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ARTICLE

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Inheritance of resistance to papaya ringspot virus-watermelon strain (PRSV-W) in 'Whitaker' summer squash line

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Abstract – *The objective of this work was to study the genetic control of the PRSV-W (Papaya Ringspot Virus – Watermelon Strain) resistance in* Cucurbita pepo *'Whitaker' line. Plants of parental lines Whitaker (resistant) and Caserta (susceptible), and of the generations F_1, F_2, BC_{11} and BC_{12} were evaluated for their reactions to PRSV-W. Caserta plants showed severe mosaic symptoms, while Whitaker grew vigorously and remained almost totally symptom-free. Most of the F_1, F_2 and backcross plants also presented severe mosaic symptoms. Data were used to test a hypothesis of monogenic inheritance under different presumed degrees of dominance, and genetic models were tested using maximum likelihood tests of genetic control. Broad-sense heritability was of 0.57 for the first evaluation. Resistance to PRSV-W in* C. pepo *'Whitaker' is due to a major gene effect summed to polygenic effects.*

Key words: Cucurbita pepo*, genetic control, potyvirus, virus resistance.*

INTRODUCTION

PRSV-W (*Papaya Ringspot Virus – Watermelon* Strain) affects all agricultural *Cucurbitaceae species*, achieving great economic importance due to its destructiveness. The virus is transmitted in a non-persistent manner by numerous species of aphids, including *Myzus persicae* and *Aphis* spp. (Vieira et al. 2010). It has become one of the most limiting factors for cucurbit crops in warm climate countries like Brazil, where aphids can easily survive throughout the year (Nascimento et al. 2011). Virus symptoms vary from chlorotic spots and mosaic to distortions, mainly in apical leaves. Flower deformations and fruit inhibition can be observed as well.

Higher virus disease incidence is related to greater aphid populations (Bateson et al. 2002). PRSV-W control is very difficult, and the method that has been widely used is the insecticide sprays to eliminate virus vectors. Cross protection with mild strain of the virus has been tested with some success, but it needs further studies before it can be recommended to farmers, due to its possible synergistic effect when the plants are infected by more than one virus. Genetic resistance appears to be the ideal virus control strategy, both economically and environmentally (Rezende and Muller 1995).

Genetic resistance has been found in *Cucurbita ecuadorensis*, *C. maxima*, *C. foetidissima* and *C. moschata* squash species. However, resistant sources have not been found in *C. pepo* (Maluf et al. 1986, Kuabara et al. 1987). 'Whitaker' was the first *C. pepo* line reported as resistant; it was developed at the Cornell University through interspecific crossing, and its resistance derives from *C. ecuadorensis* (Robinson and Reiners 1999).

Inheritance of PRSV-W resistance has been elucidated in other cucurbit crops, such as muskmelons (Pitrat and Lecoq 1983) and watermelons (Azevedo et al. 2012, Alves et al. 2014). Resistance in *C. maxima* and *C. moschata* is controlled by more than one gene. Kuabara et al. (1987) studied the inheritance of resistance to PRSV-W in *C. maxima*, and suggested control by at least two recessive alleles in different loci. The resistance of the line 'Varzea

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Alegre' (*C. maxima*) is controlled by one or few genes with additive effect (Maluf et al. 1985). Inheritance of PRSW-V resistance was studied in two *C. maxima* resistant accessions (ABL-010 and Redlands Trailblazer). Both accessions were crossed with Buttercup, which is a standard cultivar of susceptibility to PRSV. Resistance presented in Redlands Trailblazer is controlled by at least two genes with additive effects. ABL-010 resistance can be explained by the action of three genes with partial dominance. Susceptible plants were found in the segregating population of the cross between ABL-010 x Redlands Trailblazer (transgressive segregation), indicating that at least one of the loci involved in the control of the resistance in ABL-010 was not allelic to their counterpart loci in Redlands Trailblaizer (Maluf et al. 1997). 'Baiana Tropical' (*C. moschata*) resistance was found to be controlled by two or three genes with additive effects (Oliveira et al. 2003).

Even though PRSV-W resistance in *C. pepo* 'Whitaker' has been reported (Nogueira et al. 2011), its mode of inheritance has not been established yet. This study reports on the mode of inheritance of PRSV-W resistance in Whitaker *C. pepo* line.

MATERIAL AND METHODS

The experiment was carried out in the greenhouses of the Vegetable Research Station of HortiAgro Sementes S.A., Ijaci (lat 21^0 09' 24'' S, long 44^0 55' 34'' W, alt 833m asl), MG, Brazil. Whitaker (P_1) and Caserta (P_2) were used as parents in a cross designed to study the inheritance of PRSV-W resistance. Whitaker is a line reportedly resistant to three viruses: *Papaya ringspot virus – watermelon strain* (PRSV-W), *Cucumber mosaic virus* (CMV), *Zucchini yellow mosaic virus* (ZYMV). Whitaker resistance to PRSV-W is derived from *C. ecuadorensis* (Robinson and Reiners 1999). Caserta is a PRSV-W susceptible *C. pepo* standard cultivar widely grown in Brazil. F_1 (Whitaker x Caserta) plants were both self-pollinated to produce F_2 generation, and crossed to both parents to produce the reciprocal backcross families BC_{11} (Whitaker x F_1) and BC_{12} (Caserta x F_1).

Plants of the different generations were grown in soil in beds placed in a randomized design with three replications, totaling 45 Whitaker plants, 90 Caserta plants, 90 F_1 plants, 360 F_2 plants, 135 BC_{11} plants (Whitaker x F_1) and 180 BC_{12} plants(Caserta x F_1), which were evaluated for their reactions to mechanical inoculation with PRSV-W.

A Brazilian isolate of PRSV-W (identified at the Department of Plant Pathology of Universidade Federal de Lavras, Lavras, MG, Brazil) was multiplied in *C. pepo* cv. Asmara. Just before inoculation, inoculum was prepared by grinding infected leaves with mortar and pestle in 0.01M phosphate buffer, pH 7.0, with 0.1% sodium sulfite. Buffer x leaf ratio was 9:1 (9 mL of buffer for every 1 gram of infected leaf). Two inoculations were carried out: the first when the seedlings were nine days old, and the second, twelve days after the first one. Inoculation consisted of lightly dusting the cotyledonary leaves with 400-mesh carborundum, and then mechanically rubbing the inoculum with the forefinger. After inoculation, carborundum was rinsed off of the leaves with water.

Plant symptoms were rated at 10, 17 and 24 days after inoculation, using a scores from 1 to 5, adapted from Oliveira et al. (2003), as follows: 1 = no visible symptoms; 2 = most leaves with no symptoms; one or a few leaves with mild symptoms, mostly clear veins; 3 = most leaves with mosaic; symptoms varying from vein clearing to sparse chlorotic spot to chlorosis in up to 50% of the leaf area; 4 = almost all leaves with systemic mosaic coalescence of chlorotic areas, reaching up to 50% of the leaf area; 5 = almost all leaves with severe mosaic, at least one leaf with more than 50% of its area affected or severely distorted.

Means and variances were calculated for each one of the six populations, in order to calculate genetic parameters. Environmental variance ($\hat{\sigma}^2_E$) was estimated as the geometric mean of the variances of P_1, P_2 and F_1 generations. Genetic variance ($\hat{\sigma}^2_G$), and its additive ($\hat{\sigma}^2_A$) and dominance ($\hat{\sigma}^2_D$) components, as well as broad sense heritability (H^2) were estimated (Mather and Jinks 1977). Generation mean analysis was carried out based on the data by the weighted least squares method (Mather and Jinks 1977), in order to test the fitness of a simple additive-dominant model, and to estimate the mean degree of dominance (MDD).

Data were used to test a hypothesis of monogenic inheritance under different presumed degrees of dominance, as described by Gomes et al. (2000) and Menezes et al. (2005).

A truncation point (TP) was chosen in the symptom scale, so that most of P_1 plants were below the TP and most of P_2 plants were above it. The TP used was score 2.0. Hypothesis of monogenic inheritance was tested following some suppositions and procedures:

a) Data from all generations (P_1, P_2, F_1, F_2, BC_{11} and BC_{12}) have a normal distribution.

b) Means and variances of P_1 and P_2 are equal to the respective estimates obtained from the experimental data.

c) Based on normal distribution, frequencies of P_1 and P_2 plants with scores equal or lower than the truncation point were estimated.

d) The mean of F_1 generation was admitted as being: $\overline{F}_1 = (\overline{P}_1 + \overline{P}_2)/2 + MDD(\overline{P}_2 - \overline{P}_1)/2$, where $\overline{P}_1$ and $\overline{P}_2$ are the respective parental means, and MDD is the mean degree of dominance presumed.

(e) The variance of the F_1 population is equal to the respective variance estimate obtained from the experimental data.

f) The expected frequencies of F_2, BC_{11} and BC_{12} population, based on a monogenic model of inheritance, were estimated as functions of P_1, P_2 and F_1 frequencies, as follows:

$F_2 = (P_1 + 2F_1 + P_2)/4$; $BC_{11} = (P_1 + F_1)/2$ and $BC_{12} = (P_2 + F_1)/2$.

g) The frequencies of P_1, P_2, F_1, F_2, BC_{11} and BC_{12} plants £ TP were calculated by multiplying the expected frequencies by the total number of plants tested per generations.

g) Expected numbers of plants £ TP were compared with their respective observed values in each generation. The significance of the deviations was estimated with a χ^2 test, with 4 degrees of freedom. The frequency of expected plants in P_1 was added to that of P_2, in order to avoid expected frequencies equal to zero.

h) Significant χ^2 values would lead to the rejection of the hypothesis of monogenic inheritance under the degree of dominance presumed. On the other hand, a non-significant χ^2 value would lead to non-rejection of such hypothesis. Values of χ^2 for each simulation were plotted against their respective hypothetical MDD's. The interval of MDD values of which χ^2 values are below the a=0.05 critical value represents the MDD interval in which the monogenic hypothesis was not rejected.

Some genetic models were tested using maximum likelihood in mixtures of normal densities, as proposed by Gonçalves et al. (2004) and Rezende et al. (2004). Based on the means and variances components (Mather and Jinks 1977), data were subjected to a normal distribution, as follows:

$$P_1 : N(\mu - [a] - A, \sigma^2)$$

$$P_2 : N(\mu - [a] + A, \sigma^2)$$

$$F_1 : N(\mu - [d] - D, \sigma^2)$$

$$F_2 : \frac{1}{4}N\left(\mu + \frac{[d]}{2} - A, \sigma^2 + V_A + V_D\right) + \frac{1}{2}N\left(\mu + \frac{[d]}{2} + D, \sigma^2 + V_A + V_D\right) + \frac{1}{4}N\left(\mu + \frac{[d]}{2} + A, \sigma^2 + V_A + V_D\right)$$

$$BC_{11} : \frac{1}{2}N\left(\mu + \frac{[a]}{2} + \frac{[d]}{2} - A, \sigma^2 + \frac{V_A}{2} + V_D - S_{AD}\right) + \frac{1}{2}N\left(\mu - \frac{[a]}{2} + \frac{[d]}{2} + D, \sigma^2 + \frac{V_A}{2} + V_D + S_{AD}\right)$$

$$BC_{12} : \frac{1}{2}N\left(\mu + \frac{[a]}{2} + \frac{[d]}{2} + A, \sigma^2 + \frac{V_A}{2} + V_D + S_{AD}\right) + \frac{1}{2}N\left(\mu + \frac{[a]}{2} + \frac{[d]}{2} + D, \sigma^2 + \frac{V_A}{2} + V_D + S_{AD}\right)$$

where:

μ: Constant of reference

A: Additive effect of the major gene

D: Dominance effect of the major gene

[*a***]**: Polygenic additive effect

[*d*]: Polygenic dominance effect

V_A: Additive variance

V_D: Dominance variance associated with polygenic effects

S_{AD}: Additive x dominance deviation associated with polygenic effects

σ^2: Environmental variance

Normal distributions of BC_{11} and BC_{12} are composed by two normal densities and of F_2 by three normal densities.

Tests using maximum likelihood were made via LR (Gonçalves et al. 2004):

$$LR = -2ln\frac{L(M_i)}{L(M_j)},$$

where $L(M_i)$ and $L(M_j)$ are maximum likelihood functions of the models i and j, and model i should be hierarchal to model j.

For the analyses, the full genetic model admitted a major gene with additive and dominance effects, and polygenes, also with additive and dominance effects. From the complete genetic model, simpler models containing less parameters were generated (Table 1). Environmental variances were considered equal for all generations, and gene segregation was considered independent (both major genes and polygenes). Hypothesis tests of the genetic parameters were carried out based on likelihood ratio between two models (Gonçalves et al. 2004). The tests were carried out using the statistical software 'Monogen v.0.1'.

RESULTS AND DISCUSSION

Fast evolution of symptoms was observed from the first to the last evaluation (Tables 2 and 3). Some 'Whitaker' plants showed sparse chlorotic spots, due to the high virus concentration and multiplication. By that time, all susceptible plants had already died. General average score for Whitaker

was 2.0, whereas cultivar Caserta had already showed a score of about 4.0 at the first evaluation date. These data show the contrasting levels of resistance of the two parental lines involved. All Caserta plants died with severe viral symptoms, while those of Whitaker grew vigorously and remained almost totally symptom-free (Table 2).

Infected leaves of Caserta and Whitaker were used as inoculum in back inoculation in Caserta plants, in order to check for virus recovery. Caserta plants back inoculated with inocula from both parental lines presented PRSV-W symptoms, indicating that the mechanism of resistance to PRSV-W imparted by Whitaker is probably tolerance.

Table 1. Genetic inheritance models according to Rezende et al. (2004) tested for resistance to PRSV-W in summer squash

Models	Estimated parameters
1 = major gene with additive and dominance effects + polygenes with additive and dominance effects	μ, A, D, $[a]$, $[d]$, V_A, V_D, S_{AD}, σ^2
2 = major gene with additive and dominance effects + polygenes with only additive effect	μ, A, D, $[a]$, V_A, σ^2
3 = major gene with only additive effect + polygenes with additive and dominance effects	μ, A, $[a]$, $[d]$, V_A, V_D, S_{AD}, σ^2
4 = major gene with only additive effect + polygenes with only additive effect	μ, A, $[a]$, V_A, σ^2
5 = polygenes with additive and dominance effects	μ, $[a]$, $[d]$, V_A, V_D, S_{AD}, σ^2
6 = polygenes with only additive effect	μ, $[a]$, V_A, σ^2
7 = major gene with additive and dominance effects	μ, A, D, σ^2
8 = major gene with only additive effect	μ, A, σ^2
9 = only environmental effects	μ, σ^2

Table 2. Means and variances for resistance to PRSV-W in summer squash (*Cucurbita pepo*), in three evaluation dates (10, 17 and 24 days) after inoculation

	10 days		17 days		24 days	
Generation	**Mean**	**Variance**	**Mean**	**Variance**	**Mean**	**Variance**
Whitaker	1.07	0.0623	1.35	0.4097	1.63	0.5493
Caserta	3.97	1.4483	4.77	0.3607	4.96	0.0879
F_1	2.53	1.6080	3.50	1.5862	4.77	0.2926
F_2	3.29	1.2199	4.30	0.7721	4.84	0.2710
RC_{11} (Whitaker x F_1)	1.46	0.7092	2.60	1.6782	3.86	1.9285
RC_{12} (Caserta x F_1)	3.50	1.4022	4.49	0.8323	4.94	0.0800

Table 3. Frequencies of plant symptom scores in Whitaker, Caserta, and generations F_1, F_2, BC_{11} and BC_{12}, in three evaluation dates (10, 17 and 24 days) after inoculation with PRSV-W

Generation	TNP	10 days (%)					17 days (%)					24 days (%)				
		Score					Score					Score				
		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Whitaker	45	42	3	0	0	0	33	8	4	0	0	23	15	7	0	0
Caserta	90	7	3	15	26	39	0	2	2	11	75	0	0	2	0	88
F_1	90	32	9	22	26	1	10	9	15	37	19	0	0	5	10	75
F_2	360	46	20	102	168	24	5	13	32	130	180	1	1	16	17	325
BC_{11} (Whitaker x F_1)	135	98	18	13	6	0	41	18	35	34	7	15	8	21	25	66
BC_{12} (Caserta x F_1)	180	23	7	34	89	27	5	5	7	43	120	0	0	2	7	171

TNP: Total Number of plants

Table 4. Mean components, mean degree of dominance (MDD) and broad-sense heritability (H^2) for PRSV-W symptom expression in summer squash (*Cucurbita pepo*), in three evaluation dates (10, 17 and 24 days) after inoculation with PRSV-W

Parameters	10 days	17 days	24 days
m	2.7343 ± 0.1487	3.2434 ± 0.1645	3.9149 ± 0.2073
$[a]$	1.6837 ± 0.1422	1.6291 ± 0.1543	1.0401 ± 0.1916
$[d]$	0.1156 ± 0.6436	1.2726 ± 0.7319	1.2026 ± 0.5228
χ^2	0.2720	0.3781	0.7068
MDD	0.0687	0.7811	1.1562
$H^{2(\%)}$	56.92	20.15	10.80

m : parental mean
$[a]$: additive mean effect
$[d]$: non-additive (dominance) mean effect
χ^2: chi-square test for fitness of the additive-dominant model

Broad-sense heritability estimates were not uniform among the evaluations, decreasing from the first to the third evaluation (Table 4). Selection of resistant plants is expected to be effective 10 days after inoculation (H^2= 56.92%), but less effective 17 (H^2= 20.15%) or 24 (H^2= 10.80) days after inoculation, indicating the need for better control of environmental effects in the latter evaluations. These heritability estimates are lower than those reported for resistance to PRSV-W in *C. maxima* and *C. moschata*. Maluf et al. (1985), studying the resistance of the cultivar 'Varzea Alegre' (*C. maxima*), found broad and narrow-sense heritabilities of 0.58 and 0.57, respectively. Maluf et al. (1997), working with *C. maxima* genotypes, obtained broad-sense heritabilities from 0.36 to 0.59. Oliveira et al. (2003), studying *C. moschata* cultivars, observed broad-sense heritabilities from 0.39 to 0.97.

A simple additive-dominant model was able to explain the segregation data (Table 4). Since no significant deviations from the proposed model were observed by the χ^2 test, it is supposed that there is no evidence of epistatic gene effect involved in the control of resistance to PRSV-W in 'Whitaker' line. Estimates of the mean degrees of dominance were not uniform among the evaluations (Table 4). On the first evaluation, data indicated the predominance of additive gene effects, whereas on the second and third evaluations, data indicated the predominance of dominance or partial dominance gene effects in the direction to susceptibility to PRSV-W. Maluf et al. (1997), working with *C. maxima,* evaluated two sources resistant to PRSV-W, and found that the resistance of ABL-10 could be explained by the action of three genes with partial dominance, and the resistance of Redlands Trailblazer was due to at least two genes with additive effects.

The estimates of χ^2 to monogenic inheritance hypothesis could not be accepted for mean degree of dominances presumed between –1.0 and +1.0, indicating that 'Whitaker' line's resistance to PRSV-W is controlled by more than one gene (Figure 1). Frequencies distribution presented by F_2 and BCs were very different from what is expected from a monogenic control (Table 3). More than 80% of the F_2 and BC_{12} plants presented scores equal to or higher than 4.0 at

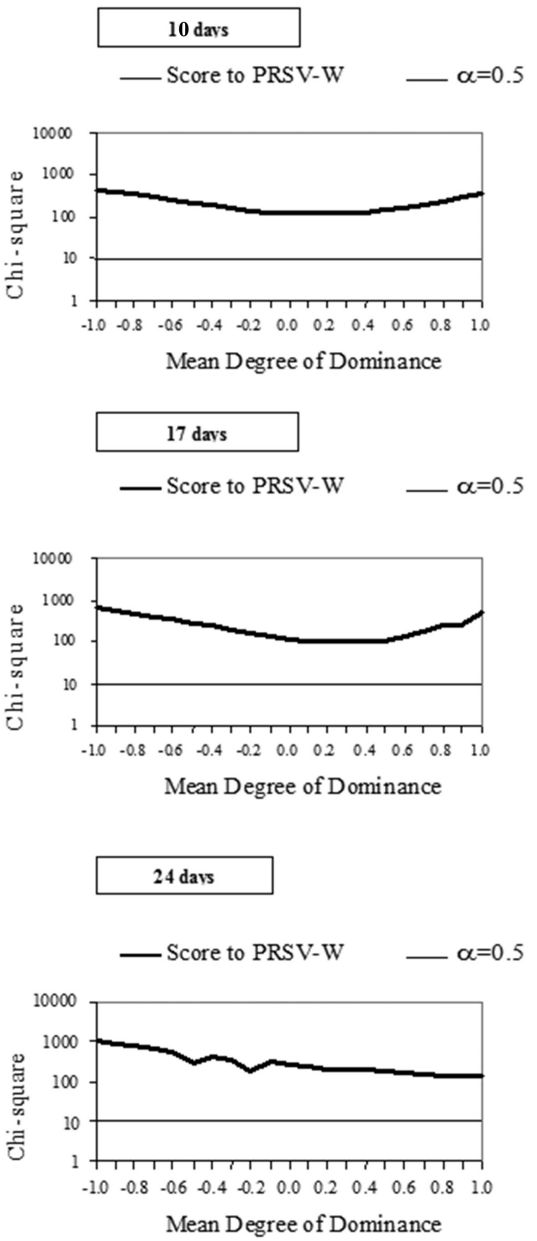

Figure 1. Monogenic hypothesis test under different presumed degrees of dominance of PRSV-W resistance in summer squash (*Curcubita pepo*), on three different evaluation dates (10, 17 and 24 days after inoculation).

Table 5. Hypotheses of inheritance tested by using maximum likelihood for resistance to PRSV-W in summer squash, in three evaluations (10, 17 and 24 days after inoculation)

Models	Degrees of freedom	χ^2_c	Prob.	χ^2_c	Prob.	χ^2_c	Prob.
		10 days		17 days		24 days	
1 vs. 5	5	133.64	0.0000	67.17	0.0000	415.05	0.0000
1 vs. 7	5	134.52	0.0000	13.01	0.0233	135.29	0.0000
5 vs. 9	5	328.17	0.0000	420.39	0.0000	841.028	0.0000
7 vs. 9	2	327.29	0.0000	474.56	0.0000	1120.79	0.0000

the last evaluation. Only two F_2 plants were found to be resistant (Table 3).

Inheritance tests carried out with maximum likelihood tests are presented in Table 5. When model 1 is confronted to model 5, the existence of a major gene summed to polygenic effects is compared to the occurrence of polygenic effects only. The test of this hypothesis was rejected, which means that there is an effect of a major gene in the control of the trait. When model 1 is confronted to model 7, the existence of a major gene summed to polygenic effect is compared to a model that assumes only a major gene effect. This hypothesis was also rejected, indicating the evidence of polygenic effects too. Tests confronting model 5 to model 9, and model 7 to model 9 (Table 5) reinforce that the control of PRSV-W resistance in *C. pepo* 'Whitaker' is more complex than what is expected from a single major gene. There are both major gene effect and polygenic effects in the control of resistance to PRSV-W in *Cucurbita pepo* 'Whitaker'.

Herança da resistência ao vírus da mancha anelar do mamoeiro estirpe melancia (PRSV-W) na linhagem de abobrinha 'Whitaker'

Resumo – *O objetivo do trabalho foi estudar o controle genético da resistência ao virus da mancha anelar do mamoeiro estirpe melancia (PRSV-W) na linhagem de abobrinha 'Whitaker'* Cucurbita pepo*. Plantas das linhagens parentais Whitaker (resistente) e Caserta (suscetível) e das gerações F_1, F_2, RC_{11} e RC_{12} foram avaliadas quanto aos sintomas causados pelo PRSV-W. Plantas da linhagem Caserta apresentaram sintomas severos de mosaico, enquanto as plantas de Whitaker permaneceram quase totalmente livres de sintomas. A maioria das plantas F_1, F_2 e dos retrocruzamentos também apresentaram sintomas severos de mosaico. Foi realizado teste de hipótese de herança monogênica sob a suposição de diferentes graus de dominância e foram testados modelos genéticos por meio de testes de máxima verossimilhança. A estimativa da herdabilidade no sentido amplo foi de 0,57 para a primeira avaliação. A resistência ao PRSV-W em* C. pepo *'Whitaker' é controlada por um gene principal mais a acão de poligenes.*

Palavras-chave: Cucurbita pepo*, controle genético, potyvirus, resistência a virus.*

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