The Inheritance of late blight resistance derived from *Solanum habrochaites*

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**Abstract** - Late blight caused by the oomycete *Phytophthora infestans* is a destructive tomato disease in Brazil and in other tropical and subtropical regions. The purpose of the present study was to analyze the inheritance of resistance to late blight and determine the genetic factors that contribute to the resistance in the inbred line “163A”. The Line “163A” resulted from an interspecific cross between *Solanum lycopersicum* and *S. habrochaites* f. *glabratum*, achieved by researchers at Universidade Federal de Viçosa. Inoculated field with mixture isolates of pathogen with 1000 spores mL⁻¹ and naturally infested field trials showed that the expression of “163A” against multiple isolates of the pathogen was stable. The genetic analysis supported the hypothesis of two recessive genes that controls the resistance. The scaling test of additive-dominance model showed that it is a good fit for the data, which confirms the absence or neglect of epistasis.

**Keywords:** *Solanum lycopersicum*, *Phytophthora infestans*, recessive, inheritance.

**INTRODUCTION**

Tomato cultivars suffer from as many as 200 diseases worldwide, of which 30 are routinely important. Out of these diseases, late blight, caused by the oomycete *Phytophthora infestans* Mont. De Bary, is a destructive tomato disease, and approximately 15-20 % of fresh tomato production costs in Brazil are for late blight control (Mizubuti 2001). While worldwide losses due to late blight and control measures are estimated to exceed $5 billion annually. The control of late blight heavily relies on the frequent application of protective fungicides, which are applied every 5-14 days. Current control methods are low efficiency and have serious operational implementation constraints, as high costs and labor demands. Late blight control is increasingly difficult due to high variability in *P. infestans*, the introduction of new pathogen isolates and increased resistance of the pathogen to fungicides (Kato et al. 1997). For cleaning tomato cultivation, using resistant cultivars is a desirable alternative to chemical control. The development of crops that possess durable genetic resistance provides the best prospect for efficient, economical and environmentally safe control of late blight (Mizubuti 2005, Bonnet et al. 2007).

In Brazil, the germplasm bank BGH (Banco de Germoplasma de Hortaliças), which belongs to Universidade Federal de Viçosa (UFV), has been established in 1960s. Currently, this germplasm is incorporated in breeding programs for new varieties represents as germplasm source of cultivated species (Silva et al. 2001). The characterization of over than 350 tomato accessions of BGH (http://www.bgh.ufv.br) resulted in defining many resistance sources, as well as to Tuta absoluta (Oliveira et al. 2009, Moreira et al. 2005), potyvirus (Zucchini yellow mosaic virus, ZYMV and Pepper yellow mosaic virus, PepYMV) (Moura et al. 2005, Juhász et al. 2008), whitefly (*Bemisia tabaci* biotype B) (Bernades et al. 2009), *Phytophthora infestans* (Abreu et al. 2008) and geminivirus (Tomato yellow spot virus, ToYSV) (Aguilera et al. 2008).
Attempts to breed late blight resistant tomato lines resulted in the identification of three dominant genes: Ph-1 on chromosome 7 (Clayberg et al. 1965, Peirce 1971), Ph-2 on chromosome 10 (Moreau et al. 1998) and Ph-3 on chromosome 9 (Chunwongse et al. 1998). Tomato varieties that carry the resistance genes Ph-1 or Ph-2 provide inadequate control against the local population of the pathogen (Cohen 2002). While Ph-3 gene has been incorporated into many breeding lines of fresh market and processing tomato due to its efficacy against broad range of *P. infestans* isolates. In recent study, the Ph-3 gene showed highly stability under field condition, unlike Ph-1 or Ph-2 (Elsayed et al. 2011).

Race-specific and polygenic resistance have been characterized and exploited in breeding, providing an efficient control of disease severity (Thabuis et al. 2004). The high variability in *P. infestans* populations throughout the world, especially for virulence, has made race-specific resistance genes almost useless in disease control (Andrivon 1994). With the lack of durability of resistance with single dominant genes that result in hypersensitive resistance (HR), it is probable that new resistance genes that result in HR will not be durable. More emphasis is being given to transfer of quantitative trait resistance to commercial tomato cultivars. Resistance to late blight has also been observed in wild *S. habrochaites* (Lobo and Navarro 1987, Kim and Mutschler 2000, Abreu et al. 2008). An interspecific *F₁* progeny (*S. lycopersicum* L. cv. Santa Clara x *S. habrochaites* f. *glabratum*) accession BGH 6902 exhibited resistance to numerous *P. infestans* isolates under field conditions of Viçosa, MG state (Abreu et al. 2008). The purpose of this work was to study the inheritance of resistance to late blight in the inbred line “163A” originated from the interspecific cross between *S. lycopersicum* L.cv. Santa Clara and *Solanum habrochaites* f. *glabratum* and investigate the efficiency of *Solanum habrochaites* as a source of resistance that could be integrated into breeding programs under tropical highly humid conditions of Brazil.

**MATERIALS AND METHODS**

**Plant material**

The inbred ‘163A’ is an advanced line that was originated from interspecific cross *S. lycopersicum* L. cv. Santa Clara x *Solanum habrochaites* f. *glabratum*. The ‘163A’ was obtained from successive generations of self-pollination and selection for late blight resistance program at the Department of Plant Science, Universidade Federal de Viçosa. The ‘163A’ was crossed to the variety ‘New Yorker’ to obtain the *F₁* seeds. The ‘New Yorker’ is a susceptible variety that has the *Ph-1* resistance gene to late blight. In the following season, the *F₁* plants were self-pollinated to obtain the *F₂* seeds. The parents, *F₁* and *F₂* generations were grown during the winter of 2009 at the Experimental Field of Plant Science Department, Universidade Federal de Viçosa, located in the city of Viçosa, Minas Gerais state, at lat 20° 45’ 14” S and long 42° 52’ 53” W and alt 648 m asl.

**Pathogen isolates and preparation of inoculum**

In order to avoid both the specific-race resistance and possible epistatic effect of vertical resistance, mixture isolates of *P. infestans* collected from several regions of tomato production fields was applied. The *P. infestans* isolates were of the A1 mating type and belonged to US-1 clonal lineage. At early morning, infected leaves were collected from the commercial fields. These leaves were transferred in polyethylene cases and kept under cold condition until reaching the laboratory. The infected leaves were placed in 30 x 40 x 5 cm plastic trays in order to multiply the primary inoculum. A single ply of facial tissue paper was plastered with water on the bottom of the tray to maintain adequate humidity for inoculum development. The trays were kept in a dark chamber at 18–20°C for 24 h. After that, the surface of fresh mycelium on the underside of leaves was lightly brushed with a toothpick. The sporangia suspension was kept in the dark at 11–12 °C, for 90–100 min, in order to release the zoospores (Nilson 2006). Uniform suspension was used to obtain an accurate sporangia count. The concentration was determined with a hemacytometer adjusted to 10⁵ sporangia mL⁻¹. The inoculation was accomplished in June 2009 at 7:30 PM, after about 2 hours of sunset, using manual backpack sprayer (20 liter volume), and applying 20 mL of the sporangia suspension per plant.

**Quantify the resistance**

Under the conditions of natural infection and artificial inoculation, the genotypes were screened against late blight. The first observation was recorded after 4 days of inoculation and then every 4 days during June 2009. The disease severity was recorded based on the proportion of area or amount of plant tissue that showed the symptom. A thin film of water on the plants using micro sprinklers (full-circle 5 m, 325ml/min/micro sprinkler) was applied as auxiliary condition for spore germination. The spray system was adjusted to turn on every 3 hours for 15 minutes over the day. In order to facilitate spore germination, the micro sprinklers were kept turned on for approximately 2 hours prior to field inoculation to provide a thin film of water. The average maximum and minimum temperatures were 25.2 and 13.7 °C, respectively, during disease development, with average relative humidity 85.7%.
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Data collection

To evaluate the disease severity of late blight, the whole plant leaves were submitted to screening. It was best to record readings independently without knowing the value given at the previous reading at each date, such as having someone else write in the field book or by using a cassette recorder. The selection to late blight resistance was done based on the minimum values of severity at the end of epidemic ($Y_{\text{max}}$).

Data analysis

Study of inheritance for resistance in a Mendelian approach was done by grouping plants into resistant, moderate resistant and susceptible classes. Three ratings were used in the classification of the resistance based on interval range of the parents (Table 1) as (1) susceptible 71-100% severity; (2) moderate 31-70% and (3) resistant 0-30%. Segregation ratios were tested for goodness-of-fit to theoretical ratios for the hypothesis that two recessive genes control the resistance. Chi-square ($\chi^2$) test was performed on the segregating population using numerical data. Quantitative analysis to resistance was accomplished applying generation analysis ($P_1$, $P_2$, $F_1$ and $F_2$ generations). Broad-sense heritability estimate ($H^2$) was obtained from the variance components of the variance estimates of $P_1$, $P_2$, $F_1$ and $F_2$ (Mather and Jinks, 1984). Narrow-sense heritability ($h^2$) was estimated by the formula $h^2 = \frac{2h^2}{(2 + k^2)}$, where $k$ is the average degree of dominance in the cross. The use of this formula assumes no epistasis, no link among loci, and equal gene effects for each loci. A scaling test for the additive-dominance model was performed with $t$-test $t = \frac{C}{\sqrt{V_C}}$ of the parameter ($C = 4$),

$$V_C = 16V_{F_2} + 4V_{F_1} + V_{F_1} + V_{F_2}$$

as indicated by Mather and Jinks (1984), with degree of freedom equal to the number of individuals in ($F_2 + F_1 + P_1 + P_2 - 4$). Minimum number of genes controlling resistance was estimated by the Wright’s minimum effective factors, calculated with $F_2$ generation data, according to Cruz et al. (2004), by the formula $n = \frac{R^2}{8V_G}$, where $R$ is the total amplitude in $F_2$ population and $V_G$ is the genetic variance in $F_2$. The means, variances, Chi-square test and other genetic parameters of the $Y_{\text{max}}$ were estimated using GENES software program (Cruz 2006).

RESULTS AND DISCUSSIONS

Clear differences in severity among tomato genotypes were observed after inoculation with $P. infestans$ under field conditions during winter 2009. Although laboratory methods can be used in resistance assay, the most effective and reliable methods are generally accepted to be natural infections or inoculated test plots under field conditions. After four days of inoculation, the disease symptoms began to emerge slightly. In the following days, the heavy rains and low temperature stimulated disease development.

Qualitative analysis

The qualitative analysis of inheritance of resistance in the parents, offspring and $F_2$ generations using test $\chi^2$ showed that the goodness of fit of the $H_2$ hypothesis that the qualitative genetic model (9:6:1) of resistance to late blight is fit with probability of 28.46% (Table 1). Furthermore, the qualitative analysis showed that the genetic model for the inheritance to resistance based on two recessive genes would not be discarded considering the genotypes (A-B-) as susceptible with presence of partial dominance for the susceptible parent. The genotypes of (A-bb/aaB-) are moderate resistant and when the both alleles being recessive (aabb), exhibit resistant. The frequency distribution of the parents, $F_1$ and $F_2$ individuals showed that for the susceptible parent New Yorker, the severity ranged from 71 to 100% with majority

### Table 1. Goodness of fit ($\chi^2$ and $P$) for qualitative genetic model of resistance to late blight ($P. infestans$) in a population of a cross between the resistant ‘163A’ and the susceptible ‘New Yorker’.

| Generation, 1: Tance to TYLCD | Total                  | Interval range (% severity) | No. of plants | Two recessive genes (9:6:1) | Goodness of fit |
|-------------------------------|------------------------|----------------------------|---------------|-----------------------------|-----------------|
|                               |                        | Min. | Max. | Number of plants per symptom class | Expected Numbers/ratio of the $F_2$ | Goodness of fit |
|                               |                        |      |      | $S$ | $M$ | $R$ | $S$ | $M$ | $R$ | $\chi^2$ | $P$ |
| New Yorker                    | 20                     | 71   | 100  | 20  | -- | -- | -- | -- | -- | -- | -- |
| 163A                          | 19                     | 17   | 30   | --  | -- | 19 | -- | -- | -- | -- | -- |
| $F_1$                         | 28                     | 75   | 100  | 28  | -- | -- | -- | -- | -- | -- | -- |
| $F_2$                         | 99                     | 15   | 100  | 53  | 36 | 10 | 55.8 | 37.1 | 6.1 | 2.51 | 28.46 |

*in the $F_2$ generation, two plants had 35% and 44% of severity. The interval based on the susceptible and resistant parents rang (1) susceptible 71-100% severity; (2) moderate 31-70% and (3) resistant 0-30%.
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Table 2. A genetic model for the inheritance of qualitative resistance against Phytophthora infestans in 163A inbred line based on two recessive genes.

| Genotype  | Proportion | Phenotype       |
|-----------|------------|-----------------|
| A- B-     | 9/16       | Susceptible     |
| A- bb     | 3/16       | Moderate resistant |
| aa B-     | 3/16       | Moderate resistant |
| aa bb     | 1/16       | Resistant       |

Segregation ratio in F1 population S:M:R = 9:6:1

(13 individuals) located in the 91-100% class. For the resistant parent 163A, the majority located in two classes ranged from 11 to 30% of severity. The individuals of F1 generation were located in the susceptible class with the same range of susceptible parent but with less number of individuals in the final degree of susceptibility (10 plants). This distribution of the F1 individuals emphasizes the dominance of susceptibility over the resistance. Furthermore, the frequency distribution of the F1 individuals in three phenotypic classes of resistant, moderate resistant and susceptible with frequency of 10.10%, 36.36% and 53.53% respectively, revealed the existence of two different loci with recessive gene effect controlling the resistant in S. habrochaites. It is worth mentioning that the moderate probability value (28.46%) of ² test in the present study could be in part due to the use of a rather relatively small population size or the possibility of including certain individual from one category to other as a result of the closed interval within the categories somewhat. To overcome this problem, it was modified the resistant category with interval 0-30, instead of 0-31, which considered previously discarding the individuals with abnormal values.

Quantitative analysis

The line ‘163A’ showed fixed behavior regarding to resistance to late blight with average mean of 24% for Ymax, whereas the variety ‘New Yorker’ exhibited average mean of 92.59% for Ymax as fully susceptible. The F1 individuals showed mean values of Ymax closed to the susceptible parent with mean of 84.87% of Ymax (Table 3), however, they had the same interval of susceptible parent. The mean performance of F2 population decreased, compared to their F1 generation, in 19.3% for Ymax (Table 3). This result could be attributed to the effect of dominance toward the susceptibility (Table 4).

The heritability in broad (Hb) and narrow sense (Hns) was 76% and 58% for Ymax and revealed the magnitude of the environmental factors on the total variation. According to the current model, the minimum number of genes controlling resistance was 1.9 ± 2.0 genes, estimated by the Wright’s minimum effective factors, and calculated with F3 generation (Table 4). This consistency with the previous finding resulted from the qualitative analysis demonstrate two recessive genes controlling the resistance in 163A. Although the resistance that was reported in this research was controlled by tow genes, the low heritability that was observed are often associated with quantitative traits, which could be attributed to the large interference of the environment factors on the expression of the trait studied (Ramalho et al. 2000). Similar finding was reported by Foolad et al (2002), which demonstrated the heritability of resistance to early blight ranging from 65 to 71%. In addition, the low heritability could be attributed to the fact that the resistance measures made by the severity are highly affected by the environmental factors, escape and Human error. Nevertheless, the results obtained from the qualitative analysis of inheritance coincide with the other quantitative analysis for resistance.

The estimated average degree of dominance was 0.77 for the severity at the end of epidemic trait indicating partial dominance (0 < d < 1), revealed that the heterozygote (F1) has a value that lies between the mean of the two lines, and 

Table 3. The means and variances of the final severity (Ymax) for parents, F1, and F2 population inoculated with P. infestans and Joint scaling test of additive-dominance model.

| Generation | (Ymax) |
|------------|--------|
|            | No of ind. | Mean | Variance | V(µ) |
| P1         | 20     | 92.59 | 92.46    | 4.62 |
| P2         | 19     | 24.00 | 23.018   | 1.15 |
| F1         | 30     | 84.87 | 230.53   | 7.68 |
| F2         | 99     | 67.75 | 40189.14 | 318.97 |

Ymax is the severity at the end of epidemic respectively, scale, C=4 including P1,P2,F1 and F2 generations, degree of freedom (167)

Table 4. The genetic parameters of the final severity (Ymax) for the F2 segregating population NY’136A’ inoculated with P. infestans.

| Parameter | Disease trait |
|-----------|---------------|
|           | Ymax          |
| Phenotypic variation | 605.9 ± 84.84 |
| Environment variation | 144.1 ± 21.96 |
| Genotypic variation | 461.8 ± 92.21 |
| Hb (%)     | 76 %          |
| Hns (%)    | 58 %          |
| Heterosis  | 44.34         |
| Average degree of dominance | 0.77 |
| Maximum value (F1) | 100.00 |
| Minimum value (F1) | 15.00 |
| Number of genes | 1.9 ± 2.0 |
homozygotes parents, but toward the susceptible one. The qualitative analysis revealed complete dominance towards the susceptible parent where the individuals of heterozygote (F_1) have lies in the same susceptible parent categories. This slight discrepancy in the degree of dominance estimated through the two approaches could be attributed to the fact that the qualitative approach is based on numerical classes and treats in them individually, in contrast to quantitative approach, whose genetic parameters are resulted from the means and variances of each generation. This type of resistance that is associated with reduction in the time course of development of symptoms is a desirable trait for plant breeder since it is often effective across a broad range of pathogen races or strains (Parlevelet 1979). The average degree of dominance towards the susceptibility indicates that partial recessive gene action controlled the resistance, whereas the inheritance of resistance to other pathogens such as *Ralstonia solanacearum* and *Colletotrichum coccodes* in tomato are quantitative with partial dominance of the alleles in tandem with the higher AUDPC (Neto et al. 2002; De Castro et al. 2007).

This wide range of severity values observed among homogenous plants of the resistant and susceptible parents indicate that the resistance to *P. infestans* is affected by environmental factors. Similar expression was recorded by the homogenous-heterozygous F_1 plants (AaBb) whose severity ranged from 75 to 100% scored as susceptible. Similar findings were reported by Irzhansky and Cohen (2006), who found that F_1 plants exhibited various levels of moderate resistance and F_2 plants segregated 3:6:7 resistant: moderately resistant: susceptible, respectively. Their data supported the hypothesis that race-non-specific resistance in *S. pimpenellifolium* L3707 is controlled by two independent genes, but partially-dominant and dominant epistatic effect. The average degree of dominance is estimated as the square root of the average squared degree of dominance. The average is over all loci and over all heterozygotes possible at each locus. In this method, each squared degree is weighted according to the frequency of the heterozygote and according to the squared difference between the genetic values of the corresponding two homozygotes. Possible biases in the average degree of dominance were previously discussed. Nonetheless, other factors such as the bias due to different signs of individual degrees of dominance, to epistasis and to linkage could be affected by the estimation of this parameter.

It is noteworthy that previous studies demonstrated that the current tomato line derived from the interspecific cross is a good source of resistance to a range of *P. infestans* that infects tomato in Brazil (Fiorini et al. 2010). Therefore, the results of this work strongly emphasize the potential interest of “136A” as donor of broad-spectrum resistance for late blight control in tomato breeding programs. Evidence for the stability of the resistance under extremely high induced and natural late blight infection pressure was provided in this study. Interestingly, in these conditions, symptoms could appear in plants of inbred lines.

The genetic analysis of resistance demonstrated that two major recessive loci control the resistance to late blight in “163A”. Recessive resistance is associated with the lack of factors/functions needed for pathogen infection. Therefore, it was possible to hypothesize that the plant host factor needed for an effective interaction of the pathogen is altered in the resistant inbred line tested in this research. Quantitative analysis also suggested the involvement of at least one more locus in the resistance, and scale test of additive-dominance mode for both disease variables predicted the predominance of this model of gene effect for resistance to late blight, which was tested in this study(Table 3). The relatively simple mode of inheritance and fairly high heritability described here make feasible the introgression of the resistance from line ‘163 A’ into tomato commercial varieties. These genes have to be selected during each round of backcrossing, including progeny test after every singular generation of backcross (e.g., BC_1, BC_2, BC_3), in order to select the individuals that possess the recessive alleles controlling the resistance. In addition, the use of molecular markers could reduce the size of populations since only homozygous plants would require further screening. This could increase breeding efficiency and reduce screening costs.

The similar level of severity observed between the F_1 and the New Yorker parent indicates that at least two dominant genes are involved in the higher level of pathogen susceptibility. Furthermore, the 9:6:1 segregation ratio of the F_2 progeny suggests that the resistance found in the 163A line requires a homozygous recessive genotype at two genes other than the non-segregation Ph-3 gene, and these additional genes not linked to Ph-3. Is it well-known that the most useful resistance to late blight reported to date is provided by the Ph-3 gene derived from *S. chilense* (Miranda et al. 2010, Elsayed et al. 2011), which is not stable under high disease pressures, but present minor frequency of resistance overcame (25.8%) compared to Ph-1, (88.7%) and Ph-2, (64.5%) (Miranda et al. 2010). However, the Ph-3 gene exhibited fixed resistance against the current isolates of southern of Brazil region (Elsayed et al. 2011).

Furthermore, combining both resistant genetic makeup could provide more efficient late blight control. However, it should be noted that this kind of resistance could be ef-
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Ineffective through pyramiding both dominance and recessive genes that could reduce incidence in nature, as restricted *P. infestans* in plants, at least until reaching the unfavorable condition that might reduce pathogen availability. Thus, based on the data of this study, a lower risk of late blight spread is expected by using the resistance shown in this research, since reduced primary inoculum spread is expected because of a lower propensity of plants to be infected. Therefore, the tomato line described here could be a valuable source of resistance to late blight. Based on the finding of the present study, suggesting new genes in *Solanum habrochaites* with recessive alleles *ph-6* and *ph-7* controlling the resistant to late blight in the inbred line "163A", originated from the interspecific cross *S. lycopersicum* L.cv. Santa Clara x *Solanum habrochaites* f. *glabratum*.

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**Heredity of resistance a requeima derivada de *Solanum habrochaites***

**Resumo** - Requeima causada por Phytophthora infestans é uma doença destrutiva de tomate no Brasil e em outras regiões tropicais e subtropicais. O objetivo do estudo foi o de analisar a herança da resistência à requeima e determinar os fatores genéticos que contribuíram para a resistência na linhagem '163A'. A linhagem '163A' resultou de cruzamento interespecífico entre *Solanum lycopersicum* e *S. habrochaites f. glabratum*. As plantas foram avaliadas por infecção natural e por inoculação com uma mistura de isolados do patógeno com 1000 esporângios mL⁻¹. A expressão de '163A' contra múltiplos isolados do patógeno foi estável. A análise genética suportou a hipótese de dois genes recessivos controlando a resistência. O teste do modelo aditivo-dominante mostrou bom ajuste para dados que confirmam a ausência ou negligenciam epistasia.

**Palavras-chave:** *Solanum lycopersicum*, Phytophthora infestans, recessivo, herança.

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