytopathology 105:1446–1457.
Dhillon, N.P.S., A.J. Monforte, M. Pitrat, S. Pandey, P.K. Singh, K.R. Reitsma, J. Garcia-Mas, A. Sharma, and J.D. McCreight. 2012. Melon landraces of India: Contributions and importance. Plant Breed. Rev. 35:85–150.
Dhillon, N.P.S., S. Sanguansil, S. Schafflter, Y.-W. Wang, and J.D. McCreight. 2016. Diversity among a wide Asian collection of bitter gourd landraces and their genetic relationships with commercial hybrid cultivars. J. Amer. Soc. Hort. Sci. 141:475–484.
Dhillon, N.P.S., S. Sanguansil, S. Srimat, B. Manjunath, P. Agarwal, Q. Xiang, M.A.T. Masud, T. Myint, H.T. Hanh, T.K. Cuong, C.H. Balatero, V. Salutan-Bautista, M. Pitrat, A. Lebeda, and J.D. McCreight. 2018. Cucurbit powdery mildew-resistant bitter gourd breeding lines reveal four races of Podosphaera xanthii in Asia. HortScience 53:337–341.
Jahn, M., H.M. Munger, and J.D. McCreight. 2002. Breeding cucurbit crops for powdery mildew resistance, p. 239–248. In: R.R. Belanger W.R. Bushnell, A.J. Dik, and T.L.W. Carver (eds.). The powdery mildews: A comprehensive treatment. APS Press, St. Paul, MN.
Kristková, E., A. Lebeda, and B. Sedlíková. 2009. Species spectra, distribution and host range of cucurbit powdery mildews in the Czech Republic, and in some other European and Middle Eastern countries. Phytoparasitica 37:337–350.
Lebeda, A., E. Kristková, B. Sedlíková, J.D. McCreight, and M.D. Coffey. 2016. Cucurbit powdery mildews: Methodology for objective determination and denomination of races. Eur. J. Plant Pathol. 144:399–410.
Lebeda, A., M.T. McGrath, and B. Sedlíková. 2010. Fungicide resistance in cucumber powdery mildew fungi, p. 221–246. In: O. Carisse (ed.). Fungicides. InTech Publishers, Rijeka, Croatia.
McDonald, B.A. and C. Linde. 2002. Pathogen population genetics, evolutionary potential, and durable resistance. Annu. Rev. Phytopathol. 40:349–379.
McGrath, M.T. 2001. Fungicide resistance in cucurbit powdery mildew: Experiences and challenges. Plant Dis. 85:236–245.
McGrath, M.T. 2017. Powdery mildew, p. 62–64. In: A.P. Keinath, W.M. Wintemantel, and T.A. Zitter (eds.). Compendium of cucurbit diseases and insect pests. 2nd ed. APS Press, St. Paul, MN.
McGrath, M.T., H. Stanisiewska, N. Shishkoff, and G. Casella. 1996. Fungicide sensitivity of Sphaerotheca fuliginea populations in the United States. Plant Dis. 80:697–703.
Scully, B.T. and W.T. Federer. 1993. Application of genetic theory in breeding for multiple virus resistance, p. 167–195. In: M.M. Kyle (ed.). Resistance to viral diseases of vegetables. Timber Press, Portland, OR.