Cucurbit Powdery Mildew Race Identification by Triplet-Septet and Disease Progress Estimation[1]

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ABSTRACT

The Triplet-Septet (TS) set of melon cucurbit powdery mildew (CPM) race differentials (CPMRD) was established to provide an international means for objective and uniform identification and designation on CPM races. The Area Under Disease Progress Stairs (AUDPS) method for disease progress estimation was derived from the Area Under Disease Progress Curve (AUDPC) method, and both have been used to evaluate disease progress on other crops. We aimed to identify a melon CPM race on the TS melon CPMRD, and estimate disease progress thereon using AUDPC and AUDPS. Plants were inoculated at the 3 to 4 true leaf stage. Severity of CPM infection was evaluated on the 21 TS melon CPMRD at 15, 22, 32, and 41 days after inoculation (DAI) using a visual scale. The CPM population in the greenhouse was identified as race S based on reactions of a set of 11 commonly used melon CPMRD, and it may also be designated as 127.127.126 on the TS melon CPMRD. AUDPS identified higher levels of disease than AUDPC, and its results agreed with those obtained by the commonly used melon CPMRD conventional race identification methods (current and Triplet-Septet). AUDPS can be used to evaluate the disease progress on CPM.

Keywords: AUDPC; AUDPS; *Cucumis melo*; genetic resistance; *Podosphaera xanthii.*

INTRODUCTION

Powdery mildew is one of the most prevalent and severe diseases of cucurbits. The obligate biotrophic fungal species *Podosphaera xanthii* [syn. *Sphaerotheca fuliginea* (Schlecht) Pollacci, syn *P. fusca*] and *Golovinomyces cichoracearum* (DC) V.P. Heluta *(*syn. *Erysiphe cichoracearum* DC. Ex Mérat) incite cucurbit powdery mildew (CPM), and are common worldwide. *G. cichoracearum* is more frequently observed in Europe and regions with temperate climate, whereas *P. xanthii* is found mainly in the Americas and tropical areas (Kuzuya *et al.*, 2006; Pérez-García *et al.*, 2009).

The fungus predominantly colonizes leaf surfaces and may occasionally infect stems and fruits. Infection can decrease photosynthetic area by early loss of leaves, resulting in low production of photoassimilates. Consequently, infected plants produce fewer and smaller fruits, with low commercial quality, especially lower soluble solids levels, and sun burned rinds (Viana *et al.*, 2001).

Control of CPM has been done by using chemical products and resistant cultivars. The pathogen has, however, developed resistance to most fungicides used for control (McGrath, 2001; 2006). Cultivars with genetic resistance

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are advantageous, because resistance is equally distributed throughout all plants in a field, and its use may decrease, or even exclude, the application of fungicides. Resistance to CPM was first available in western United States shipper type cantaloupe melon in the 1930s with the development and release of 'PMR 45' (Pryor *et al.*, 1946).

The effectiveness of genetic resistance is decreased by the occurrence of pathogen races. Numerous (> 46) *P. xanthii* races on melon have been identified, and approximately 36 sources of resistance are known in melon (McCreight, 2006; McCreight *et al.*, 2012). The development of new cultivars resistant to all races is impractical. Thus, breeding programs seek to provide resistance for regional needs, regarding the races with higher likelihood of occurrence, and that requires the constant monitoring of races.

There has been confusion about powdery mildew race identification due to different methods of evaluation and sets of melon race differentials, and nomenclature. A new objective and uniform system of pathogenic race identification and denomination has been proposed based on a uniform set of race differentials, a uniform code for scoring disease reactions, and a uniform screening methodology to classify CPM races based upon a three-part, numerical code, named Triplet-Septet (Lebeda *et al.*, 2016). This proposal established a specific set of 21 race differentials, divided into three subgroups of seven that when challenged with any isolate will generate a three-part score or code that will range from 0.0.0 (all resistant) to 127.127.127 (all susceptible), and the method is mathematically able to identify 2,097,152 races (Limpert & Müller, 1994).

Stadnik *et al.* (2001) pointed out the lack of methods that suitably measure powdery mildew in quantitative ways. Van der Plank (1963) proposed a method that allows the progressive evaluation of disease development, Area Under Disease Progress Curve (AUDPC). Simko & Piepho (2012) proposed the Area Under Disease Progress Stairs (AUDPS) to better estimate the first and last evaluations of disease severity, as compared with AUDPC.

We confirmed the pathogenic race identification of the CPM population resident in a greenhouse at Salinas using whole plants and characterizing it on melon using the alphanumeric nomenclature that developed over time and the Triplet-Septet method. Disease reactions of the melon CPM race differentials were also characterized using two methods for estimating disease progress and their respective standardized estimators.

MATERIALS AND METHODS

The experiment was carried out at U. S. Department of Agriculture (USDA), Crop Improvement and Plant Protection Research Unit, Salinas, CA, from August to October 2016. We used the triple septet melon race differentials suggested by Lebeda *et al.* (2016) (Table 1).

Seeds of the 21 Cucurbit Powdery Mildew Race Differentials (CPMRD) were germinated on moistened paper towels in plastic boxes at 25 ºC and a 12-h photoperiod, in a CPM-free growth chamber (Conviron® model 6050). Seedlings were transplanted at the cotyledon stage to 0.5 L plastic pots filled with potting mix (Sun Land™, Watsonville, CA) and grown under the same conditions as described above. Due to poor germination of 'Amarillo' seeds, only a single plant of this differential was used in the test.

Seedlings were transferred 16-days after transplanting to a greenhouse naturally infested by CPM and arranged in three randomized blocks with one plant of each CPMRD per rep. Plants were watered daily with dilute (1:100) 20N–20P–20K fertilizer solution applied via drip irrigation.

Identity of *P. xanthii* was confirmed by observing conidia with fibrosin bodies, with the aid of a light microscope. The resident CPM population was previously confirmed to be *P. xanthii* based on morphological and molecular characteristics (Bojorques Ramos *et al.*, 2011). Inoculum was subsequently collected from infected melon plants in the same greenhouse. The 3rd or 4th true leaf of each test plant was inoculated with fragments of mycelia immediately after transplanting with the aid of an artist brush.

Inoculated true leaves were evaluated at 15, 22, 32, and 41 days after inoculation (DAI). Levels of infection were assessed using a 1 to 9 visual scale as follows: 1 = no evidence of disease; 2 = trace of hyphae, no detectable sporulation; 3 = hyphae restricted, no detectable sporulation; 4 = few colonies present, sporulation; 5 = scattered colonies, sporulation; 6 = numerous colonies, sporulation; 7 = ≈ 50% of adaxial surface covered with hyphae and spores, few colonies on abaxial surface, abundant sporulation; 8 = 50-75% of adaxial surface covered with hyphae and spores, colonies on the abaxial surface, abundant sporulation; and 9 = > 75% of adaxial surface covered with hyphae and spores, numerous or coalesced colonies on abaxial surface.

Powdery mildew race identity was determined according to the reactions of the 21 differentials 41 DAI. Susceptibility was attributed to those with means ≥ 4.0 and

means < 4.0 indicated resistance, and race nomenclature was performed using both alphanumeric (McCreight *et al.*, 2012) and Triplet-Septet methods, the latter as proposed by Lebeda *et al.* (2016) but, with modifications, given the fact that the authors suggested that race identification essays should be done using excised leaf discs under axenic conditions. Each member within a triplet is assigned a unique weight that becomes its score when susceptible; when resistant it is scored zero. The total score for a triplet reveals at a glance which differentials were susceptible and which were resistant.

Disease progress was estimated using AUDPC (Van der Plank, 1963) and AUDPS (Simko & Piepho, 2012) methods. Standardized estimates for both methods were calculated (sAUDPC and sAUDPS), as stipulated by Simko and Piepho (2012). Simulated disease progress estimates were generated for each level of the 1-9 disease severity scale at the same intervals the test data were recorded in order to compare AUPDC and AUPDS estimates, and identify susceptible and resistant differentials via these disease progress estimates. Differentials with mean AUDPC, AUDPS, sAUDPC, and sAUDPS estimates lower than those obtained for the disease rating scale score 4 simulations were, thus, considered resistant.

Data were subjected to Analysis of Variance and means comparisons by Tukey HSD test. AUDPC and AUDPS, sAUDPC and sAUDPS analyses were compared by Student's *t*-test. All analyses were performed with SAS 9.4 University Edition. Data from 'Amarillo' were not included in the statistical analyses, as there was only a single plant of this differential. However, its reaction was used to identify the powdery mildew race using the alphanumeric and Triplet-Septet methods.

RESULTS AND DISCUSSION

Reactions of CPM race differentials

Infection was uniform, as represented by the low coefficient of variation for disease reaction (Table 1). *P. xanthii* colonies developed on 20 of the 21 differentials, and mean disease reaction scores ranged from 1.0 (PI 313970) to 9.0. This reaction pattern was consistent with race S. The first reports of race S were from commercial fields and experimental plots in Yuma, AZ, and Holtville, CA in 2003, when all commercial cultivars and all the commonly used melon CPMRD were susceptible; only PI 313970 was resistant (McCreight *et al.*, 2005; McCreight & Coffey, 2011).

Disease severity differed significantly among the CPMRD. On the most infected differentials, leaves were fully covered by the fungus, with intense sporulation, and mycelia were observed on stems as well. *P. xanthii* mycelia were found after the test was terminated on fruits of Ames 31282, a kind of infection not frequently reported. This accession is resistant to race *pxCh1* that was reported in China (Liu *et al.*, 2010)2010; its susceptibility to race S was previously reported (McCreight *et al.*, 2012).

Intermediate levels of infection were found on MR-1, PI 124111, and PI 124112; the latter two had the same mean disease reaction values and were statistically greater than MR-1 (Table 1). It is interesting to note that MR-1 was selected from PI 124111 for uniform reaction to downy mildew (*Pseudoperonospora cubensis* (Berk. And Curt.) Rostow) and *P. xanthii* races 1, 2, and 3 (Thomas, 1986). The lower means of these genotypes were due in part, perhaps, to the presence of blisters on their leaves, evidence of a foliar response that is a non-race-specific form of resistance (Sedlářová *et al.*, 2009) first recognized in hops (Salmon, 1917; Salmon, 1919). Blisters have the appearance of water-soaked and raised lesion spots, sometimes chlorotic or necrotic at the center, that restricts or blocks the pathogen development on the foliar surface (McCreight, 2003; McCreight & Coffey, 2011). Nevertheless, mycelial growth was consistently observed on leaves from secondary stems of MR-1, and on both primary and secondary stems of PI 124111 and PI 124112.

PI 313970 was the only resistant differential; it exhibited blister-like resistance, and it was statistically different from the other differentials. Resistance to race S is controlled by a single, recessive gene (McCreight & Coffey, 2011). Although rated resistant for foliar reaction, *P. xanthii* colonies were observed on stems of this differential; yet this kind of infection is not considered by the severity scales so far. This discrepancy between the foliar and stem reactions was previously observed on some plants of the same genotype by McCreight & Coffey (2011), who attributed it to a possible heterogeneity of virulence factors in powdery mildew populations. PI 313970 was susceptible to race F in Czech Republic (Lebeda & Sedláková, 2004; Sedlářová *et al.*, 2009), as well as races SD and SDW that were isolated in Yuma, AZ and Imperial Valley (Coffey *et al.*, 2006).

Application of the Triplet-Septet method to this data set generated the Triplet-Septet code for race S: 127.127.126 (Table 1). The totals for the first two triplets indicate all members of the two triplets were susceptible. The score of the third triplet indicates the first member (weight = 1) was

resistant and the others susceptible.

Almost all (20/21) melon powdery mildew race differentials are susceptible to race 127.127.126. Thus, it is expected that cultivars developed using any of the susceptible differentials as sources of CPM resistance will be susceptible to this race as well. That information may help the choice of cultivars to be eventually cultivated in areas affected by powdery mildew.

Table 1: Reactions of 21 melon cucurbit powdery mildew race differentials in the melon Triplet-Septet (Lebeda *et al.*, 2016) to an isolate of *Podosphaera xanthii* in a greenhouse 41 days after inoculation, their summary disease reactions, and Triplet-Septet groups, weights and scores

Differential	Mean disease reaction[z]	Summary reaction[y]	Triplet-Septet		
			Group	Weight	Score
Iran H	9.0 a	S	1.1	1	1
Védrantais	9.0 a	S	1.2	2	2
PI 179901	9.0 a	S	1.3	4	4
PI 234607	9.0 a	S	1.4	8	8
AR HBJ	9.0 a	S	1.5	16	16
PMR 45	9.0 a	S	1.6	32	32
PMR 6	8.7 a	S	1.7	64	64
Septet total					127
WMR 29	9.0 a	S	2.1	1	1
Edisto 47	8.7 ab	S	2.2	2	2
PI 414723	9.0 a	S	2.3	4	4
PMR 5	9.0 a	S	2.4	8	8
PI 124112	7.0 b	S	2.5	16	16
MR-1	5.3 c	S	2.6	32	32
PI 124111	7.0 b	S	2.7	64	64
Septet total					127
PI 313970	1.0 d	R	3.1	1	0
Noy Yzre'el	9.0 a	S	3.2	2	2
PI 236355	9.0 a	S	3.3	4	4
Negro	8.3 ab	S	3.4	8	8
Amarillo[x]	9.0	S	3.5	16	16
Nantais Oblong	9.0 a	S	3.6	32	32
Ames 31282	9.0 a	S	3.7	64	64
Septet total					126
F test	40.83**				
Mean	8.13				
CV (%)	6.16				
Alphanumeric race nomenclature: S[w]					
Triplet-Septet code					127.127.126

[z]Means followed by the same letter do not differ significantly at $P < 0.05$ by Tukey HSD test.
[y]R = resistant, S = susceptible; **Significant at $P < 0.01$ by *F* test.
[x]Reaction based on a single plant, not included in analysis of variance.
[w]McCreight and Coffey, 2011.

The Triplet-Septet method was based on a similar approach adopted for downy mildew (*Bremia lactucae*) races on lettuce, proposed by Van Ettekoven & Van Arend (1999). This method is recommended by the International Bremia Evaluation Board (IBEB) (ISF, 2016), and internationally adopted by lettuce seed companies.

The Triplet-Septet is a recently proposed method and being slowly adopted. Lebeda *et al.* (2012) monitored the occurrence of powdery mildew races on *Cucurbita maxima*, *Cucurbita moschata*, *Cucurbita pepo*, and *Cucumis melo* in Czech Republic. Five *G. cichoracearum* (51.15.103, 55.63.119, 51.31.103, and 54.15.113), and 12 *P. xanthii* races (55.78.124, 23.0.124, 55.13.125, 51.12.116, 127.63.127, 55.14.125, 23.4.125, 55.5.125, 55.15.125, 55.0.126, and 55.47.125) were found. The aforementioned codes were obtained using a preliminary set of 21 differentials, that utilized 'Solatur' not Ames 31282 (Lebeda *et al.*, 2016) as the last member of the third triplet (group 3.7). Thus, respective comparisons of the scores of the first two septets of the 2012 (preliminary) and 2016 (current) triple septets reveal the unique characteristic of race S, namely it infects all commonly used melon CPM race differentials, with the exception of PI 313970. The third septets differ by one member (Ames 31282 *vs.* 'Solatur') and may not, therefore, be directly compared, though the two alternates are highly susceptible to many isolates (Lebeda *et al.*, 2016).

Lebeda *et al.* (2012) noted that the use of the triplet code revealed a great potential to identify new sources of resistance to powdery mildew, which may favor gene pyramiding. The same authors also emphasized that some differentials, previously considered as equivalents for powdery mildew resistance, showed different reactions, possibly due to the complexity and variability of mechanisms for resistance in the hosts, as well, the wide number of powdery mildew races, especially *P. xanthii*. Another explanation may result from the fact that many differentials possess two or more genes (Pitrat *et al.*, 1998) for resistance to *P. xanthii*, that though defeated may contribute some level of resistance (Simko *et al.*, 2014).

Disease progress

Analysis of variance identified significant differences ($P < 0.01$) among the differentials for all disease progress measurements in the greenhouse study (Table 2).

Student's *t*-tests showed significant differences ($P < 0.01$) between AUDPC and AUDPS, and between sAUDPC and sAUDPS (Table 2). Means separations by the four methods were similar, nearly identical, with a single difference between AUDPC and AUDPS, and no differences between sAUDPC and sAUDPS.

AUDPS estimated higher disease levels than AUDPC, with overall means of 220.05 and 168.12, respectively. In contrast, lower overall means were observed for sAUDPS (6.35) in comparison to sAUDPC (6.46). Nevertheless, Simko and Piepho (2012) emphasize that the efficacy of each method should be determined by the higher *F* test value, and lower CV, and square root of error means square (EMS). Accordingly, AUDPS and sAUDPS were slightly more efficient for evaluating powdery mildew progress in this test than AUDPC and sAUDPC, respectively (Table 2).

PI 313970 had the lowest disease progress estimates by all four disease progress estimator methods. Comparing the resistance delimiter (Table 2) to the values from this genotype, it can be considered resistant. Moreover, the Tukey HSD test separated it from the other differentials, as observed for the Triplet-Septet method. The concordance between the alphanumeric, Triplet-Septet and disease progress estimators indicate that powdery mildew race identification can be also performed by AUDPC and AUDPS. It is common for multiple evaluations of field, greenhouse and growth chamber CPM tests, so disease progress analysis does not add much complication to CPM testing.

The four disease progress estimates were highest on 'Iran H', although it was only statistically different from PI 124112, PI 124111 and MR-1, all of which exhibited intermediate levels of disease, and the resistant PI 313970.

In general terms, the sAUDPC and sAUDPS results reflected those obtained by AUDPC and AUDPS. However, Simko and Piepho (2012) explained that such standardizations are necessary to compare the efficiency of the two estimators, because they mathematically consider different time periods. AUDPS adds half of the average interval duration between observations, to the first and last disease assessments.

AUDPS was considered more efficient than AUPDC, by identifying higher disease levels and statistical significance, and lower CV. Nevertheless, there are reports successfully using AUDPC to estimate powdery mildew progress. Mitchell *et al.* (2007), studied the susceptibility of Galia-type melons to CPM using AUDPC and identified cultivars Nestor, Galileo, and Vicar as resistant. McGrath and Shishkoff (2003), using the same method, analyzed the efficiency of biologic products controlling

CPM infections, but lowest disease levels were obtained by chemical control, more specifically, the fungicides Chlorothalonil and Myclobutanil.

AUDPS has been considered practicable to identify resistance levels in other crops. Simko *et al.* (2014), detected four QTLs for resistance in lettuce to powdery mildew (*G. cichoracearum sensu strictu*) that explained 35 to 42% of the phenotypic variation, through AUDPS associated with disease percentage assessments. Two QTLs (*qDM2.1 and qDM5.1*) for resistance to downy mildew (*Bremia lactucae*) were identified in lettuce using AUDPS.

AUDPS was used to estimate disease progress for *Ralstonia solanacearum* on inbred lines of *Solanum* spp. in order to identify molecular markers associated with resistance. Susceptible 'Quatree carrées' and 'Spunta' had AUPDS values of 54.1 and 53.7, respectively, whereas 'MST 32/1' exhibited moderate (34.3) resistance, and resistant 'CRA 66' and 'Hawaii 7996' exhibited high-level resistance with values of zero (Baichoo & Jaufeerally-Fakim, 2017).

Based on the successful studies of AUDPS in other crop species, and the results obtained in this experiment, disease progress estimation methods may facilitate identification of new sources of resistance to CPM in melon as well as other cucurbit species.

Table 2: Disease progress estimates for 20 melon powdery mildew race differentials using Area Under Disease Progress (AUDPC), Area Under Disease Progress Stairs (AUDPS), and standardized values for the two disease progress estimators (Simko and Piepho, 2012); 41 days after inoculation in a greenhouse, Salinas, CA

Differential	AUDPC[z]	AUDPS[y]	sAUDPC[y]	sAUDPS[y]
Iran H	202.50 a[x]	266.06 a	7.8 a	7.7 a
Védrantais	196.17 ab	259.72 a	7.5 ab	7.5 a
PI 179901	191.33 ab	252.00 ab	7.4 ab	7.3 ab
PI 234607	170.33 abc	223.78 abc	6.6 abc	6.5 abc
AR HBJ	196.17 ab	255.39 ab	7.5 ab	7.4 ab
PMR 45	185.33 ab	246.00 ab	7.1 ab	7.1 ab
PMR 6	155.00 abc	201.22 abc	6.0 abc	5.8 abc
WMR 29	198.50 a	260.61 a	7.6 a	7.5 a
Edisto 47	177.67 abc	229.67 abc	6.8 abc	6.6 abc
PI 414723	169.50 abc	225.83 abc	6.5 abc	6.5 abc
PMR 5	171.67 abc	219.33 abc	6.6 abc	6.3 abc
PI 124112	145.50 bc	190.28 bc	5.6 bc	5.5 bc
MR-1	123.50 c	165.39 c	4.8 c	4.8 c
PI 124111	128.83 c	167.83 c	5.0 c	4.8 c
PI 313970	34.50 d	43.17 d	1.3 d	1.2 d
Noy Yzre'el	177.33 abc	235.11 ab	6.8 abc	6.8 ab
PI 236355	181.07 ab	231.02 abc	7.0 abc	6.7 abc
Negro	186.83 ab	243.17 ab	7.2 ab	7.0 ab
Nantais Oblong	192.17 ab	249.94 ab	7.4 ab	7.2 ab
Ames 31282	182.50 ab	238.83 ab	7.0 ab	6.9 ab
F test	14.37**	15.89**	14.37**	15.89**
t-test	-30.31**		7.50**	
Mean	168.12	220.05	6.46	6.35
CV (%)	9.98	9.57	9.98	9.57

[z]Van der Plank, 1963.
[y]Simko and Piepho, 2012.
[x]Means within columns followed by the same letter do not differ significantly at $P < 0.05$ by Tukey HSD test.
**Significant at $P < 0.01$.

Simulated AUDPC, AUDPS, sAUDPC, and sAUDPS estimates for disease reaction score 4 were 104.0, 138.67, 4.0, and 4.0, respectively (Table 3). Those values were considered the resistant–susceptible delimiters, whereby differentials with respective mean estimates equal or greater than those values were considered susceptible. Simulations must be done for each experiment, because the resistance delimiters may vary according to the rating scale used, number of assessments, and intervals among assessments (Simko & Piepho, 2012). PI 313970 is the only CPM differential with values lower than those aforementioned, thus, its resistance was confirmed quantitatively as well.

Table 3: Simulated data for disease progress using Area Under Disease Progress (AUDPC), Area Under Disease Progress Stairs (AUDPS), and standardized values for the two disease progress estimators, sAUDPC and sAUDPS (Simko and Piepho, 2012). A 1 to 9 disease rating scale was used to generate simulated raw data 15, 22, 32 and 41 days after inoculation (DAI)

Disease rating	Disease rating simulated				AUDPC[z]	AUDPS[y]	sAUDPC[y]	sAUDPS[y]
	15 DAI	22 DAI	32 DAI	41 DAI				
1	1	1	1	1	26.00	34.67	1.00	1.00
2	2	2	2	2	52.00	69.33	2.00	2.00
3	3	3	3	3	78.00	104.00	3.00	3.00
4	4	4	4	4	104.00	138.67	4.00	4.00
5	5	5	5	5	130.00	173.33	5.00	5.00
6	6	6	6	6	156.00	208.00	6.00	6.00
7	7	7	7	7	182.00	242.67	7.00	7.00
8	8	8	8	8	208.00	277.33	8.00	8.00
9	9	9	9	9	234.00	312.00	9.00	9.00

[z]Van der Plank, 1963.
[y]Simko and Piepho, 2012.

Melon CPM incited by race S is equivalent to race 127.127.126, as determined by Triplet-Septet method. PI 313970 is resistant to race 127.127.126 of melon CPM. Powdery mildew isolates identified by previous methods should be characterized using the Triplet-Septet method in order to relate previous alpha numeric race designations with their respective Triplet-Septet codes. So doing would facilitate communication among established and new plant breeders and pathologists, and would help them to more clearly relate race designations (with respect to the pathogen identity and host resistance profiles of cultivars and breeding lines) with extensionists, professional crop advisors, and farmers.

The susceptible differentials as determined by the alphanumeric and Triplet-Septet methods exhibited different levels of susceptibility. Disease progress estimates provide accurate disease assessments when done at equally distributed time intervals, and permit identification of multiple QTL for disease reaction. Such an approach may, therefore, be used to identify non-race specific resistance to CPM to stabilize reactions of melon genotypes to powdery mildew whenever new pathogenic races appear in endemic *P. xanthii* populations.

The AUDPS method of disease progress estimation proved better than AUDPC in this test for evaluation of melon genotypes for resistance to cucurbit powdery mildew. Resistance delimiters must, however, be calculated in all experiments, because they may vary as a function of the rating scale, number of assessments and intervals between assessments (Simko & Piepho, 2012).

CONCLUSION

The previous race S is equivalent to the race 127.127.126 by method Triplet-Septet. The Genotype PI 313970 is resistant to the race 127.127.126 of *Podosphaera xanthii.*

The differentials showed different levels of susceptibility.

The method AUDPS is indicated to evaluate genotypes for resistance to powdery mildew. However, resistance delimiters must be calculated in all experiments, since they may vary in function of the rating scale, number of assessments and interval between assessments.

Powdery mildew isolates identified by previous methods should be reclassified with the method Triplet-Septet, to be possible associations among previous researches and the new ones, aiming the standardization.

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REFERENCES

Baichoo Z & Jaufeerally-Fakim Y (2017) *Ralstonia solanacearum* upregulates marker genes of the salicylic acid and ethylene signaling pathways but not those of the jasmonic acid pathway in leaflets of Solanum lines during early stage of infection. European Journal of Plant Pathology, 147:615-625.

Bojorques Ramos C, Maruthachalam K, McCreight JD & Garcia Estrada RS (2011) *Podosphaera xanthii* but not *Golovinomyces cichoracearum* infects Cucurbits in a Greenhouse at Salinas, California. Cucurbit Genetics Cooperative Report, 33-34:24-28.

Coffey MD, McCreight JD & Miller T (2006) New races of the cucurbit powdery mildew *Podosphaera xanthii* present in California. Phytopathology, 96:25.

ISF - International Seed Federation (2016) The International Bremia Evaluation Board (IBEB) aims to identify new races of *Bremia lactucae* that pose a significant threat to the European lettuce industry. Available at: <http://www.worldseed.org/our-work/plant-health/other-initiatives/ibeb/>. Accessed on: May 27th, 2020.

Kuzuya M, Yashiro K, Tomita K & Ezura H (2006) Powdery mildew (*Podosphaera xanthii*) resistance in melon is categorized into two types based on inhibition of the infection processes. Journal of Experimental Botany, 57:2093-2100.

Lebeda A, Křístková E, Sedláková B, McCreight JD & Coffey MD (2016) Cucurbit powdery mildews: methodology for objective determination and denomination of races. European Journal of Plant Pathology, 144:399-410.

Lebeda A & Sedláková B (2004) Disease impact and pathogenicity variation in Czech populations of cucurbit powdery mildews. In: Lebeda A & Paris H (Eds.) Progress in Cucurbit Genetics and Breeding Research. Czech Republic, Palacky University Olomouc. p.281-287.

Lebeda A, Sedláková B, Křístková E, Vajdová M & McCreight JD (2012) Application of a new approach for characterization and denomination of races of cucurbit powdery mildews - a case study of Czech pathogen populations. In: X EUCARPIA meeting on genetics and breeding of Cucurbitaceae, Antalya. Proceedings, Çukurova University. p.172-180.

Limpert E & Müller K (1994) Designation of pathotypes of plant pathogens. Journal of Phytopathology, 140:346-358.

Liu L, Chen Y, Su Z, Zhang H & Zhu W (2010) A sequence-amplified characterized region marker for a single, dominant gene in melon PI 134198 that confers resistance to a unique race of *Podosphaera xanthii* in China. HortScience, 45:1407-1410.

McCreight JD (2003) Genes for resistance to powdery mildew races 1 and 2U.S. in melon PI 313970. HortScience, 38:591-594.

McCreight JD (2006) Melon-powdery mildew interactions reveal variation in melon cultigens and *Podosphaera xanthii* races 1 and 2. Journal of the American Society for Horticultural Science, 131:59-65.

McCreight JD & Coffey MD (2011) Inheritance of resistance in melon PI 313970 to cucurbit powdery mildew incited by *Podosphaera xanthii* race S. HortScience, 46:838-840.

McCreight JD, Coffey MD, Sedlakova B & Lebeda A (2012) Cucurbit powdery mildew of melon incited by *Podosphaera xanthii*: Global and western U.S. perspectives. In: Sari N, Solmaz I & Aras V (Eds.) X EUCARPIA meeting on genetics and breeding of Cucurbitaceae, Antalya. Proceedings, Çukurova University. p.181-189.

McCreight JD, Coffey MD, Turini TA & Matheron ME (2005) Field evidence for a new race of powdery mildew on melon. HortScience, 40:888-894.

McGrath MT (2001) Fungicide resistance in cucurbit powdery mildew: experiences and challenges. Plant Disease, 85:236-245.

McGrath MT (2006) Occurrence of fungicide resistance in *Podosphaera xanthii* and impact on controlling cucurbit powdery mildew in New York. In: Cucurbitaceae, North Carolina. Proceedings, Universal Press. p.473-482.

McGrath MT & Shishkoff N (2003) First report of the cucurbit powdery mildew fungus (*Podosphaera xanthii*) resistant to strobilurin fungicides in the United States. Plant Disease, 87:1007.

Mitchell J, Cantliffe D, Sargent S, Datnoff L & Stoffella P (2007) Fruit Yield, Quality Variables, and Powdery Mildew Susceptibility of Galia Melon Cultivars Grown in a Passively Ventilated Greenhouse. Proceedings of Florida State Horticultural Society, 120:162-167.

Pérez-García A, Romero D, Fernández-Ortuño D, López-Ruiz F, De Vicente A & Torés JA (2009) The powdery mildew fungus *Podosphaera fusca* (synonym *Podosphaera xanthii*), a constant threat to cucurbits. Molecular Plant Pathology, 10:153-160.

Pitrat M, Dogimont C & Bardin M (1998) Resistance to fungal diseases of foliage in melon. In: McCreight JD (Ed.) Cucurbitaceae '98: Evaluation and enhancement of cucurbit germplasm. United States of America, ASHS Press. p.167-173.

Pryor DE, Whitaker TW & Davis GN (1946) The development of powdery mildew resistant cantaloupes. Proceedings of the American Society for Horticultural Science, 47:347-356.

Salmon ES (1919) On forms of the hop (*Humilus lupulus* L.) resistant to mildew (*Sphaerotheca humuli* (D.C.) Burr.). The Journal of Agricultural Science, 8:455-460.

Salmon ES (1917) On the resistance to fungicides shown by the hop-mildew (*Sphaerotheca humuli* (D.C.) Burr.) in different stages of development. Annals of Applied Biology, 1:93-96.

Sedlářová M, Lebeda A, Mikšíková P, Duchoslav M, Sedláková B & McCreight JD (2009) Histological aspects of *Cucumis melo* PI 313970 Resistance to *Podosphaera xanthii* and *Golovinomyces cichoracearum*. Journal of Plant Diseases and Protection, 116:169-176.

Simko I & Piepho HP (2012) The area under the disease progress stairs (AUDPS): Calculation, advantage, and application. Phytopathology, 102:381-389.

Simko I, Rauscher G, Sideman RG, McCreight JD & Hayes RJ (2014) Evaluation and QTL mapping of resistance to powdery mildew in lettuce. Plant pathology, 63:344-353.

Stadnik MJ, Kobori RF & Bettiol W (2001) Oídios de cucurbitáceas. In: Stadnik MJ & Rivera MC (Eds.) Oídios. Jaguariúna, Embrapa Meio Ambiente. p.217-254.

Thomas CE (1986) Downy and powdery mildew resistant muskmelon breeding line MR-1. HortScience, 21:329.

Van der Plank JE (1963) Plant diseases: epidemics and control. New York, Academic Press. 349p.

Van Ettekoven K & Van Arend AJM (1999) Identification and denomination of new races of *Bremia lactucae*. In: 99th Eucarpia Meeting on Leafy Vegetables Genetics and Breeding, Olomouc. Proceedings, Palacky University. p.105-107.

Viana FMP, Santos AA, Freire FCO, Cardoso JE & Vidal JC (2001) Recomendação para controle das principais doenças que afetam a cultura do melão na Região Nordeste. Fortaleza, Embrapa Agroindústria Tropical. 21p. (Circular Técnica, 12).