



Research Article

Selection and Inheritance of Tomato Resistance against *Ralstonia solanacearum*

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ABSTRACT

Ralstonia solanacearum is a plant pathogen causes wilting which is a major obstacle in the cultivation of tomato plants. In plant breeding, knowledge of the source of resistance genes and inheritance patterns is important in the development of bacterial wilt resistant varieties. This study aimed to obtain bacterial wilt resistant lines and to find out the inheritance pattern of tomato resistance to bacterial wilt. Selection of resistant plant involved the selected breeding lines from irradiation and crossing collections of the Genetic Laboratory, Faculty of Agriculture, Universitas Gadjah Mada. Introduced lines of H-7996 and F₁ Permata and Timoti were used as a control. H-7996 as resistant parents and GM2 as susceptible parents, and their offspring include F₁ GM2 x H-7996, F₁ reciprocal, F₂, Back Cross 1 (F₁ x GM2), and Back Cross 2 (F₁ x H-7996) used in testing inheritance patterns. Inoculation was carried out 1 week after planting by pouring 100 ml of water suspension of *R. solanacearum* (10⁸ cfu/ml) on the roots. Completely Randomized Design (CRD) was used in this experiment. The scoring observation was carried out every week for one month. This study showed that Permata as a control was the most resistant, while Timoti and H-7996 were medium resistant. The CLN, G6, G8, and G7 lines were susceptible medium, yet only G8 and G7 with the smallest percentage of disease intensity and not significantly different than Timoti. The resistance gene to bacterial wilt on H-7996 was controlled by genes in the cell nucleus with additive-dominant gene action. Resistance to bacteria has a moderate level of heritability.

Keywords: inheritance, *Ralstonia solanacearum*, resistance, tomato

INTRODUCTION

Tomato (*Solanum lycopersicum*) is one of important fruit vegetables in the world. Tomato are not only used as fruit or vegetables but also as industrial raw materials. One of the obstacle in tomato production is bacterial wilt caused by *Ralstonia solanacearum* (Champoiseau & Momol, 2009). *R. solanacearum* is soil-borne pathogens (Hayward, 1991) become endemic in an area with attacks reached 71% (Sikirou *et al.*, 2017). *R. solanacearum* infects the plant through a natural opening or wound in the root, producing putrescine as a virulent pathogenic metabolite (Lowe-Power *et al.*, 2017), and colonize xylem. The bacteria cells cause blockage of xylem and inhibit the absorption of nutrients and water, hence the plants wilt (Arwiyanto, 2014). Bacterial wilt reduce tomato production by up to 91% (Yuliar *et al.*, 2015).

The control techniques, such as physical, chemical, biological and technical culture have been applied (Nawangsih, 2005). The control technique which is

easier, cheaper, and environmentally friendly is the use of resistant varieties. In Indonesia, the number of resistant varieties to bacterial wilt is slightly available. A study conducted by Rao *et al.* (1975) using 23 varieties from the USA and Philippines, only found one resistant variety to *R. solanacearum* isolates from India. This finding showed that geographical differences determine the pathogenicity of pathogens and the specific resistant characteristics of varieties.

Resources of resistance to *R. solanacearum* have been found, yet some are specific to certain races and only tomato cultivars of H-7996 have the most stable characteristic of resistance. That cultivar has a high survival rate up to 97% in 12 locations of 11 countries in Asia, America, and Australia. This cultivar is widely used as a subject to study resistance genes against *R. solanacearum* (Wang *et al.*, 2013). In Indonesia, H-7996 has been used by Arwiyanto *et al.* (2015) as a rootstock, suppress bacterial growth, to reduce the incidence of wilt and thus better

production. Breeding scheme of resistant tomatoes to bacteria is necessitated. The source of resistant genes, knowledge of inheritance schemes, type of resistance, and the mechanism of tomato resistance to bacteria have been mastered by plant breeders (Seah *et al.*, 2007), in order to the breeding of resistant varieties is in the right direction. The use of selected germplasm, resulting in good characteristics, yet it is needed to be assessed for resistance to *R. solanacearum*. Therefore, this study aimed to obtain bacteria resistant lines and to find out their inheritance and dominance schemes.

MATERIALS AND METHODS

The lines used for screening resistance to *R. solanacearum* were from Gamma-ray radiation collection of the Laboratory of Genetics, Faculty of Agriculture, Universitas Gadjah Mada (UGM): G4, G6, G7, G8, G9, and G10. Further generation lines collections from Genetic Laboratory of Genetics, Faculty of Agriculture, UGM were Gamato 1, Gamato 2, Gamato 4, Gamato 5, GM2 and CLN. The H-7996 lines recommended by AVRDC, F1 Timoti, and Permata was used as a resistant variety.

Cultivars for testing inheritance patterns of resistance to bacteria used were H-7996 as resistant cultivars and GM2 as susceptible cultivars. Parents of GM2 and H-7996 were crossed to produce F₁ GM2xH-7996 and the reciprocal crossing (H-7996xGM2). Then some F₁ seeds were planted together with both parents and the rest was stored for the next crop season. F₁ plants were partially left to self-pollinate to produce F₂ seeds and some were crossed to both parents to produce backcross (BC) offspring. Backcross 1 (BC_{1.1}) was F₁xGM2 and backcross 2 (BC_{1.2}) was F₁xH-7996. After harvesting, the seeds were sown in sterile media. Planting material used to estimate the number of controlling genes and interactions between genes was carried out by planting parents, F₁ and reciprocates (BC_{1.1}, and BC_{1.2}), F₂. The number of parent plants and F₁ was 30 plants, BC_{1.1}, BC_{1.2} were 150 plants, and F₂ were 250 plants, respectively. *R. solanacearum* used was race 1, biovar 3, phyllo type 1 collection of Phytopathology Laboratory was obtained from endemic areas of Seyegan, Sleman, the Special Region of Yogyakarta. Bacteria was grown on YPGA (Yeast Peptone Glucose Agar, 5 g yeast extract,

peptone 10 g, glucose 10 g, agar 15 g, distilled water 1.000 ml). Inoculation was carried out on plants have 5 leaves or 7 days after transplanting by drenching 100 ml of water suspension with a density of 1×10⁸ cfu/ml in the root area of the plant (Kim *et al.*, 2016).

Disease symptoms were observed every 7 days for 28 days after inoculation. The evaluation was carried out by scoring used by Robert *et al.* (1988):

- 0 : no symptoms
- 1 : 1–25% of wilted leaves
- 2 : 26–50% of wilted leaves
- 3 : 51–75% of wilted leaves
- 4 : 76–100% of wilted leaves

Classification of resistance used the mean method and disease intensity. Lines have a mean score of < 2 were categorized as resistant (R), 2 ≤ x ≤ 3 was medium resistant (MR), and > 3 was susceptible (S) (Kim *et al.*, 2016). Calculation of disease intensity used formula 1 (Arwiyanto *et al.*, 1994). Classifying the level of resistance to bacterial wilt based on disease intensity referred to Table 1.

$$IP = \frac{\sum_{i=1}^k kxnk}{ZxN} \times 100\% \quad (1)$$

k = scoring scale (k: 0, 1, 2, 3, 4)

nk = number of plants attacked by disease on a scale of k

nk = number of plants inoculated

Z = highest symptom scale

Table 1. Level of resistance to bacterial wilt

Disease intensity	Resistance Level
0	High Resistant (HR)
1 – 5	Resistant (R)
5 – 20	Medium Resistant (MR)
21 – 51	Medium Susceptible (MS)
> 51	Susceptible (S)

Source: Janaki & Putturaju (2012)

Estimation of genetic parameters used a joint scale test (Mather & Jink, 1982) with three genetic parameter estimators: [m], [d], and [h]. If the assumptions were not met, estimations were carried out with 6 parameters [m], [d], [h], [i], [j], and [l]. This test was conducted to determine the action of genes that control the resistance to *R. solanacearum*.

T-test was used to analyze the maternal effect (Singh & Chaudary, 1977). Estimation of the heritability value used the following formula (Warner, 1952):

$$h^2 = \frac{\sigma^2 A}{\sigma^2 A + \sigma^2 D + \sigma^2 I + \sigma^2 E} \quad (2)$$

$\sigma^2 A$ = additive variant

$\sigma^2 D$ = dominant variant

$\sigma^2 I$ = epistasis variant

$\sigma^2 E$ = environmental variant

The estimation of variant above was obtained from the calculation of the tested population variants (Mather & Jink, 1982). According to Stansfield (1991), if the value of heritability (h^2) < 0.2 was categorized as low, $0.2 < h^2 < 0.5$ was medium, and $h^2 > 0.5$ was high.

RESULTS AND DISCUSSION

The results of scoring variants analysis of wilt and disease intensity showed that there were differences in resistance between the tested lines/cultivars. Based on wilting score and disease intensity showed that all further generation lines from crosses were susceptible to bacterial wilt (Table 2), because tomato Gondol, as parents, has no source of resistance genes. Gondol tomatoes are special commercial tomatoes, yet very susceptible to bacterial wilt (Harjadi & Halim, 1980), hence lines from their offspring segregation were also susceptible to bacterial wilt. GM2 was classified as susceptible to *R. solanacearum*. Based on the wilt scoring, the irradiated lines have better resistance compared to the collections from Gondol with GM, except G4 was classified as susceptible. G9 and G10 were medium resistant; G8, G6, CLN, and G7 were resistant. G7 and G8 were not significantly different from H-7996 and Timoti, but only G7 was not significantly different from Permata.

Based on the classification of wilt scoring, G7, G8, G6, CLN, and H-7996 were classified as resistant, but based on classification the disease intensity was medium susceptible and medium resistant (Table 2). Permata as control was classified as high resistant in wilt scoring and disease intensity. G7 strain was a high resistant line, but based on the classification of disease intensity was medium susceptible, while

Table 2. Resistance of tomato lines against *Ralstonia solanacearum*

Line	Scoring	Category	DI (%)	Category
GM2	3.25ab	S	86.31a	S
Gamato 1	3.60a	S	90.48a	S
Gamato 5	3.5a	S	88.10a	S
Gamato 4	3.55a	S	76.99a	S
G4	3.05abc	S	76.39a	S
Gamato 2	3.05abc	S	76.99a	S
G9	2.20bcd	MR	54.96ab	S
G10	2.00cd	MR	49.80ab	MS
CLN	1.95de	R	49.01ab	MS
G6	1.85de	R	46.43ab	MS
G8	1.15def	R	29.76bc	MS
G7	1.00egf	R	25.40bc	MS
H-7996	0.75gf	R	13.10cd	MR
Timoti	0.60gf	R	20.63bcd	MR
Permata	0.15g	R	4.76d	R

Remarks: Values followed by different letter were significantly different according to DMRT ($\alpha = 0.05$). S = susceptible, MS = medium susceptible, MR = medium resistant, R = resistant, and DI = disease intensity.

H-7996 and Timoti were medium resistant. Several studies on H-7996 showed a different resistance level. According to Hai *et al.* (2008), H-7996 was resistant to Pss186 (race 1, biovar 4) and Pss4 (race 1 biovar 3), but high susceptible to Pss190 (race 1 biovar 4). *R. solanacearum* was a complex species because they are heterogeneous species (Arwiyanto, 2014). According to Lebeau *et al.* (2011), tomato plants have a high susceptibility to the diversity of *R. solanacearum* strains. Therefore, specific resistance to certain strains was present.

In the other hand, Laeshita & Arwiyanto (2017) stated that H-7996 cultivars are medium susceptible despite using the same line. This was probably due to using a different inoculation method. Laeshita & Arwiyanto (2017) used artificial wound to root during inoculation, but this study did not use that method. In this study, no wound was carried out because the conditions were expected like conditions in the field. According to Hayward (1991), the symptoms of wilting caused by infections by *R. solanacearum* are influenced by pathogenic strains, inoculation methods, and environmental factors such as temperature.

The screening results revealed that the lines of the UGM Genetic Laboratory collection had a lower resistance compared to H-7996 and resistant controls. Furthermore, H-7996 is used as a parent to analyze gene action from the bacterial resistance. H-7996 is

a pure strain hence can be used as a line to study inheritance resistance and has been widely used. The susceptible line used as parents was GM2. The results of reciprocal testing (Table 3) showed that F₁ resistance and reciprocity were not significantly different. This indicates resistance to bacterial wilting is controlled by genes in the cell nucleus and is not affected by cytoplasmic of female parents. The number of controlling genes and inheritance patterns was

estimated by using a combined scale test of the population of parents, F₁, F₂ and reciprocal (Figure 1). The controlling gene action that set a trait can be estimated based on the distribution of the frequency of these characteristics in the F₂ population. Scoring distribution in population F₂ showed the presence of two peaks leading to resistance and susceptibility. According to Acquah (2007), this result indicated that the gene controlling resistance to bacterial wilt is controlled by major genes.

Table 3. T-test between F1 and F1R

	N	Average	Variant	t cal.
F1	30	3.60	1.49	0.413ns
F1R	30	3.47	1.64	

Remarks: * significantly different; ns = not significantly different

Chi-square test (Table 4) showed the ratio of 3:1 and was not significantly different at the level of $\alpha=5\%$. A 3:1 comparison indicated that the controlling gene of this characteristic is a single gene and the resistant gene is a recessive gene. The distribution of scoring

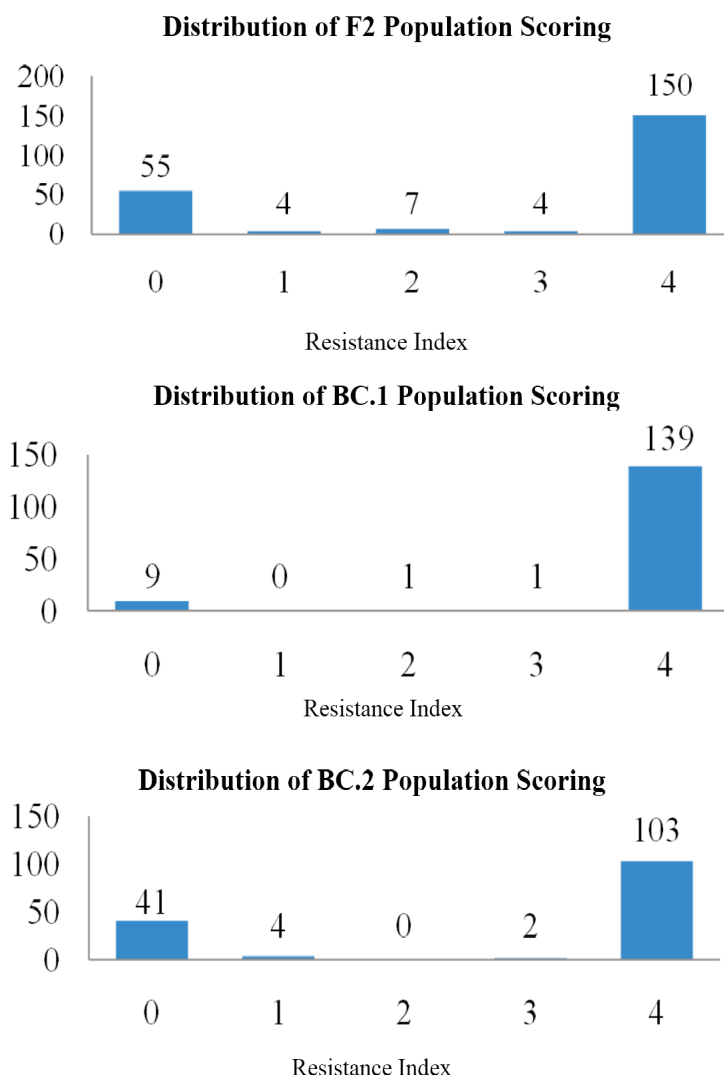


Figure 1. Diagram of distribution of population scoring F₂, BC_{1.1}, and BC_{1.2}; note: index score 0 (no symptoms/resistant); 1 (1–25% wilted leaves); 2 (26–50% wilted leaves); 3 (51–75% wilted leaves); 4 (76–100% wilted leaves/dead)

Table 4. Mendel Chi-Square Test

	Comparison	Observation Value		Significance Value		$\alpha = 0.05$ 3.84
		Resistant	Susceptible	Resistant	Susceptible	
Complete dominance	3:1	55	165	57	173	0.435ns
Suppressor genes	13:3			43	187	5.8*
Complementary genes	9:7			101	129	30.49*
Duplicate genes	15:1			14	216	126*

Remarks: * = significantly different; ns = not significantly different

BC_{1.1} (Figure 1) showed that the formation of one peak with a susceptible nature to *R. solanacearum*. If the dominant assumption was completed, then crossed F₁ (Aa) x susceptible parent (AA) will get BC_{1.1} 100% susceptible. In this study, BC_{1.1} (F₁ x GM2) showed the results as hypothesized, which was almost 100% susceptible. Assumptions on crosses BC_{1.2}: F₁ (Aa) x parent resistant (aa), will get a ratio of 1 Aa (susceptible): 1 aa (resistant). In this study, a comparison of 2.5 susceptible: 1 resistant was resulted from crossing and different as hypothesized. It is suspected that there was inter-locus interaction or epistasis.

Estimating genetic parameters using three parameters used the assumption that the controlling gene was a major gene, thus only additives (AA, aa) and dominance (Aa). Based on the combined scale test method (Table 5) with 3 parameters: [m], [d], and [h], showed that the additive-dominant concept was completed and that controlling the resistance to bacterial wilt was influenced by [d] additive gene and [h] dominant gene. The values of [d] and [h] resulted negative values, -0.38 and -1.395, respectively. This finding showed that the tendency of traits leads to susceptible parents were controlled by recessive genes. The values of magnitude [d] and [h] showed that the dominance was over dominance of susceptible parents. Based on the calculation of the degree of dominance there was over dominance of 3.65, but based on the test t-test with the assumption Ho: [d]

Table 5. Estimated value of genetic parameters

B	Alleged Value
m	3.63* ± 0.069
[d]	-0.38* ± 0.065
[h]	-1.395* ± 1.267
χ^2 cal.	3.192ns
[h]/[d]	3.65ns

Remarks: * = significantly different; ns = not significantly different

= [h] the value was 0.83 and the hypothesis was accepted. According to Mather and Jink (1982), perfect dominance occurred when [d] was equivalent to [h]. Therefore, the existing dominance was a perfect dominant. The calculation of heritability (Table 6) showed the value of 0.41. This means that the characteristic of inheritance was moderate. This may be the reason for the BC_{1.2} crossing not as hypothesized because the inheritance was classified as moderate. Opena (1994) stated that two backcrosses with resistant parents were enough to get a good level of bacterial wilt resistance.

Table 6. Variant values and heritability

	Value
Environment Variant (Var E)	1.55
Additives Variant (Var A)	1.77
Dominant Variant (Var D)	-0.30
Epistasis Variant (Var I)	1.21
Narrow Heritability (h)	0.41

Based on this study, it was concluded that the resistance control gene H-7996 against *R. solanacearum* isolates from Seyegan, Sleman (race 1, biovar 3, phyllotype 1) was the major gene with perfect dominance. The results obtained from this study were similar with Wang *et al.* (2000), using the strain Pss4 (race 1, biovar 3), reported that resistant genes were controlled by major genes, but when using strains GMI8271 were controlled by polygenic. Grimault (1995) and Thakur *et al.* (2004) reported that the controlling gene for bacterial wilt was controlled by a single recessive. Grimault (1995) conducted a field test using strain 8217 (race 1, biovar 1) stated that the resistant gene from H-7996 was controlled by a single gene. This differences indicated that the gene controlling resistance to the bacteria *R. solanacearum* varies depending on the specific

strains. These findings showed that there was a gene-for-gene concept for resistance to *R. solanacearum*.

CONCLUSION

The G7 and G8 lines have better resistance compared to the Genetic Laboratory collection lines. The action of resistant genes in H-7996 against isolates of *R. solanacearum* race 1 biovar 3 phyllotype 1 from Seyegan, Sleman was additive-dominant. The controlling gene resistant to bacterial wilt was controlled by genes in the cell nucleus with moderate levels of heritability.

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