

**Breeding of Tomato for Resistance to Bacterial Wither****ABSTRACT**

Bacterial wither is a disease that is of global importance because it is difficult of control and often compromises the whole crop. The use of resistant varieties is the main form of control of this disease. The objective of this work was to carry out a literature review with the main factors related to the genetic breeding of tomato plants aiming at resistance to bacterial wither. It was found different information related to the genetic control of tomato resistance in relation to the number of genes and their interaction due to the high genetic diversity within the *Ralstonia solanacearum* species complex. The high host-pathogen interaction reflects on different breeding strategies depending on the environment and the source of resistance used.

*Keywords: Solanum lycopersicum; Ralstonia spp.; heritage; plant breeding.*

**1. INTRODUCTION**

The tomato has as its center of origin the Andean region that covers part of Chile, Colombia, Ecuador, Bolivia and Peru [1]. In Mexico it was the place where its domestication by indigenous tribes took place, integrating to the Aztec culture [2]. The introduction of this culture in Brazil occurred in the late century XIX by European immigrants [3].

The botanical classification of the tomato underwent several modifications over time. In the middle of century XVI the first botanists they classified as *Solanum pomiferum*. Tournefort in 1694 named it as *Lycopersicon*, a century later Linnaeus (1753) termed the genre again as *Solanum*. Miller classified this vegetable twice as *Lycopersicon* (1754) and *Lycopersicon esculentum* (1768) [4]. After morphological and molecular studies the tomato was re-assigned to the genus *Solanum*. Currently, its taxonomic classification is as follows: Magnoliophyta division, Magnoliopsida class, Solanales order, Solanaceae family, *Solanum lycopersicum* species. In addition to the cultivated species *S. lycopersicum* there are twelve other wild species [5, 6].

The tomato is a dicotyledonous, herbaceous, with flexible hairy stem and soft when young, becoming fibrous and angular with the passage of time. The leaves measure 11 to 32 cm in length and are composed of an odd number of leaflets. They are alternated and petiolate, of oval to oblong form. It is a plant of habit of indeterminate or determined growth [7].

The root system is composed of main root, secondary and adventitious. The main or pivoting can reach 5m deep. Secondaries are stimulated when the main and adventitious root undergo stress in transplant. In general, 70% of the root system is in the first 20 cm of the soil surface [1, 8].

41 It is an autogamous species, with a natural crossing percentage in general, lower than 5%  
42 [9]. The flowers are small, with a diameter varying from 1.5 to 2 cm. Are hermaphrodites with  
43 cleistogamy, corolla and yellow stamens small size. They have five sepals, five wide  
44 lanceolate petals and six anthers. Each plant can have 20 simple or branched  
45 inflorescences, with four to eight flowers each. The anthers are welded forming a cone that  
46 surrounds the stigma. The anthesis occurs in two flowers at a time in each inflorescence [9,  
47 10].

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49 The fruits are fleshy, succulent berries, with size and mass differentiated according to the  
50 cultivar, being bilocular, trilocular or plurilocular [7, 11]. They consist of film, pulp, placenta  
51 and seeds. Their colors may vary from yellow to red-orange, depending on the lycopene /  $\beta$ -  
52 carotene ratio [12]. The fruit is of the climacteric type and can complete the maturation after  
53 the harvest and, usually develops in the period of seven to nine weeks after fertilization of  
54 the ovum [13].

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56 The seeds are small, oval, of gray cream color, possessing 2 to 3 mm in diameter [14]. The  
57 type of cultivar greatly influences the number of seeds, having some more than 200 per fruit.  
58 For germination the optimum temperature is between 18 to 24 °C, under conditions of  
59 temperature outside the ideal, germination delay and reduction in emergency uniformity may  
60 occur [15]. The vegetative phase of the tomato is very short, as flowering and fruiting occur  
61 along with vegetative growth [15].

62  
63 The tomato is a perennial plant, but due to its form of cultivation it is explored as annual [8].  
64 This culture adapts to a wide variation of latitude, cultivation methods, types of soil and  
65 temperatures [1]. Most cultivars have a cycle of 95 to 125 days. However, the cultivation  
66 period depends on climatic conditions, soil fertility, irrigation intensity, pest / disease attack  
67 and planting season [11]. Despite adapting well to various cropping situations, the ideal for  
68 culture is a cool, dry climate, with temperatures between 20 °C to 25 °C per day and 11 °C  
69 to 18 °C per night. Temperatures above 35 °C hinder the development of the plant and  
70 fruiting by providing abortion of flowers and falling of new fruits [8].

## 71 72 **2. BACTERIAL WITHER IN TOMATO**

73  
74 The cultivated tomato (*Solanum lycopersicum*) has a narrow genetic base, which makes a  
75 species more susceptible to biotic stresses. Thus, it is interesting that as cultivars show  
76 resistance to the greatest number of pests and possible diseases, especially as difficult to  
77 control, such as: fusion wither, stemphylium stain, bacterial wither, vertical wither, turns  
78 head, geminivirosis, meloidoginose and bacterial wither [11].

79  
80 The various wild species of tomato are of great importance for breeding, serving as a  
81 germplasm bank with multiple characteristics. Among them, we can mention: *S. hirsutum*:  
82 resistance to bacterial canker, black pint, septoriosi, tomato moth and mites; *S.*  
83 *peruvianum*: resistance to root knot nematodes, *Verticillium dahliae* of wither, black pint,  
84 head turns and bacterial canker; *S. pennellii*: resistance to mites and fusarium wither and *S.*  
85 *pimpinellifolium*: resistance to bacterial canker, black pint, fusarium wither, re-burn,  
86 stemphylium stain and bacterial wither [16].

87  
88 The first classification of the causative agents of bacterial wither was as *Bacillus*  
89 *solanacearum* by [17]. Over time, the following nomenclatures were adopted: *Bacterium*  
90 *solanacearum* [18], *Pseudomonas solanacearum* [17, 19], *Phytomonas solanacearum* [17,  
91 20], *Burkholderia solanacearum* [17, 21] and *Ralstonia solanacearum* [17, 22]. According to  
92 [23], *R. solanacearum* is considered a complex of species divided into phylotypes (4),  
93 sequevares (59) [24], clades (8) [25] and clones [23].

94  
95 From the phylogenetic analysis of the partial sequence of the endoglucanase gene and the  
96 ITS region, DNA-DNA hybridization, biochemical, cultural and physiological characteristics  
97 [26] proposed the taxonomic reclassification of the *R. solanacearum* complex in three  
98 independent species and subspecies. *Ralstonia pseudosolanacearum* consists of isolates  
99 belonging to phylotypes I and III, originating in Asia and Africa, respectively. *R.*  
100 *solanacearum* by phylotype II isolates (IIA and IIB), originated in the American continent and  
101 that probably possess two subspecies. The isolates of phylotype IV originated from Indonesia  
102 were reclassified into three subspecies of *R. syzigii*, where *R. syzigii* subsp. *indonesiensis*  
103 grouped the wilt-causing isolates of *Ralstonia* in Solanaceae, *R. syzigii* subsp. *syzigii* the  
104 isolates previously denominated of *R. syzigii* and as *R. syzigii* subsp. *celebesensis* of blood  
105 disease bacterium [26].  
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107 The species of the *R. solanacearum* complex are gram negative, their format is straight rods  
108 or slightly curved, with approximately 0.5-1.0 x 1.5-4.0 µm. Are non-sporogenic, mobile  
109 through one or more polar flagella and aerobic. Its growth occurs in temperature between 25  
110 and 35 °C [27]. These bacteria inhabit the soil and invade the root system by means of  
111 wounds, multiplies rapidly within the xylem and hereby is distributed throughout the plant.  
112 The result of colonization is the obstruction of the vessels by the accumulation of  
113 exopolysaccharides, blocking the translocation of water and nutrients. The main symptoms  
114 are darkening of the xylem vessels and sudden wither with no change in green coloration.  
115 The darkening of the vessels is due to the transport of substances resulting from the  
116 oxidation of phenols, resulting in melanin. It is worth mentioning that depending on the  
117 combination of several factors the disease can appear in any stage of development of the  
118 tomato [28, 29, 30].  
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120 As for most phytobacteria, controlling bacterial wither is very difficult. Therefore, it is  
121 recommended to make the integrated management, since the use of isolated measures is  
122 not efficient to avoid losses. Among the isolated measures, chemical control has low  
123 efficiency and is extremely damaging to the environment [31]. Some recommended control  
124 measures are: soil water management in order to avoid waterlogging; to avoid injuries  
125 caused by nematodes, insects or agricultural implements; avoid moving soil from disease  
126 outbreaks to other areas; elimination of diseased, infected and invasive volunteers from the  
127 Solanaceae family; perform crop rotation for at least one year with grasses; grafting on  
128 resistant grafts and the use of resistant cultivars [32, 33].  
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130 In Brazil and in the State of Pernambuco, the species *R. pseudosolanacearum* and *R.*  
131 *solanacearum* [24, 34] have been reported so far. It is believed that *R. solanacearum* has  
132 Brazil as the center of origin and diversity, while *R. pseudosolanacearum* was introduced  
133 from Asia. The disease is present in all mesoregions of the State of Pernambuco, causing  
134 great damage to the tomato crop of the State [35]. Thus, it is clear the importance of the  
135 breeding of plants aiming the resistance to bacterial wither in an attempt to mitigate the  
136 damages caused by this disease in the tomato crop.  
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### 138 3. PLANT BREEDING FOR RESISTANCE TO BACTERIAL WITHER

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140 The use of resistant cultivars is the most efficient way to control bacterial wither in tomato  
141 plants per it presents low cost, low impact on the environment and easy adoption by the  
142 producer [36, 37].  
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144 To become the plant breeding aiming the efficiency of bacterial wither resistance, it is  
145 necessary to emphasize that in Brazil the *R. solanacearum* complex presents a great  
146 genetic diversity. This is composed by 13 **sequевares** of Solanaceae (I-17, I-18, IIA-41, IIA-

147 50, IIA-58, IIA-59, IIB-2, IIB-25, IIB-28, IIB-54, IIB-55, IIB-56 and IIB -57). These four  
148 sequelae occur in the tomato crop: I-18, IIA-41, IIA-50 and IIB-54 [24, 34, 38, 39].

149

150 In the State of Pernambuco (Agreste and Forest Zone) were detected sequevars the I-17  
151 and I-18 which correspond to *R. pseudosolanacearum*, IIA-58 and IIA-59 representing *R.*  
152 *solanacearum* [24]. According to [39] in the semi-arid of Pernambuco are present the  
153 sequevars I-17 and I-18 of *R. pseudosolanacearum*, and sequevars IIA-50, IIA-58 and IIA-  
154 59 *R. solanacearum*. According to the same author, *R. pseudosolanacearum* is prevalent in  
155 Agreste and *R. solanacearum* in the **São Francisco** and **Sertão mesoregions**.

156

157 Survey work on complex species *R. solanacearum* in a given region is of paramount  
158 importance for the improvement of tomato aiming at resistance to bacterial wither. It is  
159 necessary to conduct programs based on the prevalent species and using local isolates to  
160 represent the situation in the screening stages from the inoculation of the pathogen [40].

161

162 In addition to understanding the diversity of the *R. solanacearum* complex, it is necessary to  
163 identify the sources that can be used in the development of resistant cultivars. In the  
164 literature, there are studies that identify sources of resistance in tomato germplasm [41, 42].  
165 Among these there are some accessions of *Solanum pimpinellifolium* and even of the  
166 cultivated species *Solanum lycopersicum* [43]. In the literature there are reports mainly of the  
167 following resistant cultivars Saturn, Venus, Caraiba, Hawaii 7996, Hawaii 7997, Hawaii 7998,  
168 Yoshimatsu, Drica and CRA-66. The cultivar Hawaii 7996 is considered international  
169 standard of resistance to bacterial wither, being used in several studies in an attempt to  
170 understand the genetic mechanism of resistance [9].

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172 At the molecular level, QTLs were found on chromosomes 6 and 4, which together represent  
173 56% of the resistance [44]. Recent work using the Hawaii 7996 source of resistance  
174 identified QTLs on chromosomes 12 (Bwr-12) and 6 (Bwr-6) ( ). The presence of QTL Bwr-6  
175 represents a challenge for plant breeding, because it is in association with small fruits or that  
176 can crack when they are ripe, and with susceptibility to of the galls nematodes (*Meloidogyne*  
177 spp.) and begomovirus [37, 45].

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179 According to [46] obtaining a stable cultivar is very difficult, due to the resistance of the *R.*  
180 *solanacearum* complex species to be specific to the locality. With the cultivation of these  
181 cultivars, it is necessary to carry out studies aiming at an integrated control, reducing the  
182 selection pressure to avoid the rapid supplanting of the resistance [47]. [48] evaluated 35  
183 sources of resistance to bacterial wither in 11 countries and observed for most sources  
184 different levels of disease incidence. The local specificity may be related to the dependence  
185 of environmental conditions, mainly in relation to temperature and humidity, as well as the  
186 pathogen diversity in each country [49].

187

188 According to [40] there are some fundamental points as strategies for breeding aiming at  
189 resistance to bacterial wither. i) the cultivars developed must be resistant and with desirable  
190 agronomic characteristics; ii) the cultivars grown must withstand local isolates and iii) most of  
191 the cultivars developed have the genetic control of the polygenic resistance, making it  
192 difficult to transfer the alleles.

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194 In Brazil, the cultivar Yoshimatsu was developed by INPA, which shows high resistance to  
195 bacterial wither. This cultivar allows the extraction of resistant and fruit-quality lines to meet  
196 market requirements [9, 31]. The genetic control mechanism in the Yoshimatsu cultivar  
197 needs to be studied, since most of the work was done with other sources.

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#### 200 4. STUDY OF GENETIC CONTROL OF RESISTANCE TO BACTERIAL WITHER

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At 35 years after the rediscovery of Mendel's laws, in an attempt to understand the genetic control of the characters in progenies, there was a division of schools. In the first, called Mendelian school, it was only believed that the distribution of the characters was discreet. In the second school, called biometrics, it was argued that most of the characters had continuous distribution. In fact, what defines the type of distribution is the number of genes and the environmental effect, being able to meet the assumptions of the two schools [50].

The study of genetic control is extremely important in the development of disease resistant cultivars, there are two forms of resistance that are related to inheritance. Vertical resistance is conferred by one or more genes (monogenic or oligogenic), with expression of genes of greater effect, presenting resistance to specific breeds and usually revealing little stability. The horizontal resistance is uniform, conditioned by several genes (polygenic) of small effect, nonspecific race, usually durable, there is no differential interaction between the pathogen races and the host cultivars [37].

Resistance to monogenic genetic control diseases facilitates the production of resistant cultivars mainly using the backcrossing method which is suitable for transferring one or a few genes. However, in many cases the resistance is polygenic and strongly influenced by environmental factors, making obtaining more laborious cultivars [51].

One of the steps to carry out the study of genetic control, consists in the use of homozygous parents or endogamous lines that present contrasting expressions in relation to what one wishes to study. These individuals provide the identification of the variability involved in the segregating generations evaluated. Several generations can be used for this purpose, with inheritance studies being more common with the parents and the F1 and F2 generations. To improve the understanding of phenotypic proportions, the use of backcrosses is indicated [52].

With the generations, an experiment should be carried out evaluating the character in which one wants to understand the inheritance. In the case of resistance to bacterial wither, it is necessary to evaluate the generations submitted to the *R. solanacearum* complex species, which can be infested soil [53], by artificial inoculation [31] or using the two previously cited methods together [54]. In possession of the data is carried out a study of the phenotypic proportions observed from the comparison with the expected phenotypic proportions, according to a segregation pattern. This pattern, according to [55] is tested as follows: first a hypothesis of monogenic inheritance is established, which if not appropriate, should be adjusted to digenic inheritance and so on up to the polygenic model.

One way to test the phenotypic proportions in segregating generations is by means of the non-parametric chi-square test ( $\chi^2$ ). In this test, based on the observed and expected frequencies, the calculated chi-squared value is obtained which is compared with the tabulated value. If a monogenic inheritance hypothesis is tested and the chi-square test is significant, the result indicates that it should be discarded, because the deviations of frequencies observed in relation to the expected frequencies were not due to chance [56, 55].

From the point of view of monogenic inheritance, through a cross in which individuals are contrasting, two phenotypic classes are observed if the interaction is of complete or lethal dominance; and three classes in the interaction with absence of dominance or co-dominance. Considering digenic inheritance, four classes are observed if the interaction is of complete dominance for the two genes with the classical phenotypic ratio of 9:3:3:1. In the

253 interaction of absence of dominance for the two genes in generation F2 we have nine  
254 genotypic classes in the proportion 1:2:1:2:4:2:1:2:1 [52]. It is important to emphasize that  
255 the number of classes increases with the increase in the number of genes, thus having a  
256 diverse phenotypic classification that is highly influenced by the environmental component  
257 [57]. The breeder must be very careful in selection when dealing with quantitative  
258 inheritance, because part of the manifested variability is due to the environment, and is not  
259 inheritable [58].

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261 Considering polygenic or quantitative inheritance, the genes that make up this genetic  
262 control are divided into two classes. The first is called major-effect or Mendelian genes, and  
263 the second of genes of smaller effects or modifiers, also denominated of polygenes [59].  
264 Higher-effect genes are responsible for significant phenotypic changes. The lower-effect  
265 genes have little influence on expression if considered individually, but when they are in  
266 large numbers they produce significant phenotypic changes [52].

267

268 It is important to test the model that explains the genetic control. First, the dominant additive  
269 model is tested, if it is not appropriate, the model is tested with epistasis. Considering a  
270 model without epistasis, the evaluation can be performed by the scale test (set), proposed  
271 by Cavalli in 1952 reported by [59], in which starting from the segregating generations it is  
272 recommended to estimate the mean components by the least squares method. To facilitate  
273 the resolution of the systems there are some recommended applications such as MAPGEM  
274 [60] and GENES [61].

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276 In an inheritance study it is important to perform the estimation of the components of mean,  
277 in which the parameters  $m$ ,  $a$  and  $d$ , which represent the average of the parents are  
278 obtained, the additive gene effects, and the non-additive gene effects (dominance),  
279 respectively. From these, one can obtain the average degree of dominance ( $GMD = [d] / [a]$ ),  
280 which helps in analyzing the predominant interaction between each pair of alleles, which  
281 ranges from absence of dominance (0), partial dominance (between 0 and 1), complete  
282 dominance (1) and overdominance (greater than 1) [52].

283

284 In relation to the bacterial wither of the tomato, there are several reports regarding the  
285 genetic control of resistance. This decreases the efficiency of breeding programs in the  
286 development of resistant cultivars and with acceptable agronomic attributes. The different  
287 results can be explained by different methodologies in conducting the genetic control study,  
288 sources of resistance, isolated from the different species of *R. solanacearum* complex,  
289 environments and finally the interaction between all these fundamental points [40, 62].

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291 The literature shows that the response of the different cultivars is more quantitative than  
292 qualitative [49]. there are many studies reporting from monogenic inheritance [63] to  
293 polygenic [64, 65]. Another great difference is observed in relation to the dominance and  
294 interaction between the genes [31, 53, 66]. The main results of some inheritance studies can  
295 be observed in table 1.

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305 Table 1. Relationship between researchers, sources of resistance and the main results  
 306 obtained in the genetic control of resistance to bacterial wither in tomato.  
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Researchers	Sources of resistance	Main results of genetic control
[67]	PI27080	Oligogenic with recessive action
[68]	Saturn e Vênus	Oligogenic with partial dominance
[69]	Vênus, VC-4 e H7741	Polygenic with additive effects
[70]	VC-48, VC-9, VC-11 e VC-8	Oligogenic or polygenic with partial dominance and epistasis
[71]	CRA-66 e IHR663123	Genes with recessive action and a dominant gene
[64]	Sem identificação	Polygenic with additive effects
[72]	Hawaii 7998	Monogenic dominant
[65]	Hawaii 7998	Polygenic
[73]	Hawaii 7997	Genes with recessive action
[74]	CL-32-d-01-19GS	Monogenic with partial dominance
[75]	Híbridos de Hawaii 7998	Partial dominance
[63]	Hawaii 7996	Monogenic dominant
[66]	D-9 e Hawaii 7998	Partially recessive with partial dominance towards susceptibility
[54]	Hawaii 7998, Caraíba e Yoshimatsu	Gene block with dominance and with additive effects
[31]	Hawaii 7998, Rotam-4 e Yoshimatsu	Oligogenic or polygenic with partial dominance and with additive effect
[53]	Drica	Oligogenic or polygenic with partial dominance
[76]	Hawaii 7998	Monogenic recessive
[77]	Hawaii 7998, BT-18 e TBL-4	More than one gene with additive effect and dominance

308 In the literature some studies are available with the genetic analysis of resistance using  
 309 molecular markers mainly in the cultivar Hawaii 7996. Depending on the isolate and the  
 310 evaluated cultivars, there are different QTLs [44, 78, 79]. In this way, it can be inferred that  
 311 the genetic control of resistance is quite variable.  
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313 In some studies it is reported inheritance of recessive resistance, having binding of these  
 314 resistance genes to small-sized fruits or what they crack [66, 67, 73] observed that the  
 315 association of resistance to bacterial wither and small fruit is not constant, having in their  
 316 works satisfactory results in the selection of progenies that combine favorable alleles for  
 317 these characteristics.  
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319 To increase efficiency in assessing potential of populations, based on the means and  
 320 variances it is possible to estimate the genetic parameters which are fundamental to  
 321 breeders in establishing effective selection strategies [80, 81].  
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323 In the F2:3 generation it is already possible to select resistant homozygous progenies which  
 324 may give rise to lines for future obtaining resistant cultivars besides identifying susceptible

325 and segregating progenies. With the evaluation of progenies F2:3 it is possible to carry out  
326 the confirmation of the inheritance study, especially in the quantification of possible larger  
327 genes [52, 82].

328  
329 Most of the genetic control studies of resistance to bacterial wither were carried out with  
330 foreign cultivars. Therefore it is necessary to carry out the study of genetic control using  
331 resistant national cultivars such as Gina, C-38-D, Compacto-6 and Yoshimatsu [83]. Among  
332 these, Yoshimatsu deserves special mention for its high resistance [9].

333  
334 According to [84], the change in the resistance pattern and the methodology used modifies  
335 the result of the inheritance study. In addition, it is believed that genetic controls for species  
336 alone may differ. Knowledge of inheritance can improve the efficiency of breeding programs,  
337 since individual isolates of these species vary with respect to epidemiology.

## 338 339 **5. CONCLUSION**

340  
341 It was found different information related to the genetic control of tomato resistance in  
342 relation to the number of genes and their interaction due to the high genetic diversity within  
343 the *Ralstonia Solanacearum* species complex.

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345 The high host x pathogen interaction reflects on different breeding strategies depending on  
346 the environment and the source of resistance used.

## 347 348 **COMPETING INTERESTS**

349  
350 Authors have declared that no competing interests exist.

## 351 352 353 **REFERENCES**

- 354  
355 1. Alvarenga MAR. Tomato: field production, greenhouse and hydroponics. 2<sup>a</sup> ed. Lavras:  
356 UFLA; 2013. English.
- 357  
358 2. Peralta IE, Spooner DM. History, origin and early cultivation of tomato (Solanaceae) In:  
359 Razdan MK, Mattoo AK. editors. Genetic improvement of solanaceous crops. 2<sup>a</sup> ed. Enfield:  
360 Science Publishers; 2007. English.
- 361  
362 3. Harvey M, Quilley S, Beynon H. Exploring the tomato: transformations of nature, society  
363 and economy. Cheltenham: Edward Elgar; 2002. English.
- 364  
365 4. Peralta IE, Knapp S, Spooner DM. Nomenclature for wild and cultivated tomatoes. Rep. of  
366 Tom. Gen. Coop. 2006;56:6-12. English.
- 367  
368 5. Carneiro MS, Vieira MLC. Genetic maps in plants. *Bragantia*, 2002;61(2): 89-100. English.
- 369  
370 6. Brickell CD, Baum BR, Hettterscheid WLA, Leslie AC, Mcneill J, Trehane P, et al.  
371 International code of nomenclature of cultivated plants. *Acta Hort.* 2004;647:1-123.  
372 English.
- 373  
374 7. Filgueira FAR. New Manual of Olericultura: modern agro-technology in the production and  
375 commercialization of vegetables. 3<sup>a</sup> ed. Viçosa: UFV; 2012. English.
- 376

- 377 8. Puiatti M, Balbino JMS, Fonseca MJO, Ronchi CP. Physiology of tomato development. In:  
378 INCAPER, editors. Tomato. Vitória: INCAPER; 2010. English.  
379
- 380 9. Nick C, Silva DJH. Tomato breeding. In: Nick C, Borém A, editors. Breeding vegetables.  
381 Viçosa: UFV; 2016. English.  
382
- 383 10. Silva JBC, Giordano LB. World and national production. In: Silva JBC, Giordano LB,  
384 editors. Tomato for industrial processing. Brasília: Embrapa Vegetables; 2000. English.  
385
- 386 11. Camargo FP, Alves HS, Camargo Filho WP, Vilela NJ. Production chain of industrial  
387 tomatoes in Brazil: review of 1990, regional production and prospects. Econ. Inf.  
388 2006;36(11):7-20. (2006). English.  
389
- 390 12. Botella-Paiva P, Rodriguez-Concepcion M. Carotenoid biotechnology in plants for  
391 nutritionally improved foods. Phys. Plant. 2006;126:369-381. English.  
392
- 393 13. Carmo CAS, Caliman LF. Climate, planting season and cultivating. In: INCAPER,  
394 editors. Tomato. Vitória: INCAPER; 2010. English.  
395
- 396 14. Bradford KJ, Chen F, Cooley MB, Dahal P, Downie B, Fukunaga KK, et al. Physiology of  
397 tomato development Yang H, Yim KO Gene expression prior to radicle emergence in  
398 imbibed tomato seeds. In: Black M, Bradford KJ, Vazquez-Ramos, editors. Seed Biology:  
399 Advances and Applications. New York: CAB International; 2000. English.  
400
- 401 15. Kinet JM, Peet MM. Tomato. In: Wien HC, editors. The physiology of vegetables crops.  
402 New York: CAB International; 1997. English.  
403
- 404 16. Maluf WR. Tomato genetic improvement tool. Lavras: UFLA; 2000. English.  
405
- 406 17. Smith EF. A bacterial disease of tomato, pepper, eggplant and Irish potato (*Bacillus*  
407 *solanacearum* nov. sp.). United States Department of Agriculture: Division of Vegetable  
408 Physiology and Pathology. 1896;12:1-28. English.  
409
- 410 18. Chester FD. Report of the mycologist: bacteriological work. Del. Agric. Exp. Stn. Bull.  
411 1898;10:47-137. English.  
412
- 413 19. Smith EF. Bacteria in relation to plant disease. Washington: Carnegie Institution; 1914.  
414 English.  
415
- 416 20. Bergey DH. Manual of systematic bacteriology: the Proteobacteria. 1<sup>a</sup> ed. New York:  
417 Springer-Verlag; 1923. English.  
418
- 419 21. Yabuuchi E, Kosaro Y, Oyizu H, Yano I, Hotta H, Hashimoto Y, et al. Proposal of  
420 *Burkholderia* gen. nov. and transfer of seven species of the genus *Pseudomonas* homology  
421 group II to the new genus, with the type species *Burkholderia cepacia* (Palleroni and  
422 Holmes, 1981) comb. nov. Microb. and Imm. 1992;36(12):1251-1275. English.  
423
- 424 22. Yabuuchi E, Kosako Y, Oyaizu H, Yano I, Hotta H, Hashimoto Y, et al. Transfer of two  
425 *Burkholderia* and an *Alcaligenes* species to *R.* gen. nov. – Proposal of *R. pickettii* (Ralston,  
426 Palleroni and Doudoroff, 1973) com. nov., *R. solanacearum* (Smith, 1896) comb. nov. and *R.*  
427 *eutropha* (Davis, 1969) comb. nov. Microb. and Imm. 1995;39(11):897-904. English.  
428

- 429 23. Fegan M, Prior P. How complex is the *Ralstonia solanacearum* species complex. In:  
430 Allen C, Prior C, Hayward AC, editors. Bacterial wilt disease and the *Ralstonia*  
431 *solanacearum* species complex. 2<sup>a</sup> ed. Saint Paul: APS Press; 2005. English.  
432
- 433 24. Silva JR. Diversity of isolates of *R. solanacearum* from the North and Northeast regions  
434 of Brazil. Recife, Rural Federal University of Pernambuco; 2014. English.  
435
- 436 25. Wicker E, Lefeuvre P, Cambiaire JC, Poussier S, Prior P. Contrasting recombination  
437 patterns and demographic histories of the plant pathogen *R. solanacearum* inferred from  
438 MLSA. Inter. Soc. for Microb. Ecol. Jour. 2012;6(5):961-974. English.  
439
- 440 26. Safni I, Cleenwerck I, De-Vos P, Fegan M, Sly L, Kappler U. Polyphasic taxonomic  
441 revision of the *R. solanacearum* species complex: proposal to emend the descriptions of *R.*  
442 *solanacearum* and *R. syzygii* and reclassify current *R. syzygii* strains as *R. syzygii* subsp.  
443 *syzygii*, *R. solanacearum* phylotype IV strains as *R. syzygii* subsp. *indonesiensis* subsp.  
444 nov., banana blood disease bacterium strains as *R. syzygii* subsp. *celebesensis* subsp. nov.  
445 and *R. solanacearum* phylotypes I and III strains as *R. pseudosolanacearum* sp. nov. Inter.  
446 Jour. of Syst. and Evol. Microb. 2014;64(9):3087-103. English.  
447
- 448 27. Agrios GN. Plant pathology. 5<sup>a</sup> ed. San Diego: Elsevier; 2005. English.  
449
- 450 28. Liu HL, Zhang SP, Schell MA, Denny TP. Pyramiding, unmarked deletions in *R.*  
451 *solanacearum* shows that secreted proteins in addition to plant cell-wall degrading enzymes  
452 contribute to virulence. Mol. Plant-Microb. Inter. 2005;18(12):1296-1305. English.  
453
- 454 29. Hikichi Y, Yoshimochi T, Tsujimoto S, Shinohara R, Nakaho K, Kanda A, et al. Global  
455 regulation of pathogenicity mechanism of *R. solanacearum*. Plant Biot. 2007;24(1):149-154.  
456 English.  
457
- 458 30. Amorim L, Rezende MAJ, Bergamin Filho A. Manual of phytopathology: principles and  
459 concepts. 1<sup>a</sup> ed. Agronomic Ceres: Ouro Fino; 2011. English.  
460
- 461 31. Oliveira WF, Giordano LB, Lopes CA. Inheritance of resistance in tomato to withered  
462 bacterial. Fitop. Bras. 1999;24:49-53. English.  
463
- 464 32. Lopes CA, Quezado-Soares AM. Diseases caused by bacteria in tomato. In: Zambolim  
465 L, Vale FXR, Costa H, editors. Control of plant diseases: vegetables. Viçosa: UFV; 2000.  
466 English.  
467
- 468 33. Lopes CA, Mendonça JL. Enxertia in tomato for control of bacterial withered. Brasília:  
469 EMBRAPA; 2014. English.  
470
- 471 34. Santiago TR, Lopes CA, Caetano-Anolles G, Mizubuti ESG. Phylotype and sequevar  
472 variability of *R. solanacearum* in Brazil, an ancient centre of diversity of the pathogen. Plant  
473 Pathol. 2016;66:383-392. English.  
474
- 475 35. Mariano RLR, Melo RAG, Holanda VT, Cabral GB, Silva MSSG. Survey of the  
476 phytobacterioses of the state of Pernambuco in the 1987-1988 biennium. Braz. Phyto.  
477 1989;14(2):158-169. English.  
478
- 479 36. Filgueira FAR. Solanaceae: modern agro-technology in tomato, potato, pepper, eggplant  
480 and jiló production. Lavras: UFLA; 2003. English.  
481

- 482 37. Lopes CA, Boiteux LS. Breeding for resistance to bacterial diseases. In: Fritse-Neto R,  
483 Borém A, editors. Plant breeding for biotic stress conditions. Viçosa: UFV; 2012. English.  
484
- 485 38. Rodrigues LMR, Destefano SAL, Silva MJ, Costa GGL, Maringoni AC. Characterization  
486 of *R. solanacearum* from Brazil using molecular methods and pathogenicity tests. Jour. of  
487 Plant Pathol. 2012;94(3):505-516. English.  
488
- 489 39. Albuquerque GMR. Resistance to bacterial wither in tomato: diversity of *Ralstonia* spp. in  
490 Pernambuco, selection of wild accesses and genetic characterization of resistance. Recife,  
491 Rural Federal University of Pernambuco; 2017. English.  
492
- 493 40. Huet G. Breeding for resistances to *R. solanacearum*. Mini review article. In: Allen C,  
494 Prior P, Hayward AC, editors. Bacterial wilt disease and the *R. solanacearum* species  
495 complex. 2<sup>a</sup> ed. Saint Paul: APS Press; 2014. English.  
496
- 497 41. Egashira H, Kuwashima A, Imanishi S, Ishiguro H, Fukushima K, Kaya T. Screening of  
498 wild accessions resistant to gray mold (*Botrytis cinerea* Pers.) in *Lycopersicon*. Acta Phys.  
499 Plant. 2000;22:324-326. English.  
500
- 501 42. Pico B, Sifres A, Elia M, Diez MJ, Nuez F. Searching for new resistance sources to  
502 tomato yellow leaf curl virus within a highly variable wild *Lycopersicon* genetic pool. Acta  
503 Phys. Plant. 2000;22:344-350. English.  
504
- 505 43. Scott JW, Wang JF, Hanson P. Breeding tomatoes for resistance to bacterial wilt, a  
506 global view. Acta Hortic. 2005;695:161-168. English.  
507
- 508 44. Thoquet PJ, Olivier C, Sperisen P, Rogowsky H, Laterrot H, Grimsley N. Quantitative  
509 trait loci determining resistance to bacterial wilt in tomato cultivar Hawaii7996. Mol. Plant-  
510 Mic. Int. 1996;9(9):826-836. English.  
511
- 512 45. Yuliar YAN, Toyota K. Recent trends in control methods for bacterial wilt diseases  
513 caused by *R. solanacearum*. Microb. Envir. 2015;30:1-11. English.  
514
- 515 46. Hanson PM, Wang JF, Licardo O, Hanudin SYM, Hartman GL, Lin YC. Variable reaction  
516 of tomato lines to bacterial wilt evaluated at several locations in Southeast Asia. Hortsc.  
517 1996;31:143-146. English.  
518
- 519 47. Lopez MM, Biosca EG. Potato bacterial wilt management: new prospects for an old  
520 problem. In: Allen C, Prior P, Hayward AC, editors. Bacterial Wilt Disease and the *R.*  
521 *solanacearum* Species Complex. Saint Paul: APS Press; 2005. English.  
522
- 523 48. Wang JF, Hanson P, Barnes JA. Worldwide evaluation of an international set of  
524 resistance sources to bacterial wilt in tomato. In: Prior P, Allen C, Elphinstone J, editors.  
525 Bacterial Wilt Disease. Molecular and Ecological Aspects. Berlin: Springer-Verlag; 1998.  
526 English.  
527
- 528 49. Prior P, Steva H, Cadet P. Aggressiveness of strains of *Pseudomonas solanacearum*  
529 from the French West Indies (Martinique and Guadeloupe) on tomato. Plant Dis.  
530 1990;74:962-965. English.  
531
- 532 50. Camargo LEA. Genetic analysis of resistance and pathogenicity. In: Bergamin Filho A,  
533 Kimati H, Amorim L, editors. Manual of phytopathology: Principles and concepts. São Paulo:  
534 Agrônômica Ceres; 1995. English.

- 535  
536 51. Borém A, Miranda GV. Plant breeding. 6<sup>a</sup> ed. Viçosa: UFV; 2013. English.  
537
- 538 52. Ramalho APR, Abreu AFB, Santos JB, Nunes JAR. Applications of quantitative genetics  
539 in the improvement of autogamous plants. Lavras: UFLA; 2012. English.  
540
- 541 53. Lima Neto AFL, Silveira MA, Souza RM, Nogueira SR, André CMG. Inheritance of  
542 bacterial wilt resistance in tomato plants cropped in naturally infested soils of the state of  
543 Tocantins. Crop Breed. and Appl. Biot. 2002;2(1): 2002. English.  
544
- 545 54. Menezes D. Genetic Analysis of a Diallelic Crossing in Tomatoes (*Lycopersicon*  
546 *esculentum* Mill). Recife, Rural Federal University of Pernambuco; 1998. English.  
547
- 548 55. Viana JMS, Cruz CD, Barros EG. Genetics: Fundamentals. Viçosa: UFV; 2012. English.  
549
- 550 56. Siegel S, Castellan Júnior NJ. Nonparametric Statistics for Behavioral Sciences. São  
551 Paulo: Artmed-Bookman; 2008. English.  
552
- 553 57. Allard RW. Principles of plant breeding. 2<sup>a</sup> ed. New York: John Willey e Sons; 1999.  
554 English.  
555
- 556 58. Falconer DS. Introduction to quantitative genetics. Viçosa: UFV; 1987. English.  
557
- 558 59. Mather K, Jinks JL. Biometrical genetics. 3<sup>a</sup> ed. Cambridge: University Press; 1982.  
559 English.  
560
- 561 60. Ferreira DF, Zambalde AL. Simplification of the analysis of some special techniques of  
562 agricultural experimentation in Mapgen and related software. In: Congress of the Brazilian  
563 society of informatics applied to agriculture and agroindustry. Belo Horizonte: Annals; 1997.  
564 English.  
565
- 566 61. Cruz CD. GENES - A software package for analysis in experimental statistics and  
567 quantitative genetics. Acta Sci. 2013; 35(3): 271-276. English.  
568
- 569 62. Persley GJ, Batugal P, Gaparin D, Vander PZ. Summary of discussion and  
570 recommendations. Bacterial Wilt Disease in Asia and the South Pacific. ACIAR. 1985;13:7-  
571 13. English.  
572
- 573 63. Grimault V, Prior P, Anais GA. A monogenic dominant resistance of tomato to bacterial  
574 wilt in Hawaii 7998 is associated with plant colonization by *Pseudomonas solanacearum*.  
575 Jour. of Phyt. 1995;143:349-352. English.  
576
- 577 64. Ferrer ZA. The nature of resistance in a tomato tolerant to *Pseudomonas solanacearum*.  
578 Phytopathology. 1984;74:1014. English.  
579
- 580 65. Hayward AC. Biology and Epidemiology of Bacterial Wilt Caused by *Pseudomonas*  
581 *solanacearum*. Ann. Rev. of Phyt. 1991;29:65-87. English.  
582
- 583 66. Monma S, Sakata Y, Matsunaga H. Inheritance and selection efficiency of bacterial wilt  
584 resistance in tomato. JARQ. 1997;31:195-204. English.  
585
- 586 67. Acosta JC, Gilbert JC, Quinon VL. Heritability of bacterial wilt resistance in tomato. Proc.  
587 of Amer. Soc. for Hort. Sc. 1964;84:455-462. English.

- 588  
589 68. Digat B, Derieux MA. Study of the varietal resistance of tomato to bacterial wilt II. The  
590 practical value of F1 hybrids and their contribution to the genetic basis of resistance. In:  
591 Proceedings of the annual meeting Caribbean food crops society. Mayaguez: Augustine;  
592 1968. English.  
593
- 594 69. Graham KM, Yap TC. Studies on bacterial wilt. I. Inheritance of resistance to  
595 *Pseudomonas solanacearum* in Tomato. Mal. Agr. Res. 1976;5:1-8. English.  
596
- 597 70. Mew TW, Ho WC. Varietal resistance to bacterial wilt in tomato. Plant Dis. 1976;60:264-  
598 268. English.  
599
- 600 71. Tikoo SK, Anand N, Ramkrishna. Presence of two independent genetic systems for  
601 resistance to bacterial wilt (*Pseudomonas solanacearum*) in tomato. Int. gen. cong.  
602 1983;15:12-23. English.  
603
- 604 72. Scott JW, Somodi GC, Jones JB. Bacterial spot resistance is not associated to bacterial  
605 wilt resistance in tomato. Proc. of the Flor. St. Hort. Soc. 1988;101:390-392. English.  
606
- 607 73. Somodi GC, Jones JB, Scott JW. Comparison of inoculation techniques for screening  
608 tomato genotypes for bacterial wilt resistance. Bacterial Wilt. ACIAR. 1992;45:120-123.  
609 English.  
610
- 611 74. Peter KV, Gopalakrishnam TR, Rajan S, Kumar PGS. Breeding for resistance to  
612 bacterial wilt in tomato, eggplant and pepper. Bacterial Wilt. ACIAR. 1992;45:183-190.  
613 English.  
614
- 615 75. Scott JW, Somodi GC, Jones JB. Testing tomato genotypes and breeding for resistance  
616 to bacterial wilt in Florida. In: Hartman GL, Hayward AC, editors. Bacterial wilt. Canberra:  
617 ACIAR; 1993. English.  
618
- 619 76. Thakur AK, Kohli UK, Kumar M. Inheritance of resistance to bacterial wilt in tomato  
620 (*Lycopersicon esculentum* Mill.). Ind. Jour. of Gen. and Plant Breed. 2004;64(1):79-80.  
621 English.  
622
- 623 77. Sharma KC, Sharma LK. Genetic studies of bacterial wilt resistance in tomato crosses  
624 under mid-hill conditions of Himachal Pradesh. Jour. of Hil. Agr. 2015;6(1):136-137. English.  
625
- 626 78. Danesh D, Aarons S, McGill GE, Young ND. Genetic dissection of oligogenic resistance  
627 to bacterial wilt in tomato. Mol. Plant-Mic. Int. 1994;7:464-471. English.  
628
- 629 79. Mangin B, Thoquet P, Olivier J, Grimsley NH. Temporal and multiple quantitative trait loci  
630 analyses of resistance to bacterial wilt in tomato permit the resolution of linked loci.  
631 Genetics. 1999;151:1165-1172. English.  
632
- 633 80. Cruz CD. Principles of quantitative genetics. Viçosa: UFV; 2012. English.  
634
- 635 81. Cruz CD, Carneiro PCS, Regazzi AJ. Biometric models applied to genetic breeding. 3<sup>a</sup>  
636 ed. Viçosa: UFV; 2014. English.  
637
- 638 82. Fiorini CVA, Gomes LAA, Libânio RA, Maluf WR, Campos VP, Licursi V, et al.  
639 Identification of progenies F2:3 of homozygous lettuce resistant to gnats nematodes. Hort.  
640 Bras. 2007;25:509-513. English.

641

642 83. Makishima N, Miranda JEC. Cultivation of Tomato (*Lycopersicon esculentum* Mill.).  
643 Brasília: EMBRAPA Vegetable; 1992. English.

644

645 84. Monma S, Sakata Y. Inheritance of resistance to bacterial wilt in tomato. Bacterial Wilt.  
646 ACIAR. 1992;45:149-153. English.

647