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Generation mean analysis of resistance to downy mildew in adult muskmelon plants

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Abstract Downy mildew (*Pseudoperonospora cubensis*) is the most devastating disease in muskmelon (*Cucumis melo*). A generation mean analysis study was designed to determine the types of gene action and estimate the heritability for resistance to downy mildew in four selected crosses of muskmelon. Generation mean analysis revealed that genetic dominance may be of greater importance for expression of resistance to downy mildew in both greenhouse and field experiments and in all the crosses. The F_1 mean was significantly lesser than the mid-parent value and skewed towards resistant parent in all the crosses. Negative sign associated with gene effects indicated, in those crosses, that disease level could be decreased in relation to midparent. All the crosses expressed significant and positive additive (d) gene effects. Dominance (h) and dominance × dominance (l) gene effects had opposite sign in all crosses and both experiments, which implied duplicate type of gene action. High mid-parent heterosis in all the crosses indicated strong dominance effects (as combination of parental alleles) for resistance to downy mildew. In all the crosses, both resistant and susceptible parent contributed one or more dominant/partially dominant factors for resistance. Estimates of broad-sense heritability were high and relatively consistent in both experiments. The two different screening experiments showed that fixable gene effects (d + i) were lower than the non-fixable gene effects (h + l) in all the crosses indicating greater role of non-additive effects in the inheritance of resistance to downy mildew. Resistance to downy appeared to be controlled mainly by dominance effects, therefore the inbred lines IIHR 121 and IIHR 122 could be used strategically to exploit heterotic effects.

Keywords *Cucumis melo* · Downy mildew · *Pseudoperonospora cubensis* · Generation mean analysis · Quantitative resistance · Dominance

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Introduction

Downy mildew [*Pseudoperonospora cubensis* (B & C) Rost.] is the most common and devastating disease in cucurbits. During cool and moist weather is most serious problem in muskmelon (*Cucumis melo* L.) worldwide. The disease is confined mostly to the leaves, although the fruit of infected plants may be of poor quality due to loss of foliage.

The principal method of controlling downy mildew is by protective fungicides. However, protective fungicides are less effective when used on susceptible varieties or when disease pressure is heavy. The most efficient systemic fungicide metalaxyl, lost efficacy due to variation of fungicide resistance in populations of *Pseudoperonospora cubensis* (Katan 1982; Samoucha and Cohen 1985; Urban and Lebeda 2007). The occurrence of strains of *Pseudoperonospora cubensis* resistant/tolerant to some fungicides encouraged research on resistance breeding. Six pathotypes can be distinguished based on host compatibilities between various isolates and different cucurbit taxa (Cohen et al. 2003; Thomas et al. 1987). Development of cultivars with inherent resistance to downy mildew is one of the most effective and economical means of controlling the downy mildew.

There have been many reports on the inheritance of resistance to downy mildew in muskmelon (Cohen et al. 1985; Epinat and Pitrat 1989; Epinat and Pitrat 1994a, b; Kenigsbuch and Cohen 1992; Thomas et al. 1988). Early records postulated single dominant (Angelov and Krasteva 2000), two incompletely dominant (Cohen et al. 1985; Thomas et al. 1988) and two partial dominant (Kenigsbuch and Cohen 1989, 1992) genes to be responsible for resistance to downy mildew in muskmelon. Although the adult plant stage is usually the important stage for resistance screening, often seedlings were assessed for disease reactions in controlled conditions. Cohen et al. (1984) has shown large positive correlations between seedlings and adult plant reactions and between greenhouse and field disease responses. Perchepied et al. (2005) reported genetic analysis of partial resistance to downy mildew using a recombinant inbred line (RIL) population derived from 'PI 124112' and using quantitative evaluations. In most cases, monogenic or digenic resistance to downy mildew using a discontinuous scale (1–4 reaction types) has been reported.

The choice of an appropriate breeding method for improvement of quantitative characters depends largely on gene action. However, the effect of individual gene cannot be measured. Therefore, the effect of individual genes must be considered along with suitable statistical procedures to obtain genetic information. Generation mean analysis has been used to detect types of gene action involved in several quantitatively inherited traits including disease resistance (Dias et al. 2004; St. Amand and Wehner 2001). Information about nature and magnitude of gene actions involved in resistance for downy mildew can be useful for breeding downy mildew resistant cultivars/varieties in muskmelon. Therefore, a generation mean analysis study was designed to determine the types of gene action and estimate the heritability for resistance to downy mildew in field and greenhouse conditions.

Materials and methods

Plant materials

Downy mildew resistant lines IIHR 121 and IIHR 122 were derived from segregating melon lines introduced from Russia, EC 564749 and EC 564750, respectively which both belongs to *Cucumis melo* var. *cantaloupensis*. The lines were purified for five generations before use in this study. IIHR 121 and IIHR 122 were crossed with downy mildew susceptible variety Punjab Sunehri (Punjab Agriculture University, Ludhiana, India), cultivar RM 43 (popular cultivar in Northern states of India, highly resistant to powdery mildew) and inbred line IIHR 681 (belongs to species *Cucumis callosus*, resistant to fruit fly) to derive four F_1 hybrids viz. Punjab Sunehri × IIHR 122, RM 43 × IIHR 121, IIHR 681 × IIHR 121 and IIHR 681 × IIHR 122. These F_1s were crossed to their parents P_1 (susceptible) and P_2 (resistant) to get BC_1P_1 and BC_2P_2 generations, respectively. On the same F_1s, F_2 seeds were generated by self-pollination. The experimental material comprised of six generations (P_1, P_2, F_1, F_2, BC_1P_1 and BC_2P_2) derived from each of the four crosses was screened in field and greenhouse conditions for resistance to downy mildew during 2006–07 at the Indian Institute of Horticultural Research (IIHR), Bangalore, India.

Inoculation in greenhouse

Six generations of each cross were raised in 50 unit plastic potting trays containing sterilized coco-peat as growth media in the greenhouse in September 2006. For each cross, 10 plants each in P_1, P_2 and F_1, 80 plants in F_2 and 40 plants in back cross generations were raised in three replicates. Inoculation and post inoculation procedure as proposed by Cohen et al.

(1984) and Kenigsbuch and Cohen (1992). The source pathogen was isolated from a muskmelon growers' field near Bangalore, India. A colony of *Pseudoperonospora cubensis* maintained on susceptible variety, Arka Jeet in greenhouse at 18–26°C. Infected leaves from Arka Jeet were collected and gently washed in distilled water to release the spores. Test plants were inoculated on adaxial leaf surfaces with sporangial suspension containing 10,000 sporangia per milliliter using atomizer. The concentration of spores was measured with a hemocytometer. The inoculated plants were kept in high humidity black polythene tent for about 20 h and returned to greenhouse bench. On the seventh night, seedlings were again placed in high humidity black polythene tent for 20 h to allow fungal sporulation. Disease reactions were noticed on 8th day after inoculation. Plants were maintained for 6 weeks after inoculation. Seedlings were hand watered every day. Nutrition solution containing 150 mg N, 150 mg P and 150 mg K per liter of water was supplied every week. One spray of micronutrients @ 0.5 ml/l of water was supplied at 2–3 leaf stage. Seedlings trays were arranged with proper spacing on greenhouse benches to allow the spread of growing plants.

Inoculation in field

Seedlings of all the generations of each cross were raised in 50 unit plastic potting trays containing sterilized coco-peat as growth media in greenhouse. At two-leaf stage, seedlings were transplanted to main field in November 2006. Seven days after transplanting, the adaxial leaf surface was sprayed with a sporangial suspension containing 10,000 sporangia per milliliter by hand sprayer. Susceptible variety Arka Jeet was planted at regular intervals all over the field for uniform spread of disease. Seedlings were spaced at 3.0 m between beds (centre to centre) and 0.45 m within bed. Field plots with 10 plants each were arranged in three randomized blocks. The P_1, P_2 and F_1 were planted one plot per block, F_2 planted in eight plots per block and BC_1P_1 and BC_2P_2 each planted in four plots per block.

Disease assessment

Cohen et al. (1984) used percent leaf loss to describe the reaction of older plants in field plots and correlated lesion type in artificial inoculation at 2-leaf stage in greenhouse to facilitate the selection of resistant plants. Perchepied et al. (2005) used two variables: the disease score at the final scoring date and the area under the disease progress curve (AUDPC) to assess the disease in seedlings and adult plants. In the present study, disease was assessed 30 days after inoculation, which coincides with the flowering stage in the greenhouse experiment and 50 days after inoculation, which coincides with fruit development stage in field experiment. Plants in greenhouse were assessed at the flowering stage (30 days after inoculation) for understanding of resistance of adult plants. Each plant was visually assessed for percent leaf area infected, using linear 0 to 5 scale indicating average grade of all the leaves. 0 = healthy and no symptoms, 1 = 1–5%, 2 = 6–10%, 3 = 11–20%, 4 = 21–30%, 5 = >30% of total leaf area covered with chorotic and/or necrotic symptoms. The Percent Disease Index (PDI) was calculated using the formula proposed by Wheeler (1969): PDI = Sum of numerical values/(number of leaves graded × maximum rating) × 100.

Statistical analysis

Estimates of PDI were transformed to arcsine values prior to generation mean analysis. The means and variances were calculated as suggested by Hayman (1958). The presence of epistasis was detected by using A, B, C and D scaling tests proposed by Mather (1949) and Hayman and Mather (1955). Just the significance of either one or two scaling test implies the insufficiency of simple additive-dominance model. The model proposed by Hayman (1958) estimated gene effects. The gene effects were defined in Hayman's notations as [m] = mean of the F_2 generation, [d] = additive gene effect, [h] = dominance gene effect, [i] = additive × additive gene effect, [j] = additive × dominance gene effect and [l] = dominance × dominance gene effect. The type of epistasis was determined only when dominance (h) and dominance × dominance (l) effects were significant, when these effects had the same sign, the effects were complementary while different signs indicated duplicate epistasis (Kearsey and Pooni 1996). Generation mean analysis was computed using statistical program SPAR1 (Indian Agricultural Research Institute, New Delhi, India). Broad-sense

heritability was calculated using Allard's approach (1960): $H = (\sigma^2_{F_2} - \sigma^2_E)/\sigma^2_{F_2}$ in which $\sigma^2_{F_2}$ is phenotypic variance of F_2 population and σ^2_E is environmental variance. Mid-parent heterosis estimated as the percentage deviation of the F_1 from the mid-parent values.

Results

Generation means

Means and standard errors for parents, F_1, F_2 and backcross generations are presented in Table 1. The F_1 mean was significantly lesser than the midparent value in all the crosses. In both screening experiments and all crosses, the mean of F_2 exceeded that of the F_1 and BC_2P_2 but lower than BC_1P_1 generations. In general, mean of BC_2P_2 generations were lesser than the mean of F_1 generations in all the crosses. Individual F_2, BC_1P_1 and BC_2P_2 progeny observed that transgressed with extreme mean PDI values than either parent in both screening experiments. This could be due to contrasting parental means. However, contrasting parents are prerequisite for generation mean analysis (Mather and Jinks 1971). All the progenies in BC_1P_1 populations expressed lower disease ratings than the susceptible parents, RM 43 and IIHR 681.

Gene effects and heterosis

Scaling tests indicated presence of epistasis in all the crosses. All the six genetic parameters were significant in all crosses except RM 43 × IIHR 121 in both screening experiments (Table 2). All the crosses expressed significant and positive additive (d) gene effects in both experiments. In field experiment, dominance × dominance (l) interaction effects was the largest component of gene effects in three out of four crosses. Dominance (h) and dominance × dominance (l) gene effects signed opposite in all crosses and both experiments. However, in greenhouse experiment, significant and negative additive × additive effects expressed in crosses IIHR 681 × IIHR 122 and IIHR 681 × IIHR 121. The sum of additive effects (d + i), in terms of fixable component was much lower than the sum of dominance effects (h + l) in terms of non-fixable component in all the crosses in both experiments. However, negative sign

Table 1 Means and standard errors of six generations of four muskmelon crosses screened for resistance to downy mildew in field and the greenhouse conditions

Generation	Field condition			Greenhouse condition			
	Punjab Sunehri × IIHR 122	RM 43 × IIHR 121	IIHR 681 × IIHR 121	IIHR 681 × IIHR 122	Punjab Sunehri × IIHR 122	RM 43 × IIHR 121	IIHR 681 × IIHR 121	IIHR 681 × IIHR 122
P_1	55.08 ± 0.58 (26)	57.50 ± 0.51 (28)	55.00 ± 0.88 (27)	55.00 ± 0.88 (27)	42.13 ± 0.61 (27)	70.47 ± 0.77 (26)	49.73 ± 0.50 (29)	49.73 ± 0.50 (29)
P_2	8.80 ± 0.40 (29)	5.60 ± 0.53 (28)	5.60 ± 0.50 (28)	8.80 ± 0.50 (27)	12.88 ± 0.59 (30)	4.80 ± 0.60 (26)	4.80 ± 0.45 (28)	12.88 ± 0.59 (30)
F_1	16.27 ± 0.50 (25)	20.55 ± 0.40 (27)	11.52 ± 0.44 (30)	9.37 ± 0.57 (30)	9.87 ± 0.59 (28)	18.30 ± 0.75 (27)	14.43 ± 1.02 (27)	18.88 ± 0.69 (26)
F_2	23.82 ± 0.53 (224)	21.52 ± 0.58 (229)	20.19 ± 0.58 (231)	19.47 ± 0.67 (222)	18.85 ± 0.42 (218)	22.80 ± 0.73 (231)	26.40 ± 0.68 (228)	29.16 ± 0.45 (223)
BC_1P_1	54.88 ± 0.58 (108)	36.88 ± 0.56 (104)	41.03 ± 0.40 (102)	44.28 ± 0.48 (111)	43.50 ± 0.85 (110)	30.70 ± 0.47 (100)	32.48 ± 0.33 (111)	34.17 ± 0.50 (113)
BC_2P_2	14.34 ± 0.13 (104)	11.23 ± 0.11 (109)	12.56 ± 0.12 (104)	15.55 ± 0.15 (95)	15.87 ± 0.59 (113)	13.18 ± 0.63 (106)	6.30 ± 0.57 (104)	9.23 ± 0.41 (106)

Number in parentheses indicate the number of plants screened for each generation

Table 2 Estimates of gene effects, type of epistasis and midparent heterosis (%) for resistance to downy mildew in four muskmelon crosses screened in field and the greenhouse conditions

Gene effects and heterosis	Field conditions				Greenhouse conditions			
	Punjab Sunehri × IIHR 122	RM 43 × IIHR 121	IIHR 681 × IIHR 121	IIHR 681 × IIHR 122	Punjab Sunehri × IIHR 122	RM 43 × IIHR 121	IIHR 681 × IIHR 121	IIHR 681 × IIHR 122
[m]	23.82**	21.52**	20.19**	19.47**	18.85**	22.80**	26.40**	29.16**
[d]	40.54**	25.65**	28.47**	28.73**	27.63**	17.52**	26.18**	24.93**
[h]	27.50**	0.87	7.65*	19.27**	25.67**	−22.77**	−40.88**	−42.25**
[i]	43.17**	10.13**	26.43**	41.79**	43.31**	−3.43	−28.04**	−29.83**
[j]	17.40**	−0.30	3.78**	5.64**	13.00**	−15.31**	3.72**	6.51**
[l]	−85.20**	−2.15	−49.99**	−78.92**	−87.29**	27.55**	33.88**	43.41**
Epistasis	D[b]	–	D	D	D	D	D	D
mph (%)[a]	−49.06**	−34.9**	−61.97**	−70.62**	−64.12**	−58.77**	−57.58**	−39.69**

*, ** Significant at $P \leq 0.05$ and $P \leq 0.01$, respectively

[a] *mph* mid-parent heterosis

[b] *D* duplicate epistasis, – no epistasis

Table 3 Estimates of broad-sense heritability for resistance to downy mildew in four crosses of muskmelon screened in field and the greenhouse conditions

Crosses	Field conditions	Greenhouse conditions
Punjab Sunehri × IIHR 122	0.90	0.74
RM 43 × IIHR 121	0.92	0.88
IIHR 681 × IIHR 121	0.86	0.83
IIHR 681 × IIHR 122	0.89	0.77

associated with gene effects indicated, in those crosses, disease level could be decreased in relation to midparent. Mid-parent heterosis ranged from −70.62 to −34.9% ($P \leq 0.01$) in field experiment and −64.12 to −39.69% ($P \leq 0.01$) in the greenhouse experiment (Table 2).

Heritability estimates

Estimates of broad-sense heritability are presented in Table 3. The estimates for broad-sense heritability ranged from 0.86 to 0.92 averaging 0.88 in field experiment and 0.74 to 0.88 averaging 0.81 in greenhouse experiment, respectively.

Discussion

Generation mean analysis revealed genetic dominance may be of greater importance in all the crosses and both the screening experiments. The mean PDI of F_1 in all the crosses indicated that crosses may be accompanied by dominance effects. Further high magnitude of significant and negative midparent heterosis in all crosses confirmed predominance of dominance effects. The marked skewness of the BC_2P_2 means and low variance in BC_2P_2 compared to the BC_1P_1s is an indicative of genetic dominance in field conditions. Negative dominance or dominance × dominance gene effects in all the crosses and in both experiments tends to increase negative heterosis. Internal cancellation of oppositely signed dominance and dominance × dominance gene effects could reduce heterosis. But high mid-parent heterosis detected in two different screening conditions in all the crosses could be mainly due to dominance/over-dominance and should be exploited through heterosis breeding. All crosses expressed duplicate type of epistasis in both experiments, except RM 43 × IIHR 121 in field experiment. In a diallel analysis, Epinat and Pitrat (1994a) predicted numerous additive loci and duplicate type of epistasis for resistance to downy mildew in muskmelon.

Generation mean analysis assumes unidirectional distribution of genes between the two parents. Evidence of transgressive segregants in F_2 and BC_1P_1 generations in all the crosses would be due to the presence of at least one factor for resistance in the susceptible parent (Punjab Sunehri, RM 43 and IIHR 681). Further, absence of segregants more susceptible than RM 43 and IIHR 681 in the BC_1P_1 generations of crosses RM 43 × IIHR 121, IIHR 681 × IIHR 122 and IIHR 681 × IIHR 121 confirmed that this factor in susceptible parent is dominant/partially dominant. In all the crosses, both resistant and susceptible parent contributed one or more dominant/partially dominant factors for resistance. A stable resistance may be obtained by a combination of complete and partial resistance factors (Perchepied et al. 2005). Epistatic interactions detected failed the analysis assumption on direction of distribution of genes between the parents. Epistasis effects would seriously bias any attempt to partition the genetic variances of the segregating generations into additive or dominance components. Therefore only broad-sense heritability estimates were calculated. The estimates of broad-sense heritability were high and relatively consistent in both screening experiments suggests that the transfer of resistance factors to recipient parents by donor parents is highly possible. High heritability can increase the prevalence of a particular trait under selection (Erin 2002). Epinat and Pitrat (1994a) reported high heritability and low non-additivity for resistance to downy mildew in muskmelon.

Inadequacy of Mendalian genetics strengthens the hypothesis of more than one gene control of resistance to downy mildew in the present study. The frequency distribution of responses of segregating F_2, BC_1P_1 and BC_2P_2 generations were continuous in all the crosses, indicating downy mildew resistance is a quantitative trait (data not presented). Epinat and Pitrat (1994a, b) reported continuous variation for the resistance character ranking from a high resistance to high susceptibility to downy mildew in muskmelon. Quantitative or partial resistance may reduce the selection pressure for virulence in the pathogen population and could thus stabilize the host-pathogen system (Crill 1977). Race non-specific type of resistance is characterized by continuous variation in phenotypic appearance and complex polygenic inheritance (Black 1970; Umaerus 1970). Most of the downy mildew resistant lines used in previous studies were derived from Indian melon line which belongs to *Cucumis melo* var *reticulatus*, but the resistant lines, IIHR 121 and IIHR 122, used in the present study were derived from two Russian lines belongs to *Cucumis melo* var.*cantaloupensis*. Therefore, factor/s for resistance would be different from factor/s reported from the other sources of resistance to downy mildew in muskmelon.

Two different screening experiments revealed that fixable gene effects (d) and (i) were lower than the non-fixable (h) and (l) gene effects in all the crosses indicating greater role of non-additive effects in the inheritance of resistance to downy mildew. Breeding methods like diallel selective mating or biparental mating in early segregating generations might prove to be effective approaches. However, high mid-parent heterosis indicated that the resistance is controlled mainly by dominance effects, therefore the inbred lines IIHR 121 and IIHR 122 could be used strategically to exploit heterotic effects (e.g., diallel analysis). Intermating certain desirable segregants followed by selection might also be useful breeding strategy to obtain progenies with higher level of resistance then either parent. Identification of linked markers can be used to select for the rare recombinants that combine the favourable alleles. Genetic dissection of quantitative resistance to downy mildew in muskmelon allows the development of marker-assisted selection for breeding, the characterization of the genes underlying resistance QTLs of genetic resources and the isolation of the corresponding genes.

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