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Full Length Research Paper

Genetic analysis of resistance of wild melon to *Podosphaera xanthii* race 2F

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Yuntian-930 is a wild melon that is highly resistant to powdery mildew (PM). To determine the inheritance of resistance to PM, Yuntian-930 was crossed with another cultivated melon, Hualaishi, which is susceptible to PM. Inheritance of resistance to *Podosphaera xanthii* race 2F. in six plant generations (P_1, P_2, F_1, B_1, B_2, and F_2) was studied using the mixed major-gene plus polygene inheritance mode with joint analysis method. Inheritance of resistance in wild melons fitted the model of two pairs of additive-dominance-epistasis major genes plus additive-dominant-epistasis polygene. The additive, dominant and epistatic effects between the two major genes played an important role in inheritance. The estimated values of heritability of major genes in B_1, B_2 and F_2 were 62.98, 58.58 and 90.89% and those of polygene heritability were 28.93, 31.47 and 3.22%, respectively. The ratios of the environmental variance to phenotype variance were 5.89 to 9.94%. Thus, resistance of Yuntian-930 to PM was controlled not just by two major genes, but was also affected by polygene and the environment. The selection efficiency for a major gene of F_2 was highest in the resistance breeding program.

Key words: Wild melon, powdery mildew, resistance, *P. xanthii* race 2F, inheritance.

INTRODUCTION

Growth of powdery mildew (PM) in melons (*Cucumis melo* L.) occurs throughout the world in all seasons and cultivation systems. It is the main obstacle to green production and causes much loss of produce. Various germplasm resources in melons having resistance to PM are known. An effective way to control PM is to breed PM-resistant melon cultivars. Germplasm resources for melon PM resistance and genetic patterns of the resistance have been widely studied. However, due to the diverse resistance of melons to pathogens, findings on the inheritance of resistance of melons to PM vary. Many investigations suggest that resistance of melons to PM is controlled by a single dominant gene or an incomplete dominant gene (Cohen et al., 1986; Epinat et al., 1993; Harwood et al., 1968; Teixeira et al., 2008; Wang et al., 2005; Zhang et al., 2008). However, the resistance genes involved are different, since the melon varieties and physiologic types studied are different. For instance, the inheritance of resistance to race 1 of *Podosphaera xanthii* in the melon PMR 45 is controlled by the dominant gene, *Pm-A*. The resistance of the cultivar Nantais oblong toward *Golovinomyces cichoracearum* is controlled by the single dominant gene, *Pm-H* (Epinat et al., 1993). The resistance of AF426-R and K7-1 to *P. xanthii* race 1 (Teixeira et al., 2008) and *P. xanthii* race 2F. (Zhang et al., 2008), respectively, are governed by the single dominant genes, *Pm-1* and *Pm-2F*, respectively.

The single dominant genes, *Pm-4* and *Pm-5*, control the resistance of the Seminole cultivar toward *P. xanthii* (Harwood et al., 1968). Resistance of PI 124111 to races 1 and 2 of *P. xanthii* is controlled by the single dominant gene *Pm-3* and the partial dominant gene *Pm-6*, but the two genes are not linked to each other (Cohen et al., 1986). Resistance of PI 124111 to PM is controlled by a pair of incomplete dominant genes (Wang et al., 2005), and resistance of A120 to PM is controlled by a pair of recessive genes (Zhao et al., 2009). Many scholars believe that resistance of melons to PM is jointly controlled by several genes (Bohn et al., 1964; Cheng,

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Abbreviations: PM, Powdery mildew; **JSA,** joint segregation analysis; **AIC,** akaike's information criterion; **DI,** disease indexes; **QTL,** quantitative trait locus.

2006; Kenigbuch et al., 1992; Pryor, 1942; Yuste-Lisbona et al., 2010). For example, resistance of melons to *P. xanthii* race 2 is jointly controlled by at least two incomplete dominant genes (Pryor, 1942), or by the incomplete dominant gene *Pm-2* and two modifier genes with epistatic effect to *Pm-2* (Bohn et al., 1964); alternatively, it may also be controlled by several dominant, recessive, and modifier genes (Kenigbuch et al., 1992), or by two major genes with additive-dominance-epistasis effect (Cheng, 2006). Resistance of TGR-1551 to races 1, 2, and 5 of *P. xanthii* is governed by two independent genes (one dominant and one recessive), which means that the genetic control involves dominant-recessive epistasis (Yuste-Lisbona et al., 2010). It is thus clear that inheritance of resistance of melons to PM is evidently complex. Since the specific strain of PM has not been identified in some studies, the genetic basis of resistance to PM in melons cannot be effectively utilized; this hinders gene pyramiding. Gai and Wang (1998) and Gai et al. (2003) proposed the joint segregation analysis (JSA) of multiple populations for mixed major genes with polygene inheritance. Through this design, the genetic effects of a major gene, the collective genetic effects of polygenes, and their heritability values may be evaluated. Moreover, the parameter estimation algorithm for the JSA model has been improved through an expectation and iterated maximization method (Zhang et al., 2003).

The best-fitting genetic model is primarily selected based on the Akaike's information criterion (AIC) (Akaike, 1977; Knott et al., 1991). JSA has been applied to the analysis of inheritance traits in various types of crops and vegetables, including rice (Wang et al., 2000; Xue et al., 2007; Zhu et al., 2008), wheat (Ge et al., 2004; Hou et al., 2006), maize (Zhan et al., 2001), soya bean (Cao and Yang, 2002; Wang and Gai, 1997, 2001), cotton (Hao et al., 2008; Yuan et al., 2002; Zhang et al., 2006), rapeseed (Qi et al., 2001; Tian et al., 2009; Xu et al., 2010; Zhang et al., 2008), chickpea (Anbessa et al., 2006), broccoli (Liu et al., 2009), eggplant (Pang et al., 2008), pepper (Chen et al., 2006; Chen and Chen, 2006), tomato (Li et al., 2006), Chinese cabbage (Zuo et al., 2009), cucumber (Zhang et al., 2007) and melon (Cheng, 2006; Zhang et al., 2009). Wild melon, Yuntian-930 (P_1), from Yunnan (China), is a stable germplasm resource that is highly resistant to PM. It has been bred by the Watermelon and Melon Research Group of Northwest A and F University through many years. In the current study, it was crossed with Hualaishi (P_2), melon cultivar susceptible to PM. Inheritance of resistance of all six plant generations (P_1, P_2, F_1, B_1, B_2, and F_2) to *P. xanthii* race 2F. were studied, using the mixed major-gene plus polygene inheritance mode with joint analysis method of multiple generations. The objectives of the current paper were to elucidate the genetic mechanisms of PM resistance of Yuntian-930, and to obtain PM resistance germplasm resources and the theoretical basis of selection of the resistance gene.

The present study also aimed to enrich the knowledge base of hereditary laws of disease resistance breeding, and to provide basic information for breeding PM-resistant melon cultivars.

MATERIALS AND METHODS

Experimental materials

Yuntian-930 (P_1, highly resistant to PM) and Hualaishi (P_2, susceptible to PM), were the parent plants. Both were inbred lines. F_1 was obtained by the cross, $P_1 \times P_2$. F_2 was obtained by self-pollination of F_1. B_1 was the cross $F_1 \times P_1$, and B_2 was from $F_1 \times P_2$. Using the phenotypic information from the six generations, the genetic law of resistance of Yuntian-930 to PM was analyzed. The PM was collected from infected leaves of melons in the greenhouse of Xintiandi Facilities Limited (Yangling and Shaanxi). The mildew was identified as *P. xanthii* race 2F, using 13 melon differential hosts (Hosoya et al., 2000; James, 1994; Kenigsbuch and Cohen, 1992; Křístková et al., 2004).

Experimental methods

All six generations were cultivated in the horticultural facilities of Northwest A and F University in April, 2009. Plants were grown under normal conditions, and no chemical agents were used. When the plants grew five true leaves, the plants were made susceptible to disease by inoculating them with PM and subsequently cultivating them at 25 to 30°C/20 to 25°C (day/night) and relative humidity of 70 to 80%. Plants were sufficiently susceptible 15 d after inoculation; at this point, five leaves of each plant were examined from the base of the stem. The investigation plant numbers of P_1, P_2, F_1, B_1, B_2, and F_2 were 15, 15, 15, 50, 50 and 104, respectively.

PM evaluations

Disease grading standards were the following (Cheng, 2006; Zhang et al., 2008): class 0, no symptoms; class 1, area with diseased spots (fuzzy white powder) is 1/3 of the total leaf area; class 2, area with diseased spots (clear white powder) is 1/3 to 2/3 of the total leaf area; class 3, area of diseased spots (contiguous thick white powder) is > 2/3 of the total leaf area; class 4, thick white powder, leaf yellowing and necrosis; and class 5, necrotic leaf area is > 2/3 of the total leaf area. The disease indexes (DI) of PM (Greenhouse) were defined as follows: high resistance, $0 < DI \leq 25$; resistance, $25 < DI \leq 45$; middle resistance, $45 < DI \leq 65$; susceptibility, $65 < DI \leq 80$; and high susceptibility, $DI > 80$.

Genetic analysis

The details of JSA were described in several articles (Anbessa et al., 2006; Gai et al., 1998, 2003; Hao et al., 2008; Wang and Gai, 2001; Zhang et al., 2010). The basic assumptions in JSA are the following: 1) diploid nuclear inheritance involves no maternal or cytoplasmic effects, no interaction or linkage between major genes and polygenes, and no selection; 2) the effects of polygenes and the environment in any segregating population follow a normal distribution; and 3) the variances within the two homozygous parents (P_1 and P_2) and the F_1 populations are equal (Gai et al., 2003). Multiple genes with different effects may control a quantitative trait. The gene with a large (or small) effect is considered as a major (or minor) gene. In the mixed inheritance model and JSA, the trait variation in each segregating population is

Table 1. The frequency distribution of powdery mildew disease index for six generations.

Generation	Frequency					Numbers	Mean disease index
	≤25	25–45	45–65	65–80	>80		
P_1	14	1				15	17.07 ± 6.50^{fF}
F_1				8	7	15	81.73 ± 5.21^{bB}
P_2				2	13	15	94.13 ± 5.21^{aA}
B_1	10	20	18	2		50	39.28 ± 19.66^{eE}
B_2		3	16	24	7	50	70.24 ± 30.64^{cC}
F_2	5	48	8	34	9	104	55.96 ± 22.39^{dD}

Small letters indicate 5% significant differences and capital letters denote 1% significant differences.

assumed to be the result of variation in the distribution of major gene(s) modified by polygenes and the environment. The joint analyses of P_1, P_2, F_1, B_1, B_2, and F_2 may involve one or more of the following genetic models: one major gene (model A), two major genes (model B), pure polygenes (model C), one major gene plus polygenes (model D), and two major genes plus polygenes (model E). First, the AIC value was used to select the best-fit model. Here, $AIC=-2L_c(\Phi)+2N$, in which $L_c(\Phi)$ is the maximized log-likelihood and N is the number of independent parameters in the model.

The model with the smallest AIC value is chosen as the best-fit model. The model that best explained the variation of a quantitative trait was analyzed using x^2, Smirnov, and Kolmogorov statistics. If no significant difference was found, the model with the smaller parameter value will be chosen. Finally, estimates of the genetic parameters were calculated from the estimates of component distributions in the best-fit genetic model. Data for additive, dominant, and epistatic effects, and genetic variances and heritability for major gene(s) and polygenes were generated. The program for segregation analysis of the single and multiple generations was compiled with Turbo C^{++} [available on request; (http://jpkc.njau.edu.cn/swtj/)]. For the mixed inheritance model, the phenotypic value (p) could be expressed as $p = m + g + c + e$, where m, g, c, and e represent the population means, major gene effects, polygene effects, and environmental effects, respectively. The value of g varies with major gene genotypes, and c and e are normally distributed variables. Thus, the phenotypic variation (σ_p^2) could be expressed as major gene (σ_{mg}^2), polygene (σ_{pg}^2), and environmental (σ^2) variations. The phenotypic variation is defined as $\sigma_{pg}^2 = \sigma_p^2 - \sigma_{mg}^2 - \sigma^2$; the heritability of the major gene is $h_{mg}^2 = (\sigma_{mg}^2/\sigma_p^2) \times 100\%$, where $h_{pg}^2 = (\sigma_{pg}^2/\sigma_p^2) \times 100\%$.

RESULTS

Frequency distributions of resistance to PM

The frequency distributions of the DI values in the six populations are listed in Table 1. The DI of P_1 is 17.07 ± 6.50, which denotes high resistance. The DIs of P_2 and F_1 are 94.13 ± 5.21 and 81.73 ± 5.21, respectively, which suggest high susceptibility. The difference between DI values of P_1 and P_2 is highly significant. The value of F_1 is close that of the susceptible parent P_2; this indicates that resistance of Yuntian-930 to PM is recessive. The DIs of B_1, B_2, and F_2 demonstrate continuous skewing or a bimodal distribution, and do not fit the normal distribution, which indicate that the genetic resistance of Yuntian-930 to PM is consistent with the model mixed major gene plus polygenes.

Selection and testing of genetic models

Based on the mixed major gene plus polygene inheritance mode with joint analysis (Gai et al., 1998), the best-fitting genetic model was chosen according to the smallest AIC values. The calculated AIC values in each genetic model are listed in Table 2. The data indicate that the E-0 and E-1 models had the smallest AIC values among the 24 models. E-0 had the lowest AIC value, and E-1 had the second lowest value. Therefore, E-0 and E-1 were judged as the best-fitting genetic models and were subjected to further analysis. To select the model that best explains the genetic variation, the uniform, Smirnov, and Kolmogorov tests were applied to determine the goodness-of-fit of the E-0 and E-1. Their result was the same, which two statistics in the E-0 and E-1 indicated a significant difference (Table 3), but E-0 had the minimum AIC value (Table 2). Therefore, the E-0 model was the genetic model with the best fit (Table 3). Additionally, the results indicate that the resistance of Yuntian-930 to PM was dominated by a model involving two additive-dominance-epistasis major genes and additive-dominance-epistasis polygenes (E-0).

Estimation of genetic parameters

The first-order and second-order genetic parameters of the E-0 model and the components in each population are shown in Table 4. The additive effects of two major genes were equal ($d_a = d_b - 16.95$), and resulted in lower DI and higher resistance. The dominant effect and potential ratio of the first major gene were 20.62 and −1.22, respectively. In addition, the dominant effect and potential ratio of the second major gene were 24.44 and −1.44, respectively. The dominant effects of these two major genes were greater than their additive effects which had a negative super dominant effect and lowered resistance. The epistatic effect of additive × additive between the two major genes was 0.22 and lowered

Table 2. The maximum log-likelihood values and AIC values under various genetic models estimated through the iterated ECM (IECM) algorithm.

Model	Max-likelihood-value	AIC	Model	Max-likelihood-value	AIC
A-1	-1101.98	2211.97	D-0	-1044.41	2112.81
A-2	-1104.32	2214.63	D-1	-1052.13	2122.27
A-3	-1137.41	2280.83	D-2	-1052.13	2120.27
A-4	-1109.74	2225.49	D-3	-1052.59	2121.18
B-1	-1032.20	2084.40	D-4	-1052.32	2120.65
B-2	-1067.81	2147.62	E-0	-1021.90	2079.81*
B-3	-1114.36	2236.71	E-1	-1026.80	2083.61*
B-4	-1092.89	2191.77	E-2	-1068.95	2159.91
B-5	-1131.52	2271.04	E-3	-1039.62	2097.24
B-6	-1131.52	2269.04	E-4	-1065.31	2146.63
C-0	-1047.12	2114.23	E-5	-1073.89	2165.78
C-1	-1076.94	2167.89	E-6	-1072.65	2161.30

*AIC values of the best-fitting genetic models.

Table 3. Tests for goodness-of-fit of models E-0 and E-1.

Model	Population	U_1^2	U_2^2	U_3^2	nW^2	D_n
E-0	P_1	0.143(0.7057)	0.269(0.6037)	0.376(0.5396)	0.201(>0.05)	0.302(>0.05)
	F_1	0.000(0.9983)	0.003(0.9546)	0.056(0.8135)	0.0759(>0.05)	0.177(>0.05)
	P_2	0.057(0.8113)	0.015(0.9035)	0.193(0.6604)	0.1159(>0.05)	0.2166(>0.05)
	B_1	0.002(0.9642)	0.042(0.8371)	0.421(0.5164)	0.0635(>0.05)	0.0884(>0.05)
	B_2	0.216(0.6423)	0.030(0.8631)	1.230(0.2673)	0.1966(>0.05)	0.1673(>0.05)
	F_2	2.687(0.1012)	2.478(0.1154)	0.003(0.9585)	0.5149(<0.05)*	0.157(<0.05)*
E-1	P_1	0.020(0.8872)	0.004(0.9500)	0.089(0.7653)	0.1665(>0.05)	0.2493(>0.05)
	F_1	0.013(0.9105)	0.030(0.8619)	0.068(0.7944)	0.077(>0.05)	0.1668(>0.05)
	P_2	0.470(0.4932)	0.349(0.5545)	0.084(0.7718)	0.164(>0.05)	0.2606(>0.05)
	B_1	3.609(0.0575)	3.138(0.0765)	0.074(0.7852)	0.4041(>0.05)	0.1736(>0.05)
	B_2	0.030(0.8627)	0.651(0.4196)	6.545(0.0105)*	0.3318(>0.05)	0.2323(<0.05)*
	F_2	1.657(0.1980)	1.572(0.2100)	0.001(0.9773)	0.3781(>0.05)	0.1302(>0.05)

U_1^2, U_2^2, and U_3^2 are the statistics of the uniformity test; nW^2 is the statistic of the Smirnov test; D_n is the statistic of the Kolmogorov test. The asterisk (*) indicates different significance at $P < 0.05$.

resistance. The epistatic effects of dominant×dominant, additive × dominant, and dominant × additive between the two major genes were –35.31, –8.11, and –0.46, respectively, and all promoted resistance. Clearly, the additive, dominance, and epistatic effects between the two major genes played an important role in inheritance. In B_1, B_2, and F_2, the estimated heritabilitiy of the major genes were 62.98, 58.58 and 90.89%, respectively, and the estimated polygene heritabilitiy were 28.93, 31.47 and 3.22%, respectively. The ratios of the environmental variance to phenotype variance were 5.89 to 9.94%. Thus, the resistance of Yuntian-930 to PM may be controlled by two major genes, polygene, and the environment. The selection efficiency for the major gene of F_2 was highest in the resistance breeding program.

DISCUSSION

Many genetic laws on resistance of melons to PM have been reported, but they have remained ambiguous. Moreover, the physiological type has been identified in some studies only. Using the mixed major-gene plus polygene inheritance mode with joint analysis method of multiple generations, inheritance of resistance of Yuntian-930 to *P. xanthii* race 2F. was found to fit the additive-dominance-epistasis major genes plus additive-dominant-epistasis polygene model (E-0 model); this model describes the important role of additive, dominant, and epistatic effects of the two major genes. The polygene effect was also stronger, and was affected by the environment. Zhang et al. (2008) hypothesized that the

Table 4. Estimates of genetic parameters for resistance of melon to powdery mildew under the E-1-0 model.

Univalent parameter	Estimate	Bivalent parameter	Estimate		
			B_1	B_2	F_2
d_a	–16.95	σ_p^2	378.68	307.94	519.54
d_b	–16.95	σ_{pg}^2	109.57	96.92	16.71
h_a	20.62	σ_{mg}^2	238.50	180.40	472.21
h_b	24.44	σ_e^2	30.61	30.61	30.61
i	0.22	h_{pg}^2 (%)	28.93	31.47	3.22
j_{ab}	–8.11	h_{mg}^2 (%)	62.98	58.58	90.89
j_{ba}	–0.46				
l	–35.31				
h_a/d_a	–1.22				
h_b/d_b	–1.44				

d_a, d_b, Additive effects of the first and second major genes; h_a, h_b, dominant effects of the first and second major genes, respectively; h_a/d_a, potential ratio of the first major gene; h_b/d_b, potential ratio of the second major gene; i, The epistatic effect of additive×additive between the two major genes; j_{ab}, the epistatic effect of additive×dominant between the two major genes; j_{ba}, the epistatic effect of dominant×additive between the two major genes; l, epistatic effect of dominant×dominant between the two major genes; σ_p^2, phenotypic variance; σ_{pg}^2, polygenic variance; σ_{mg}^2, major gene variance; σ_e^2, environmental variance; h_{mg}^2 (%), major gene heritability; h_{pg}^2 (%), polygenic heritability.

resistance of K7-1 to of *P. xanthii* race 2F. is governed by the single dominant gene, *Pm-2F*. McCreight et al. (1987) proposed that the resistance of PI 414723 to *P. xanthii* race 2F. is controlled by two recessive genes with epistasis to each other. Cheng (2006) also analyzed the genetic mechanism of resistance of the melon to PM, using the mixed major-gene plus polygene inheritance mode; results of this study show that the PM resistance is controlled by two genes with the additive-dominant-epistatic effect. However, the polygene was not detected and the physiological type was not identified. The results of the current study are inconsistent with those of other investigations, probably because of the different melon materials, pathogen, environment, disease assessment system, and genetic analysis system.

In contrast to the classical Mendelian genetic theory, the genetic model of the plant quantitative traits is built on the mixture model statistics theory. Moreover, it unifies biological statistical genetics with Mendelian genetics, explains the intermediate character of each segregation population and provides a deeper and more accurate genetic analysis. Breeding resistant varieties is one of the safest and most effective ways to minimize the problems associated with PM. It is also the key to identification and screening of resistance resources for melons. At present, most melon resistance resources are derived from India, France and Spain, where they are most frequently used; some resources also originate from Japan. Wang et al. (2002) and Ma et al. (2009) also identified several resistance resources, majority of which are thin-skinned melons. However, a report of their use is not available. In the present study, Yuntian-930 was observed to be highly resistant to PM, and its heritability of the major gene plus polygene was found to be very high. In resistance breeding, the major genes may be fully utilized and the accumulation of the polygene may be considered to maintain the durability and stability of resistance. In the future, studies on molecular markers and the localization of Quantitative Trait Locus (QTL), and on molecular marker-assisted selection may be strengthened to promote the breeding of resistant varieties and improve the breeding efficiency.

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REFERENCES

Akaike H (1977). On entropy maximum principles. In: Krishnaiag G (ed.), Applications of Statistics. North-Holland Publishing Company, Amsterdam, Netherlands, pp. 27-41.

Anbessa Y, Warkentin T, Vandenberg A, Ball R (2006). Inheritance of time to flowering in chickpea in a short-season temperate environment. J. Hered., 97: 55-61.

Bohn GW, Whitaker TW (1964). Genetics of resistance to powdery mildew race 2 in muskmelon. Phytopathology, 54: 587-591.

Cohen S, Cohen Y (1986). Genetics and nature of resistance to race 2 of *Sphaerotheca fuliginea* in Cucumis melo PI 124111. Phytopathology, 76(11): 1165-1167.

Chen XJ, Chen JF, Fang R, Cheng ZF, Wang SB (2006). Inheritance of

the node for first flower in pepper (*Capsicum annuum* L.). Acta Hortic. Sin., 33(1): 152-154.
Chen XJ, Chen JF (2006). Genetic analysis of plant height in pepper (*Capsicum annuum* L.). Acta Bot. Boreal.-Occident.Sin., 26(7): 1342-1345.
Cheng ZJ (2006). The genetic mechanisms of resistance to powdery mildews and ALFP marker linked to the disease resistance in melon [M.S.Dissertation]. Nanjing: Nanjing Agricultural University.
Epinat C, Pitrat M, Bertrand F (1993). Genetic analysis of resistance of five melon lines to powdery mildews. Euphytica, 65(2): 135-144.
Gai JY, Wang JK (1998). Identification and estimation of a QTL model and its effects. Theor. Appl. Gen., 97: 1162–1168.
Gai JY, Zhang YM, Wang JK (2003). Genetic System of Quantitative Traits in Plants. Science Press House, Beijing.
Ge XX, Zhang LP, HE ZH, Zhang YM (2004). The mixed inheritance analysis of polyphenol oxidase activities in winter wheat. Acta Agvonornica Sin., 30 (1): 18-20.
Hao JJ, Yu SX, Ma QX, Fan SL, Song MZ (2008). Inheritance of time of flowering in upland cotton under natural conditions. Plant Breed., 127: 383-390.
Harwood RR, Markarian D (1968). The inheritance of resistance to powdery mildew in the cantaloupe variety Seminole. J. Hered., 59(2): 126-130.
Hosoya K, Kuzuya M, Murakami T, Kato K, Narisawa K, Ezura H(2000). Impact of resistant cultivars of melon on *Sphaerotheca fuliginea*. Plant Breed., 119: 286-288.
Hou BW, Dou BD, Zhang YM, Li SHQ, Yang JB, Liu FX, Du JK, Sun QX (2006). The Mixed Major Gene Plus Polygenes Inheritance for Female Fertility in Wheat (*Triticum aestivum* L.). Hereditas, 28(12): 1567-1572.
James DM (1994). Reactions of 20 melon cultigens to powdery mildew race 2U. S. Cucurbitaceae, 43: 813-818.
James DM, Pitrat M, Thomas CE, Kishaba AN, Bohn GW (1987). Powdery mildew resistance genes in muskmelon. J. Am. Soc. Hortic. Sci., 112(1): 63-65.
Kenigsbuch D, Cohen Y (1992). Inheritance and allelism of genes for resistance to races 1 and 2 of Sphaerotheca fuliginea in muskmelon. Plant Dis., 76(6): 626-629.
Křístková E, Lebeda A, Sedláková B (2004). Virulence of Czech cucurbit powdery mildew isolates on *Cucumis melon* genotypes MR-l and PI 1241l2. Sci. Hortic., 99(3-4): 257-265.
Knott SA, Haley CS, Thompson R (1991). Methods of segregation analysis for animal breeding data: a comparison of power. Heredity, 68: 299-311.
Li JS, Shen HL, Shi ZQ (2006). Analysis on the major gene and poly gene mixed in heritance of lycopene content in fresh consumptive tomato fruit. Hereditas (Beijing), 28(4): 458-462.
Liu EY, Liu YM, Fang ZY, Yang LM, Zhuang M, Zhang YY, Yuan SX, Sun JF, Li ZS, Sun PT (2009). Genetic analysis of head-leaf traits using mixed major gene plus polygene inheritance model in *Brassica oleracea* L. var.*italica* Planch. Acta Hortic. Sin., 36(11): 1611-1618.
Ma HY, Zu YG, Luan FS (2009). Identification of melon germplasm resistance to powdery mildew in seedling stage. J. Northeast Agric. Univ., 40(12): 18-23.
Pang WL, Liu FZ, Chen YH, Lian Y (2008). Genetic study on fruit color traits of eggplant. Acta Hortic. Sin., 35(7): 979-986.
Pryor DE (1942). The influence of vitamin B1 on development of cantaloupe powdery mildew. Phytopathology, 32: 885-895.
Qi CK. Gai JY, Zhang YM (2001). Major gene plus polygene inheritance of erucic acid content in *Brassicu napus* L.. Acta Genetica Sin., 28(2): 182-187.
Tian LS, Niu YZ, Yu QQ, Guo SX, Liu L (2009). Genetic Analysis of White Flower Color with Mixed Model of Major Gene Plus Polygene in *Brassica napus* L.. Sci. Agric. Sin., 42(11): 3987-3995.
Teixeira APM, Barreto FA da S, Camargo LEA (2008). An AFLP marker linked to the Pm-1 gene that confers resistance to Podosphaera xanthii race 1 in *Cucumis melo* L. Genet. Mol. Biol., 31(2): 547-550.
Wang JK, Gai JY (1997). Identification of major gene and polygene mixed inheritance model and estimation of genetic parameters of a quantitative trait from F2 progeny. Acta Genet. Sin., 24(5): 398-404.
Wang JK, Gai JY (2001). Mixed inheritance model for resistance to agromyzid beanfly (*Melanagromyza sojae* Zehntner) in soybean. Euphytica, 122: 9-18.
Wang JS, Song SH, Tang XW, Chen GL (2005). Genetics and Molecular Marker of the Gene for Disease to Powdery Mildew on *Cucumis melo* L.. Acta Agric. Boreali Sin., 20(1): 89-92.
Wang JS, Tang XW, Meng SC, Song SH, Chen GL, Sun ZY, Li M (2002). Identification and preliminary genetic analysis of resistance to powdery mildew on Melon. Acta Agric. Boreali Sin., 17(3): 124-128.
Wang JS, Wang JK, Zhu LH, Gai JY (2000). Major-polygene effect analysis of resistance to Bacterial Blight (*Xanthomonas campestris* pv. *oryzae*) in rice. Acta Genet. Sin., 27(1): 34-38.
Wei X, Li LH, Wang J, Fan QQ, Lan H, Wu YQ, Rong TZ, Pan GT (2008). Quantitative genetic analysis of silk cut in maize (*Zea mays* L.). Sci. Agric. Sin., 41(8): 2235-2240.
Xu AX, Huang Z, Li JH, Lu RW, Xiao ES, Zhang XS (2010). Inheritance of erucic acid content of brassica juncea in Northern Shaanxi with mixed model of major gene plus polygene. Acta Bot. Boreal.-Occident.Sin., 30(5): 0933-0938.
Xu ZH, Shou WL, Huang KM, Zhou SJ (1999). Determination of physiological race of powdery mildew and its virulence to different melon genotypes. Acta Agric. Zhejiangensis, 11(5): 245-248.
Xue Y, Jiang L, Su N, Wang JK, Deng P, Ma JF, Zhai HQ, Wan JM (2007). The genetic basic and fine-mapping of a stable quantitative-trait loci for aluminium tolerance in rice. Planta, 227: 255-262.
Yuan YL, Zhang TZ, Guo WB, Yu J, Kohel RJ (2002). Major polygene effect analysis of super quality fiber properties in upland cotton. Acta Genet. Sin., 29(9): 827-834.
Yuste-Lisbona FJ, López-Sesé AI, Gómez-Guillamón ML (2010). Inheritance of resistance to races 1,2 and 5 of powdery mildew in the melon TGR-1551. Plant Breed., 129: 72-75.
Zhan QW, Gai JY, Zhang YM, Sun ZD (2001). Development and Expression Process of Inheritance of Resistance to Cottom Worm (*Prodenia litura*) in Soybeans. Acta Genet. Sin., 28(10): 956-963.
Zhang H, Wang HS, He CX, Zhang ZB, Zhang X, Yi HP, Wu MZ (2009). Genetic Study on Sugar and Sour Traits of Melon (*Cucumis melo* L.). Acta Hortic. Sin., 36(7): 989-996.
Zhang HY, Su F, Guo SG, Yi HP, Wu MZ, Xu Y (2008). Genetic analysis and specific fragments linked to powdery mildew resistant gene Pm-2F in melon. Acta Hortic. Sin., 35(12): 1773-1780.
Zhang LW, Liu PW, Hong DF, Huang AQ, Li SP, He QB, Yang GS (2010). Inheritance of seeds per silique in *Brassica napus* L. using joint segregation analysis. Field Crops Res., 116: 58-67.
Zhang SF, Fu TD, Zhu JC, Wang JP, Wen YC, Ma CZ (2008). Genetic analysis of erucic acid in *Brassica napus L.* using mixed major gene and polygene inheritance model. Sci. Agric. Sin., 41(10): 3343-3349.
Zhang SQ, GU XF, Zhang SP, Zou ZR (2007). Inheritance of downy mildew resistance in cucumber (*Cucumis sativus* L.). Acta Bot.Boreal.-Occident.Sin., 27(12): 1342-1345.
Zhang YM, Gai JY, Yang YH (2003). The EIM algorithm in the joint segregation analysis of quantitative traits. Gene Res., 81: 157-163.
Zhao GW (2009). Researches on SRAP molecular markers linked with resistant gene to powdery mildew in melon and changes of defense enzyme activity in resistance response [M.S.Dissertation]. Beijin: Chinese Academy of Agricultural Sciences.
Zhu SY, Guo Y, Hong DL (2008). Genetic analysis on aging-resistant in rice seed. Hereditas, 30(2): 217-224.
Zhuo ZC, Wan EM, Zhang LG, Zhang MK, Hui MX (2009). Major gene plus polygene inheritance analysis of bolting trait in heading Chinese cabbage. Acta Bot. Boreal.-Occident. Sin., 29(5): 0867-0873.