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LIST OF THE CONTRIBUTIONS

Robi R, Sreelathakumary I (India) Influence of maturity at harvest on capsaicin and ascorb acid content in hot chilli (Capsicum chinense Jacq.)	
Yazawa S, Yoneda H, Hosokawa M, Fushiki T, Watanabe T (Japan)	
Novel capsaicinoid like substances in the fruits of new no pungent cultivar 'CH-19 sweet' of pepper (Capsicu annuum)	m
Valšíková M, Belko I (Slovakia) Evaluation of sweet pepper assortment (Capsicum annuu. L.)	m 21
Rivera Martinez A, Terrén Poves L, Rodriguez Bao JM, Andrés Ares JL, Fernández Paz J (Spain) Characterization of local pepper lines from Northwest Spain	n25
Fouad M (Egypt) Green pepper germplasm selection for improved production under heat and drought stress conditions	
Sheela KB, George TE, Peter KV (India) Morphological and biochemical traits of selected accession of bird pepper (Capsicum frutescens L.)	
Timina OO, Tsykaliuk RA, Orlov PA (Moldova, Belarus) The identification of genotypes quantitive characters be cluster-regression analysis	
Mishra AC, Singh RV, Ram HH (India) Studies on genetic variability in Capsicum (Capsicum annuum L.) under mid hills of Uttaranchal	
Mishra AC, Singh RV, Ram HH (India) Studies on genetic divergence in Capsicum (Capsicum annuum L.) in Uttaranchal hills	

Mini S	Variability, heritability and genetic advance in wax type chilli (Capsicum annuum L.)	49
Sreela	thakumary I, Rajamony L (India) Correlation and path coefficient analysis for yield in hot chilli (Capsicum chinense Jacq.)	53
Mathe	w D, Doijode SD, Madhavi Reddy K (India) Correlation and path coefficient analysis in five species of Capsicum	57
Johri :	S, Singh RV, Mishra AC (India) Combining ability studies in Indian and exotic germplasm of Capsicum (Capsicum annuum L.)	61
Malath	Per se performance and heterosis of two hybrids of chillies (Capsicum annuum L.) for qualitative traits in three different seasons	65
Manju	PR, Sreelathakumary I. (India) Genetic divergence in hot chilli (Capsicum chinense Jacq.).	69
	D, Hundal JS, Dhillon TS, Singh P, Kaur S, Chawla N	
(India)	Development of disease resistant hot pepper hybrids suitable for processing	73
Nowa	czyk P, Nowaczyk L (Poland) Yielding of Capsicum frutescens L. soft-flesh breeding forms	77
Pande	eva R (Bulgaria) Incomplete anthocyaninless mutations in Capsicum annuum L. x C. chinense Jacq. Hybrids	81
Liu W	Y, Gniffke PA (Hong Kong, Taiwan) Stability of AVRDC's cytoplasmic male sterile (cms) pepper lines grown under low temperatures	85

Mezghani M, Jemmali A, Tarchoun N (Tunisia) Implication of the cambial tissue in the essential calluformation on hot pepper (Capsicum annuum L.) petio explants cultivated in vitro	le -
Soniya EV, Nair GM (India) Multiple shoot regeneration and indirect organogenesis chilli pepper (Capsicum annuum L.)	
Girija D, Fatima AG, Kuriakose LS, Nazeem PA, Joseph L, Indira P, Beena PS, Shaju KV (India) A viable protocol for direct regeneration of bell peppe (Capsicum annuum L.) cv. 'California Wonder'	er 97
Irikova T, Rodeva V (Bulgaria) Anther culture of pepper (Capsicum annuum L.): the effect of nutrient media	ot
Arnedo Andrés MS, Garcés Claver A, Esteban Chapapría J, Peiró Abril JL, Palazón C, Luis Arteaga M, Gil Ortega R (Spain)	
Application of anther culture and molecular markers to pepper breeding program for diseases resistance	a 105
Reddick BB, Habera LF (U.S.A.) New resistance to plant viruses in pepper	109
Herison C, Rustikawati, Sudarsono (Indonesia) Genetics of resistance against Cucumber Mosaic Viru (CMV) in hot pepper (Capsicum annuum L.)	s 113
Horváth J, Kovács J, Kazinczi G,Takács AP (Hungary) Reaction of Capsicum genotypes to Obuda Pepper Virus Tobacco Mosaic Virus and Cucumber Mosaic Virus	i, 117
Singh Y, Sood S (India) Screening of sweet pepper germplasm for resistance to bacterial wilt (Ralstonia solanacearum)	o 121
Baral J, Sy O, Bosland P.W. (U.S.A.) A comparison between a detached leaf and a whole plan method for screening Phytophthora foliar blight resistance in chile (Capsicum annuum)	า

Mar. Mat

Vajnan T, Khirbhat SK, Mehra R (India) Biocontrol of fruit rot of Capsicum using antagonistic microorganisms	129
Khirbhat SK, Vajnan T, Mehra R (India) Cultural and pathogenic variations among nine isolates of Colletotrichum capsici causing fruit rot of Capsicum	131
Daliya T, Wilson D (India) Ranking of brinjal genotypes using selection index values	135
Panda B, Singh YV, Ram HH (India) Combining ability studies for yield and yield attributing traits in round-fruited eggplant (Solanum melongena L.) under Tarai condition of Uttaranchal, India	137
Thangamani C, Jansirani P, Veeraraghavathatham D (India) Evaluation of F ₁ hybrids of brinjal (Solanum melongena L.) for yield ans quality	141
Doshi KM (India) Influence of biochemical factors on the incidence of shoot and fruit borer infestation in eggplant	145

GENETICS OF RESISTANCE AGAINST CUCUMBER MOSAIC VIRUS (CMV) IN HOT PEPPER (Capsicum annuum L.)

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Key words: Capsicum annuum, CMV, inheritance, resistance

Abstract

The genetic nature of cucumber mosaic virus (CMV) resistance was studied in three crosses, viz C1024 (resistance) x CA87067 (susceptible), C1024 x Chilli (susceptible), and C1034 (resistance) x CA87067. CMV resistance was controlled by nuclear, recessive simple gene. Broad and narrow sense heritability estimates were high.

Introduction

Cucumber mosaic virus (CMV) is one of the most prevalent and widespread virus infecting hot pepper (Duriat, 1996). In Indonesia, eradication of infected plants and insecticide application to control insect vectors in order to restrain CMV was known ineffective. This is because the virus has wide range of hosts (Palukaitis *et al.*, 1992) and insect vectors are always exist in the field. Therefore, planting of high-yielding and CMV resistance cultivars is the only effective and sustainable disease management strategy.

High-yielding and CMV resistance cultivar development can be accomplished through breeding programs which combine gene(s) controlling resistance against CMV into a high-yielding genotype. From the previous work, we identified several pepper accessions potential to be the sources of CMV resistance controlling gene(s) (Herison, et al., 2003). The present study was conducted to understand the genetic nature of CMV resistance controlling genes in these accessions. This information would be of great importance in designing an effective breeding program for high-yielding and CMV resistance.

Material and Methods

Four genotypes were used in this study, they were accession C1024, C1034, CA87067, and Chilli. Accession C1024 and C1034, derived from PBC375 and KA-2 respectively, were advance breeding lines selected for CMV resistance. CA87067 was a CMV susceptible line carrying TMV resistance character. Chilli was a CMV susceptible line potentially high-yielding. The parents, F1, F1resprocal, BC1, BC2, and F2

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generations from the crosses of C1024 x CA87067, C1024 x Chilli and C1034 x CA87067 were evaluated for CMV resistance.

All seedlings of each generation were grown individually in 200 ml plastic pot containing sterilized potting mix. Seedlings were maintained in an insect proof glasshouse with 50% shading intensity. Source of CMV isolate, method of mechanical inoculation, and disease examination were similar to those of the previous works (Herison, et al., 2003). The severity of symptoms were scored following the scoring after Dolores (1996). Plants were classified into resistant, intermediate susceptible, and susceptible reaction type, when their symptoms were scored 0, 1-3, and 4-5, respectively.

The existence of maternal effect and magnitude of gene action were estimate by calculating the ratio potency after Petr and Frey (1966). The presence of major gene effect in governing resistance against CMV was identified through Shappiro and Wilk normality test on the frequency distribution of F2 generation. Significant deviation from the normal distribution of F2 generation was an indicator of the presence of major gene effect. To estimate the number of genes controlling CMV resistance, the observed number of each group of plants with resistance, intermediate susceptible, and susceptible reaction type within the F2 generation were tested for various genetic ratios by chi-square analysis. Heritability in Broad sense and narrow sense were estimated by the method after Allard (1960) and Warner (1952), respectively.

Result and Discussion

The mean severity of symptom of F1s was similar to that of their reciprocals in all crosses (Table 1). This result indicated that there was no maternal effects controlling the inheritance of CMV resistance. The mean severity of F1 populations was higher than the midparent values, and the mean severity of F2s were higher than those of F1s and inclined toward susceptible parents. In all crosses, the calculated ratio potency were negative. The results conformed to the conclusion of Singh and Thakur (1977) and Rusko and Csillery (1980) that the genetic nature of CMV resistance was under control of recessive genes. The frequency distribution of F2 population in all three crosses were significantly deviate from normal distribution. This suggested that there were major genes involved the expression of CMV resistance in all crosses.

In C1024 x CA87067 cross, F2 sample population consisting of 191 plants segregated for 8 plants with resistant (R) reaction types, 35 plants with intermediate susceptible (I) reaction types and 148 plants with susceptible (S) reaction type (Table 2). This segregation fit with a 1(R):3(I):12(S) ratio, indicating that in this cross there were two segregating major genes governing CMV resistance between C1024 and CA87067 with dominant epistatic gene action. In cross of C1024 x Chilli, 310 plants of F2 sample population segregated for 45 plants with resistant (R) reaction types, and 265 plants with susceptible (S) type reaction. This segregation fit with ratio of 9 (R): 55 (S) indicating that in this cross there were three segregating genes between C1024 x Chilli. Involved in controlling CMV resistance with complex epistatic type of gene interaction. Meanwhile, in C1034 x CA87067 cross, an F2 sample population of 200 plants segregated for 35 plants with resistant (R) reaction types, to 165 plants with susceptible (S) type reaction. This segregation fit with a ratio of 3 (R) to 13 (S) indicating that there were two segregating genes between C1034 and CA87067 governing CMV resistance with epistatic type non-allelic interaction.

With regard to number of genes, the F2 population of C1024 x CA87067 cross supported the hypothesis of two segregating genes. Meanwhile, the segregated population of C1024 x Chilli cross support the three gene hypothesis. This result indicated that C1024 has at least 3 CMV resistance genes, and the CA87067 although it is very susceptible, it probably possessed at least one gene controlling resistance against CMV. Segregated F2 population of C1034 x CA87067 cross revealed that C1034 has at least 2 CMV resistance genes. Further study is needed whether one or all of the CMV resistance gene(s) of C1034 are similar to that of C1024.

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Table 1. Mean and standard error of scores, ratio potency, and heritability estimate in different crosses for CMV resistance in hot pepper

Generation	C1024 x CA87067	C1024 x Chilli	C1034 x CA87067		
P1 0.00 ±0.00		0.00±0.00	0.20±0.13		
P2	4.70±0.11	4.20±0.14	4.80±0.13		
F1	3.20±0.13	2.60±0.16	4.10±0.18		
F1r	3.10±0.13	0.70±0.48	4.20±0.20		
Нр	-0.270	-0.73	3.40		
h^2_{BS}	0.91	0.94	0.93		
h ² _{NS}	0.67	0.77	0.79		

P1=the first parent, P2 = the second parent, F1 = P1 x P2, F1r = P2 x P1 (the reciprocal cross), Hp = ratio potency, h^2_{BS} = hertability in broad sense, and h^2_{NS} = heritability in narrow sense

Table 2. Segregation ratio of F2 for CMV resistance and chi-square test of population for resistant to susceptible reaction type

Cross	Observed			Expected			Ratio	χ^2	P
0 / 0	R	I	S	R	I	S			
C1024 x CA87067	8	3	148	12	36	143	1:3:12	1.43	0.478
C1024 x Chilli	45		265	44		266	9:55	0.02	0.818
C1034 x CA87067	35		165	38		151	3:13		0.651

R = resistant, I = intermediate susceptible, and S = susceptible

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