RESEARCH ARTICLE

SABRAO Journal
of Breeding and Genetics
53 (1) 44-56, 2021

HERITABILITY OF KENAF (*Hibiscus cannabinus* L.) RESISTANCE TO ROOT- KNOT NEMATODES (*Meliodogyne incognita*)

PARNIDI[1,2*], L. SOETOPO[1], DAMANHURI[1] and MARJANI[2]

[1]Faculty of Agriculture, Brawijaya University of Malang, Indonesia
[2]Indonesian Sweetener and Fiber Crops Research Institute (Balittas), Malang, Indonesia
*Corresponding author email: nikicro@yahoo.co.id
Email addresses of coauthors: lita.fp@ub.ac.id, damanhuri385@yahoo.com, marjani.balittas@gmail.com

SUMMARY

Root knot nematode (*Meloidogyne* spp.) (RKN) is a plant-parasitic nematode of kenaf. RKN infestation on roots may lead to stunted plant growth in plants and a reduction in yield potential. The inheritance of resistance in kenaf to RKN *M. incognita* was investigated by crossing two different genotypes of kenaf: Karangpolos 1 (KR1), which is very susceptible to root-knot nematode with Karangploso 15 (KR15), which is moderately resistant to root-knot nematode. Resistance was evaluated using several variables, including the number of root-knot nematodes, reproductive factors, the number of 2nd stage juveniles, the number of egg mass, and the average number of eggs per egg mass. This study found that female parental genotypes did not significantly contribute to kenaf resistance to root-knot nematode. Partial dominance of a single gene was predicted to be responsible for the resistance to RKN *M. incognita*. The gene effects to all of resistance variables tested were mainly additive and dominant; additive variance was greater compared to dominant variance. The broad-sense heritability value and the narrow-sense heritability value kenaf resistance to RKN *M. incognita* for all variables were high. The results of this study are useful for determining the appropriate selection model for kenaf plant resistance to RKN *M. incognita*.

Keywords: Heritability, resistance, root knot nematode, kenaf

Key findings: Kenaf resistance against RKN *M. incognita* can be inherited to the offspring which is controlled by nucleus genes.

Manuscript received: November 19, 2020; Decision on manuscript: December 25, 2020;
Accepted: January 21, 2021.

Communicating Editor: Dr. SoonWook Kwon

INTRODUCTION

Kenaf (*Hibiscus cannabinus* L.) is a short day annual herbaceous plant belonging to the Malvaceae, a family notable for both its economic and horticulture importance. The existence of semi-wild kenaf in Africa (Kenya and Tanzania) might be an indication of its origin from this continent. Kenaf is one of important fiber sources that has potential to become an industrial crop. Kenaf fiber is used as raw material for making fiber board (door-trim, car interiors), particle board, fiber drain, geo-textile, high-quality paper (Purwati, 2009). In Africa, the leaves and the scions were used as raw or cooked food. Seeds were used for oil and various plant parts were used in medicines and in certain superstitious rites (Alexopoulou, 2013).

Root knot nematode (*Meloidogyne* spp.) (RKN *Meloidogyne* spp) is one of important plant-parasitic organisms in kenaf (Tahery *et al.*, 2011; Wokoma *et al*., 2015; Adegbite, 2018). However, RKN infestation on roots may cause stunted growth in plants, which then reduce plant production potential. Loss of kenaf yields due to root knot nematode infection might reach up to 19% (Setyo-budi *et al*., 2009), although higher losses of 32% – 67% have also been reported (Lawrence and McLean, 1992). Tahery *et al.* (2011) added that population density of 5000 root knot nematodes per 500 cm^3 of soil (5000/500 cm^3) may influence and reduce kenaf stem height, stem diameter, stem weight, and roots. Moreover, root knot nematode infection may reduce the wet weight of kenaf stems by up to 3 tons per hectare. Supriyono and Suhara (2007) reported that initial population consisted of 40 juveniles per 100 ml of soil was capable to reduce kenaf production. In short, plant damage caused by root knot nematode will also increase along with the increasing number of root knot nematode infecting root tissues.

Various ways have been developed in order to control root knot nematode population, such as utilizing chemical nematicides and/or resistant cultivars, as well as plant rotation. Unfortunately, the use of chemical nematicides is restricted as it may cause damage on both environment and human health due to its high toxicity properties. Accordingly, the use of resistant cultivars is the best alternative to suppress the nematode population and prevent loss of plant yields (De Deus Barbosa *et al.*, 2009; Davis and Stetina, 2016; Costa *et al.*, 2017). Therefore, a preliminary study regarding the genes underlying kenaf resistance to root knot nematode is required to select the best resistant cultivars for further use.

Genetic parameters for determining the resistance of kenaf to root knot nematodes have not been well studied. Several studies have been carried out in various plants in order to investigate the genes underlying plant resistance to root knot nematode. Falusi (2008) found that the resistance of roselle to root knot nematode is mainly controlled by single genes with dominant gene action. However, McPherson *et al.* (2004) suggested that two dominant genes are significantly associated with the cotton plant resistance to root knot nematode. Shahadati-moghadam *et al*. (2017) stated that genes with additive genetic effects are responsible for tobacco resistance to root knot nematode (*M. incognita* race 2). The broad-sense and the narrow-

sense heritability values were 0.93 and 0.75, respectively. The number of egg masses was 0.94 and 0.56, while the number of eggs per egg mass was 0.61 and 0.28. Wang *et al*. (2017) has identified quantitative trait loci (QTL) on chromosome 11 to be significantly associated with cotton plant resistance to root knot nematode. QTL 11 was negatively associated with the number of root knot and its egg production.

Accordingly, a study on the heritability of kenaf resistance to root knot nematode is required to plan and develop an effective and efficient breeding program strategy to obtain hybrids with high yielding genotypes with resistance to root knot nematode. The aim of this research to study the inheritance of kenaf (*H. cannabinus* L) resistance to root-knot nematodes (*M. incognita*).

MATERIALS AND METHODS

Research was carried out from November 2018 to February 2019. The field experiment was conducted at the Karangploso Experimental Garden and nematode counting were carried out at the Laboratory of Plant Pathology at Indonesian Sweetener and Fiber Crops Research Institute (ISFRI) Malang, Indonesia. Kenaf was put into 30 x 30 cm polybags with a spacing of 30 x 50 cm, and then planted with distance between replications around 100 cm. The growing media was sandy soil consisted of 55% sand, 36% silt, and 17% sterile clay, which then sterilized using 4% formalin solution.

Hybrids generated in this study were produced through crossing between moderately resistant genotype (Karangploso 15 = KR15)(P_1) and very susceptible genotype (Karangploso 1= KR1) (P_2) which then collected for further investigation, including filial 1 hybrids (F_1), reciprocal F_1 hybrids (F_{1R}), filial 2 hybrids (F_2), backcross hybrids derived from the cross between F_1 hybrids and very susceptible genotype (BC_1P_1), backcross hybrids derived from the cross between F_1 hybrids and moderately resistance genotype (BC_1P_2). Total of 30 plants were planted for each of P_1, P_2, and F_1 populations, 50 plants for each of BC_1P_1 and BC_1P_2 populations, and 200 plants for F_2 population.

The number of root knot was calculated to determine the total of root knot formed on kenaf roots. The number of 2nd stage juveniles per 100 g soil was calculated based on Taylor and Sasser (1978) and Dalmadiyo *et al*. (1989) with a purpose to determine nematode reproduction factors. The number of 2nd stage juveniles per 10 g roots was calculated based on Dalmadiyo *et al*. (1989). Egg mass was measured from 10 g of root samples which was previously immersed in a solution of floxin B (Sigma-Aldrich) at a concentration of 0.15 g/L for 15 mins (Tahery *et al*., 2011). Ten egg masses in each genotype were immersed in 0.5% sodium hypochloride (1 mL) for 1 min and homogenized thoroughly based on a method proposed by Shahadati-moghaddam *et al*. (2017). The 50 µL solutions from 3 samples were observed under light microscope, which then continued by counting the number of eggs and the number of eggs per egg mass. Resistance characters, including the number of root knot, reproduction factors, the number of 2nd stage juveniles, egg mass, and the average number of eggs per egg mass, were analyzed to predict gene actions, the genes

controlling resistance, as well as the values of both broad-sense, narrow-sense heritabilities and to predict the presence of maternal effects. The degree of dominance was calculated based on the formula postulated by Petr dan Frey (1966):

$$hp = \frac{(F_1 - MP)}{(HP - MP)}$$

hp = potential ratio
F_1 = average value of F_1,
HP = the highest average value of parental cultivars,
MP = median values of both parental cultivars.

The degree of dominance is classified based on the potential ratio as follows: HP = 0 (no dominance); HP = 1 or HP = -1 (dominant or fully recessive); 0 < HP < 1 (partially dominant); -1 < HP < 0 (partially recessive); and HP > 1 or HP < -1 (over-dominant). Estimation of the genes associated with each resistance character to nematodes was calculated using an equation formulated by Lande (1981):

$$\sigma^2_{F_2} - \frac{2\left(\sigma^2_{F_1} + \sigma^2_{P_1} + \sigma^2_{P_2}\right)}{4}$$

μ_{P1} = means of KR1 parental population
μ_{P2} = means of KR15 parental population
σ^2_{F2} = variance of F_2; σ^2_{P1} = variance of KR1
σ^2_{F2} = variance of KR15

The estimate of broad-sense heritability was given by an equation formulated by Allard (1960):

$$h^2_{bs} = \left(2\sigma^2_{F_2} - \frac{\frac{1}{3\left(\sigma^2_{P_1} + \sigma^2_{P_2} + \sigma^2_{F_1}\right)}}{\sigma^2_{F_2}}\right)$$

h^2_{bs} = broad-sense heritability
σ^2_{F2} = variance of F_2
σ^2_{P1} = variance of KR1
σ^2_{P2} = variance of KR15
σ^2_{F1} = variance of F_1.

Narrow-sense heritability was calculated based on an equation by Warner (1952):

$$h^2_{ns} = 2\sigma^2_{F_2} - \frac{\sigma^2_{BC_1P_1} + \sigma^2_{BC_1P_2}}{\sigma^2_{F_2}}$$

h^2_{ns} = narrow-sense heritability
σ^2_{BC1P1} = variance of BC_1P_1 population
σ^2_{BC1P1} = variance of BC_1P_2 population
σ^2_{F2} = variance of F_2.

The estimate heritability values were considered to be low if h^2 < 20%, moderate if 20% ≤ h^2 ≤ 50%, and high if h^2 > 50%.

The likelihood of additive and dominant models was estimated by three genetic parameters of Joint Scaling Test (Mather and Jink, 1982): (m) median value; (d) the number of additive genetic effect; (h) the number of dominant genetic effect. If the gene action does not meet the additive-dominant models, following test is carried out to check the possibility of non-allelic gene interaction using epistatic model with 6 parameters (Mather and Jink, 1982). Maternal effect was measured by comparing the median values of F_1 and F_{1R} using t-test at a significance level of 5% based on Syukur (2007). The frequency distribution of the F_2

population was tested for normality using the Kolmogorov-Smirnov method with SPSS 16 software following Syukur (2007).

RESULTS

Analysis on heritability of resistance to nematode

Analysis on heritability of resistance against RKN *M. incognita* infection including: normality test, degree of dominance and number of genes controlling the phenotype of interests, variance components, estimation of genetic models, and heritability values.

Normality test in F_2 population frequency

Normality test in F_2 population was carried out in order to investigate the number of genes controlling the resistance to root-knot nematode. The results indicated that F_2 population was non-normally distributed (P < 0.100) on all tested resistance variables to RKN *M. incognita* (Figure 1).

Degree of dominance and number of genes controlling phenotype of interest

The degree of dominance was calculated by estimating the potential ratio (*hp*) derived from median values of parental strains and F_1 hybrids using an equation formulated by Petr and Frey (1966). The potential ratio of hybrids produced from KR1 x KR15 crosses for the number of root knot nematode, reproduction factors, number of 2^{nd} stage juveniles, number of egg mass, and number of eggs per egg mass were 0.22, 0.10, 0.02, 0.55, and 0.03, respectively (Table 1). The relative position of F_1 was also supported by potential ratio (Figure 2). The potential ratio of resistance against RKN *M. incognita* infection was classified into *hp* group which is in the range of 0 to 1.

Based on the analysis of number of genes controlling kenaf resistance to root-knot nematode, there was at least one group of genes controlling this phenotype (number of root knot 0.003 = 1; reproduction factor 0.06 = 1; number of 2^{nd} stage juveniles 0.034 = 1; number of egg mass 0.015 = 1; and number of eggs per egg mass 0.003 = 1).

Estimation of genetic component

Joint scaling test was applied to determine gene actions controlling the resistance to root knot nematode (Mather and Jinks, 1982). Scaling test of each individual is carried out by comparing t-value and t-statistic to determine the significance of genetic components (Singh and Chaudhary, 1979). The estimated value obtained from individual scaling test for resistance to nematode is presented in Table 2. The number of root knot nematode (0.85), reproduction factor (0.87), the number of 2^{nd} stage juveniles (1.92), the number of egg mass (0.73) and the average number of eggs per egg mass (1.94) were smaller than t-statistic (1.96).

Heritability

Heritability value reflects the effects of genotype towards phenotype. Broad-sense heritability (h^2_{bs}) and narrow-sense heritability (h^2_{ns}) of kenaf resistance for all variables tested were high (Table 3).

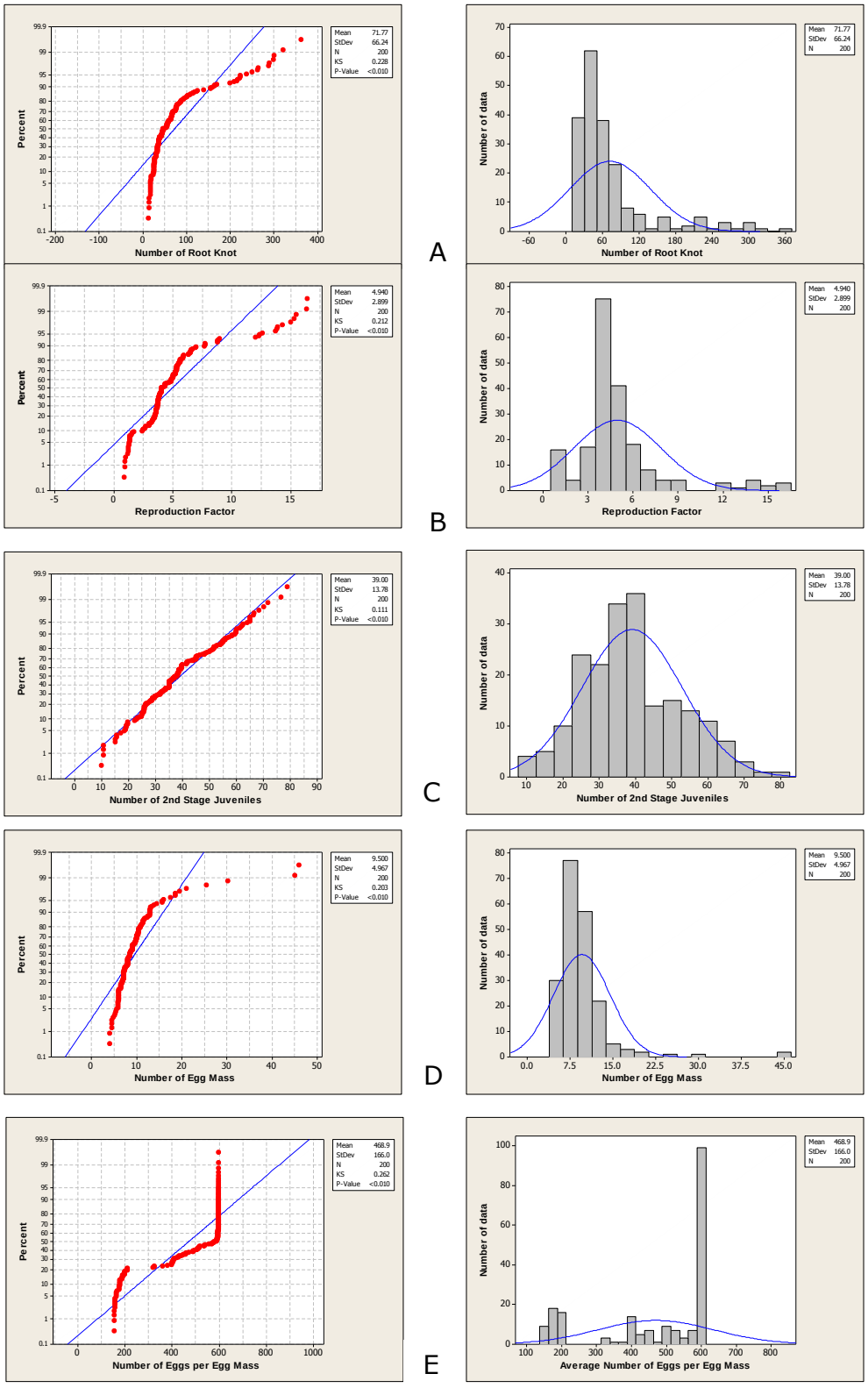

Figure 1. F_2 population distribution; (A) The number of root-knot nematode; (B) reproduction factor; (C) the number of 2nd stage juveniles; (D) the number of egg mass; and (E) the average number of eggs per egg mass.

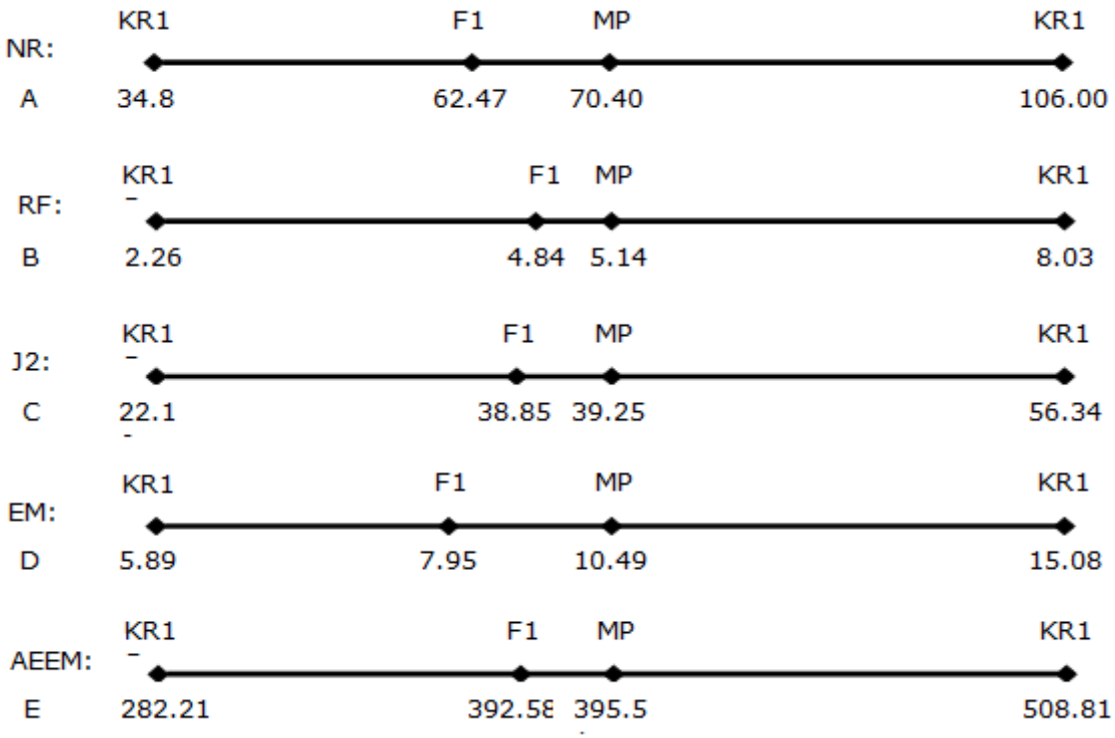

Figure 2. Schematic of the relative position of F_1 mean values to both parental genotypes for the variables of: (A) the number of root knot nematode; (B) reproduction factor; (C) the number 2nd stage juveniles; (D) the number of egg mass; (E) the average number of eggs per egg mass; MP = mid parent (median values).

Table 1. The potential ratio and number of genes controlling kenaf resistance against RKN *M. incognita* infections.

Population	Median values of resistance variables				
	NR	RF	J2	EM	AEEM
P_1 (KR1)	106.00±4.96	8.03±0.55	56.34±1.95	15.08±0.95	508.81±9.95
P_2 (KR15)	34.80±3.32	2.26±0.01	22.16±1.71	5.89±0.71	282.21±8.71
F_1	62.47±3.48	4.84±0.04	38.85±1.84	7.95±0.84	392.58±8.84
Mid Parent (MP)	70.40	5.14	39.25	10.49	395.51
Potential ratio	0.22	0.10	0.02	0.55	0.03
Gene action	Partially dominant-positive				
The number of genes controlling the phenotypes	0.003	0.06	0.034	0.015	0.003

Note: NR = the number of root knot nematode; RF = reproduction factor; J2 = the number of 2nd stage juveniles; EM = the number of egg mass; AEEM = the average number of eggs per egg mass.

Table 2. The estimated values of genetic parameters used for scaling test of kenaf resistance to RKN *M. incognita*.

Parameters	NR	RF	J2	EM	AEEM
M	348.76	22.62	193.25	47.41	2245.11
[d]	35.60	2.89	17.09	4.60	113.30
[h]	8.39	0.22	5.58	1.33	99.48
C	233.34	15.85	112.48	31.29	1317.03
SE. C	274.85	18.14	58.62	42.75	679.47
t-value	0.85ns	0.87ns	1.92ns	0.73ns	1.94ns
t-statistic	1.96	1.96	1.96	1.96	1.96

ns = not significantly different
C = 4 (F_2 mean values) – (P_1 mean values) – 2 (F_1 mean values) – (P_2 mean values) (Singh and Chaudary, 1979); m = median values; d = additive values; h = dominance values; C = gene interaction values; SE. C = standard error C; NR = the number of root knot nematode; RF = reproduction factor; J2 = the number of 2nd stage juveniles; EM = the number of egg mass; and AEEM = the average number of eggs per egg mass.

Table 3. Variety components and heritability values of kenaf resistance against RKN *M. incognita* infection.

No.	Genetic Parameters	NR	RF	J2	EM	AEEM
1	Environmental Variance (σ^2_E)	903.04	3.05	64.99	25.49	6859.23
2	Additive Variance (σ^2_D)	17322.90	57.08	739.81	411.38	109466.11
3	Dominance Variance (σ^2_H)	7463.84	18.18	389.40	186.41	39256.79
4	Phenotypic Variance (σ^2_{F2})	9717.30	15.15	532.24	114.52	71406.49
5	BC_1P_1 and BC_1P_2 Variance	12486.30	28.04	694.58	189.58	88079.92
6	Broad-sense Heritability (h^2_{bs})	79.42	80.17	65.80	75.65	86.91
7	Narrow-sense Heritability (h^2_{ns})	62.62	75.45	57.31	61.57	70.55
8	H^2_{ns}/h^2_{bs} Proportion (%)	78.85	94.11	87.11	81.39	81.17

Note: NR = the number of root knot nematode; RF = reproduction factor; J2 = the number of 2nd stage juveniles; EM= the number of egg mass; and AEEM= the average number of eggs per egg mass.

The influence of female parents was examined using t-test on all F_1 hybrids, including the reciprocal hybrids, in order to determine whether the resistance characters in kenaf to root knot nematode are maternal or not. The results indicated that there was no maternal effect as shown by *P* value higher than 0.05.

The homogeneity test of resistance variance analyzed using F-test on both F_1 and F_{1R} populations which were obtained from KR1 x KR15 crosses indicated homogeneous distribution. This result was supported by *F* value that is higher than *F*statistic at significance level of 5% (Table 4). According to this result, F_1 and F_{1R} data were then merged into F_1 population.

DISCUSSION

The resistance characters against diseases are qualitative characters characterized by an abnormal distribution of F_2 population (Figure 1). The segregation pattern of qualitative characters follows Mendel's

Table 4. The mean value and standard error of resistance to *M. incognita* infection on F_1 and F_{1R} hybrids, as well as the median and homogeneity tests on the hybrids of KR1 x KR15 crosses.

Population	NR	RF	J2	EM	AEEM
F1	62.5±7.5	4.8±0.13	38.85±1.40	7.95±0.5	392.9±15.0
F1R	60.7±7.2	4.5±0.13	37.82±1.40	7.01±0.7	387.8±15.0
Prob > (t)	0.09ns	0.11ns	0.59ns	0.27ns	0.48ns
Prob > (F)	0.85ns	0.77ns	0.94ns	0.10ns	0.87ns

ns = not significantly different, Note: NR = the number of root knot nematode; RF = reproduction factor; J2 = the number of 2nd stage juveniles; EM = the number of egg mass; and AEEM = the average number of eggs per egg mass; ns = not significantly different.

ratio and its modification. Baihaki (2000) and Wanda *et al*. (2014) stated that abnormal distribution of F_2 population indicates that the resistance characters are controlled by simple or major genes. Baihaki (2000) explained that the qualitative characters of a plant are usually controlled by one or two genes, less influenced by the environment, and relatively easy to handle for further breeding project.

The degree of dominance (*hp*) on all evaluated resistance was ranged from 0.03 to 0.55 (Table 1). If the *hp* value of a character is in the range of 0 and 1, then it indicates that the character is controlled by the action of a positive imperfect dominant gene (partial positive dominance effect). The result of this study is in line with Setyo-budi *et al*. (2009) who described that the inheritance of kenaf genotype resistance against root-knot nematode is associated with a partial positive dominant gene. Bertrand *et al*. (1995) explained that partial positive dominance effects control the plant's resistance against *Meloidogyne* spp. nematode which infects Catimor Arabica coffee. According to Roberts *et al*. (1998) the resistance of tomato varieties against *Meloidogyne* spp. based on the number of egg mass and the rate of root-knot nematode formation is controlled by partial dominant gene action. The degree of partial positive dominance effect on F_1 genotype obtained from the KR1 X KR15 cross indicated the presence of strong dominant genes which the trait inheritance is controlled by partial dominance.

The resistance characters of kenaf plants against NPA *M. incognita* were controlled by one gene. The same result was reported by Falusi (2008) who found that the rosella resistance against nematode is controlled by single gene. Shahadati-Moghaddam *et al*. (2017) also reported that the tobacco resistance against NPA *M. incognita* is associated with single gene with partial positive dominance effect. The number of control genes indicating the number of genes that are effective in controlling the expression of certain phenotypes. The most suitable model of gene action for character of kenaf resistance against NPA *M. incognita* is the dominant additive (m[d][h]) (Table 2). The value of additive variance is greater than the dominant variant indicating that genetic variance is more determined by with the action of additive genes.

The variation in plant's phenotypes is the result of diversity influenced by genetic and environmental factors. The genetic diversity, which is the focus of plant

breeding, is generally resulted from the effect of additive gene action, dominance gene action, as well as epistasis interaction (Falconer, 1960).

The phenotype of interests as observed from the broad-sense heritability value, including the number of root-knot nematode (79.42%), reproductive factors (80.17%), the number of 2nd stage juveniles (65.80%), the number of egg mass (75.65%), and the average number of eggs per egg mass (86.91%), were influenced by genetic variability. Interestingly, all the evaluated variables were influenced by additive genes such as the number of root-knot nematode (62.62%), reproductive factors (75.45%), the number of 2nd stage juveniles (57.31%), the number of egg mass (61.57%), and the average number of eggs per egg mass (70.55%), with a difference ranged from 4.82% to 16.80%. This results indicated the small role of non-additive gene action (both dominance and epistasis) on the kenaf resistance against NPA *M. incognita*.

The magnitude effect of genetic variance towards resistance phenotypes indicated the small role of environmental factor (E = the number of root-knot nematode (20.58%; reproductive factors (19.83%); the number of 2nd stage juveniles (34.20%); the number of egg mass (24.35%) and the average number of eggs per egg mass (13.09%). This result is consistent with the estimation of genetic variance component in which the environmental variance component has no significant effect on all resistance variables (Table 3).

The basis for the success of plant breeding is largely determined by the richness of genetic diversity that can be passed on from parents to offspring. Additive component is the only component of genetic variance that is inherited by the offspring, where the narrow-sense heritability value supports the idea through the genetic proportion of total phenotypes that is completely inherited to the offspring (Falconer, 1960; Poehlman and Sleper, 1995). In addition, kenaf is a self-pollinating plant, so selection should be applied on its additive effect to generate and pool superior genotypes; selection is considered to be ineffective if the superior genotype is determined by the dominance effects and epistasis interactions (interaction between genes) (Poehlman and Sleeper, 1995).

A high additive effect implies the effectiveness of selection. Basically, selection on the phenotypes with high heritability can be performed in the early generations. Barmawi *et al*. (2013) described that a phenotype/character with a high heritability value can be selected in the early generations (F_2 and F_3). In this case, the most suitable selection method is pedigree method. Pedigree selection can be applied on the early generations through recording, so the pedigree of produced strains is clear and known. Pedigree selection is based on the best individual performances originated from the best families.

This study recorded that the resistance characters of kenaf plant against NPA *M. incognita* was not influenced by the female parents (Table 4). This indicates that the nature of resistance phenotype is controlled by genes encoded by nuclear genes. The resistance properties of kenaf against NPA *M. incognita* were similar to those of arabica coffee resistance against *Radopholus similis* Cobb (Hulupi *et al*.,

2007) chili plant resistance to anthracnose (Syukur *et al*., 2007; Hapshoh *et al*., 2016) and chili plant resistance to aphids (Daryanto *et al*., 2017) which were not influenced by female parents.

CONCLUSION

The estimation of genetic parameters through the analysis of six populations indicated that the additive effects with partial dominance gene action had a large contribution on the inheritance of kenaf resistance against NPA *M. incognita*. A single gene group is assumed to be associated with the resistance characters. Moreover, the broad-sense and narrow-sense heritability values are high due to the large effect of genetic influence (both additive and dominant gene actions) towards the tested resistance variables. In addition, kenaf resistance against *M. incognita* was shown to be controlled by genes present in the cell nucleus.

ACKNOWLEDGEMENTS

We are thankful to the Agency for Agricultural Research and Development, Ministry of Agriculture, Republic of Indonesia, for providing doctoral scholarship for the researchers at the Faculty of Agriculture, Brawijaya University, Malang, Indonesia. We are also grateful towards Indonesian Sweetener and Fiber Crops Research Institute (ISFRI) and the Head of ISFRI Experimental Garden, Karangploso, for assisting this study. We thank Ir. Titiek Yulianti, M.Sc. PhD from the Indonesian Sweetener and Fiber Crops Research Institute (ISFRI) for providing laboratory facilities for supporting this study. In addition, we also thank our colleagues at Faculty of Agriculture, Brawijaya University, Malang (Farhan Amin Gemayel, Naufal Zaki dan Aditya Aji Noftara) and University of Raden Rachmad, Malang (Evi Dwi Safitri dan Rina Zazilatul Khoiroh), Indonesia for giving their suggestions during the research.

REFERENCES

Adegbite AA (2018). Response of selected kenaf cultivars to *Meloidogyne incognita* under greenhouse conditions. *Int. J. Adv. Agric. Res.* 6:55-58.

Allard RW (1960). Principles of plant breeding. John Willey and Sons Inc. New York.

Baihaki A (2000). Teknik rancang dan analisis penelitian pemuliaan (Diktat Kuliah). Fakultas Pertanian. Universitas Padjadjaran, Bandung.

Barmawi M, Yushardi A, Sa'diyah N (2013). Daya waris dan harapan kemajuan seleksi karakter agronomi kedelai generasi F_2 hasil persilangan antara yellow bean dan Taichung. *J. Agrotek Tropika*. 1(1): 20–24.

Bertrand B, Anzueto F, Pena MX, An-thony F, Eskes AB (1995). Genetic improvement of coffee for resistance to root-knot nematodes (*Meloydogyne* spp.) in Central America. Proceedings X Int. Sci. Colloq. Coffee: 630–636.

Costa GAS, Hugo VSB, Giband M, Augusto PVB, Rodrigues F, Rúbia da Rocha M (2017). Inheritance of resistance to *Meloidogyne incognita* race 3 in cotton accession TX 25. *Acta Scient*. 39(3): 331–337

Dalmadiyo G, Pusposendjojo N, Rahayu BT (1989). Effect of initial population densities of root knot nematode (Meloidogyne spp) on growth and yield of kenaf (*H. cannabinus* L.). *BPPS UGM*, *2*(1B): 165–176.

Daryanto A, Syukur M, Maharijaya A, Hidayat P (2017). Inheritance of Chili Pepper Resistance Against

Infestation of Aphis gossypii Glover. *J. Hort. Indonesia*, *8*(1), 39–47. https://doi.org/10.29244/jhi.8.1.39-47

Davis RF, Stetina SR (2016). Resistance and tolerance to nematodes in cotton. In RLB Galbieri e Jean. Nematoides fitoparasitas do algodoeiro nos cerrados brasileiros : Biologia e medidas de controle. Dados Internacionais de Catalogação na Publicação (CIP) Instituto. Cuiaba.

De Deus Barbosa AEA, da Rocha Fragoso R, de Lima e Souza DDS, Freire É, de Oliveira Neto OB, Viana AAB, Togawa RC, Guimarães LM, Martins NF, Cia E, Fernandez D, de Lima LM, Silva MCM, Rocha TL (2009). Differentially expressed genes in cotton plant genotypes infected with *Meloidogyne incognita*. *Plant Sci*. 177: 492-497.

Falconer DS (1960). Introduction to quantitative genetics. 1st ed., London.

Falusi OA (2008). Inheritance of characters in kenaf (*Hibiscus cannabinus*). *Afr. J. Biotechnol*. 7(7): 904–906.

Hapshoh S, Syukur M, Wahyu Y, Widodo (2016). Inheritance of qualitative characters of ornamental chili pepper from hybridization of chili pepper and bird pepper. *J. Agron. Indonesia* 44(3): 286–291.

Hulupi R, Nasrullah, Soemartono (2007). Inheritance of arabica coffee resistance to radopholus similis cobb. *Pelita Perkebunan 23*(1): 1–16.

Lande R (1981). The minimum number of gene contributing to quantitative variation between and within populations. *Genet*. 99: 541-553.

Lawrence G, McLean K (1992). Host status response of kenaf (*H.cannabinus*) to *Meloidogyne incognita* race 4. *M. javanica*. *Hoplolaimus magnitylus* and *Rotylenchulus reniformis. Nematropica*. 22(2): 247–250.

Mather SK and Jink JL (1982). Biometrical genetic, 3rd ed., SK Mather and JL Jink. Springer Science Business Media. Birmingham, UK.

McPherson MG, Jenkins JN, Watson CE, McCarty JC (2004). Inheritance of root-knot nematode resistance in M-315 RNR and M78-RNR cotton. *J. Cotton Sci*. 8(3): 154–161.

Petr FC, Frey KJ (1966). Genotypic correlation, dominance and heritability of quantitative characters in oat. *Crop Sci*. 6: 259-262.

Poehlman JM, Sleper DA (1995). Breeding fields crops. 4th ed., JM Poehlman and AS David.Oxford and IBH Publishing. Iowa.

Roberts PA, Matthews WC, Veremis JC (1998). Genetic mechanisms of host plant resistance to nematodes. In: Barker KR, Pederson GA and Windham GL (eds.) Plant and Nematode Interactions. Agronomy Monograph no. 36. Madison. Wisconsin, USA.

Setyo-Budi U, Sudjindro, Purwati RD (2009). Resistance variability of kenaf (*Hibiscus cannabinus* L.) genotypes to root knot nematode (*Meloidogyne incognita*). *J. Litri* 15(2): 60–65.

Shahadati-moghaddam Z, Bagheri N, Jelodar NB (2017). Inheritance of southern root-knot nematode resistance in air-cured tobacco. *Trop. Plant Pathol*. 42: 32–38.

Singh RK, Chaudhary BD (1979). Biometrical methods in quantitative genetic analysis. Edisi Revisi. Kalyani Publishers. New Delhi.

Supriyono, Suhara C (2007). Evaluasi ketahanan plasma nutfah kenaf dan kerabatnya terhadap *Fusarium* sp. dan nematoda puru akar *Meloidogyne spp.* Malang, Indinesia.

Syukur M (2007). *Genetics analysis and inheritance studies of resistance to anthracnose caused by Colletotrichum acutatum in pepper (Capsicum annuum L.)*. Institut Pertanian Bogor, Indonesia.

Syukur M, Sujiprihati S, Koswara J (2007). Inheritance of resistance to

anthracnose caused by colletotrichum acutatum in pepper (*Capsicum annuum* L.). *Bul. Agron*. *35*(2): 112–117.

Tahery Y, Aini NAS, Abdul-Hamid H, Puad MA, Norlia B (2011). Status of root knot nematode disease on kenaf cultivated on bris soil in kuala terengganu. *World Appl. Sci. J*. 5(9): 1287–1295.

Taylor A, Sasser J (1978) Biology, identification and control of root-knot nematodes *(Meloidogyne species)* raleigh. (1st ed.). United States Agency for International. Edited by Taylor A, Sasser J. North Caroliba State University Graphics. North Carolina.

Wanda N, Barmawi M, Akin HM (2014). Pola segregasi karakter ketahanan tanaman Kedelai (*Glycine max* L. Merrill) terhadap infeksi *Soybean mosaic virus* populasi F_2 keturunan Taichung X Tanggamus. *J. Penelitian Pertanian Terapan*. 15(1): 54–60.

Wang C, Ulloa M, Duong TT, Roberts PA (2017). QTL analysis of transgressive nematode resistance in tetraploid cotton reveals complex interactions in chromosome 11 regions. *Front. Plant Sci*. 8: 1–12.

Wokoma EC, Tanimola AA, Chizzy ML (2015). Evaluation kenaf (*H cannabinus*) accession for resistance to *Meloidogyne incognita. Int. J. Dev. Res*. 5(3): 3645–3650.

Warner JN (1952). A method for estimating heritability. *J. Agron*. *44*(8): 427–430. https://doi.org/10.2134/agroj1952.00021962004400080007x.