Review Paper

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].

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

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

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

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

2. BACTERIAL WITHER IN TOMATO

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

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

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

From the phylogenetic analysis of the partial sequence of the endoglucanase gene and the ITS region, DNA-DNA hybridization, biochemical, cultural and physiological characteristics [26] proposed the taxonomic reclassification of the *R. solanacearum* complex in three independent species and subspecies. *Ralstonia pseudosolanacearum* consists of isolates belonging to phylotypes I and III, originating in Asia and Africa, respectively. *R. solanacearum* by phyllotype II isolates (IIA and IIB), originated in the American continent and that probably possess two subspecies. The isolates of philotype IV originated from Indonesia were reclassified into three subspecies of *R. syzigii*, where *R. syzigii* subsp. *indonesiensis* grouped the wilt-causing isolates of Ralstonia in Solanaceas, *R. syzigii* subsp. *syzigii* the isolates previously denominated of *R. syzigii* and as *R. syzigii* subsp. *celebesensis* of blood disease bacterium [26].

The species of the R. solanacearum complex are gram negative, their format is straight rods or slightly curved, with approximately 0.5-1.0 x 1.5-4.0 µm. Are non-sporogenic, mobile through one or more polar flagella and aerobic. Its growth occurs in temperature between 25 and 35 °C [27]. These bacteria inhabit the soil and invade the root system by means of wounds, multiplies rapidly within the xylem and hereby is distributed throughout the plant. The result of colonization is the obstruction of the vessels by the accumulation of exopolysaccharides, blocking the translocation of water and nutrients. The main symptoms are darkening of the xylem vessels and sudden wither with no change in green coloration. The darkening of the vessels is due to the transport of substances resulting from the oxidation of phenols, resulting in melanin. It is worth mentioning that depending on the combination of several factors the disease can appear in any stage of development of the tomato [28, 29, 30].

As for most phytobacteria, controlling bacterial wither is very difficult. Therefore, it is recommended to make the integrated management, since the use of isolated measures is not efficient to avoid losses. Among the isolated measures, chemical control has low efficiency and is extremely damaging to the environment [31]. Some recommended control measures are: soil water management in order to avoid waterlogging; to avoid injuries caused by nematodes, insects or agricultural implements; avoid moving soil from disease outbreaks to other areas; elimination of diseased, infected and invasive volunteers from the Solanaceae family; perform crop rotation for at least one year with grasses; grafting on resistant grafts and the use of resistant cultivars [32, 33].

In Brazil and in the State of Pernambuco, the species *R. pseudosolanacearum* and *R. solanacearum* [24, 34] have been reported so far. It is believed that R. solanacearum has Brazil as the center of origin and diversity, while *R. pseudosolanacearum* was introduced from Asia. The disease is present in all mesoregions of the State of Pernambuco, causing great damage to the tomato crop of the State [35]. Thus, it is clear the importance of the breeding of plants aiming the resistance to bacterial wither in an attempt to mitigate the damages caused by this disease in the tomato crop.

3. PLANT BREEDING FOR RESISTANCE TO BACTERIAL WITHER

The use of resistant cultivars is the most efficient way to control bacterial wither in tomato plants per it presents low cost, low impact on the environment and easy adoption by the producer [36, 37].

To become the plant breeding aiming the efficiency of bacterial wither resistance, it is necessary to emphasize that in Brazil the *R. solanacearum* complex presents a great genetic diversity. This is composed by 13 sequevares of Solanaceae (I-17, I-18, IIA-41, IIA-

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

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

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

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

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

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

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

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

4. STUDY OF GENETIC CONTROL OF RESISTANCE TO BACTERIAL WITHER

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 (χ_c^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

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

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

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

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

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

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

Table 1. Relationship between researchers, sources of resistance and the main results obtained in the genetic control of resistance to bacterial wither in tomato.

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

In the literature some studies are available with the genetic analysis of resistance using molecular markers mainly in the cultivar Hawaii 7996. Depending on the isolate and the evaluated cultivars, there are different QTLs [44, 78, 79]. In this way, it can be inferred that the genetic control of resistance is quite variable.

In some studies it is reported inheritance of recessive resistance, having binding of these resistance genes to small-sized fruits or what they crack [66, 67, 73] observed that the association of resistance to bacterial wither and small fruit is not constant, having in their works satisfactory results in the selection of progenies that combine favorable alleles for these characteristics.

To increase efficiency in assessing potential of populations, based on the means and variances it is possible to estimate the genetic parameters which are fundamental to breeders in establishing effective selection strategies [80, 81].

In the F2:3 generation it is already possible to select resistant homozygous progenies which may give rise to lines for future obtaining resistant cultivars besides identifying susceptible

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

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

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

5. CONCLUSION

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 x pathogen interaction reflects on different breeding strategies depending on the environment and the source of resistance used.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Alvarenga MAR. Tomato: field production, greenhouse and hydroponics. 2ª ed. Lavras: UFLA; 2013. English.

2. Peralta IE, Spooner DM. History, origin and early cultivation of tomato (Solanaceae) In: Razdan MK, Mattoo AK. editors. Genetic improvement of solanaceous crops. 2ª ed. Enfield: Science Publishers; 2007. English.

3. Harvey M, Quilley S, Beynon H. Exploring the tomato: transformations of nature, society and economy. Cheltenham: Edward Elgar; 2002. English.

4. Peralta IE, Knapp S, Spooner DM. Nomenclature for wild and cultivated tomatoes. Rep. of Tom. Gen. Coop. 2006;56:6-12. English.

5. Carneiro MS, Vieira MLC. Genetic maps in plants. Bragantia, 2002;61(2): 89-100. English.

6. Brickell CD, Baum BR, Hetterscheid WLA, Leslie AC, Mcneill J, Trehane P, et al. International code of nomenclature of cultivated plants. Acta Hortic. 2004;647:1-123. English.

7. Filgueira FAR. New Manual of Olericultura: modern agro-technology in the production and commercialization of vegetables. 3ª ed. Viçosa: UFV; 2012. English.

8. Puiatti M, Balbino JMS, Fonseca MJO, Ronchi CP. Physiology of tomato development. In: INCAPER, editors. Tomato. Vitória: INCAPER; 2010. English.

9. Nick C, Silva DJH. Tomato breeding. In: Nick C, Borém A, editors. Breeding vegetables. Viçosa: UFV; 2016. English.

10. Silva JBC, Giordano LB. World and national production. In: Silva JBC, Giordano LB, editors. Tomato for industrial processing. Brasilia: Embrapa Vegetables; 2000. English.

11. Camargo FP, Alves HS, Camargo Filho WP, Vilela NJ. Production chain of industrial tomatoes in Brazil: review of 1990, regional production and prospects. Econ. Inf. 2006;36(11):7-20. (2006). English.

12. Botella-Paíva P, Rodriguez-Concepcion M. Carotenoid biotechnologyin plants for butritionally improved foods. Phys. Plant. 2006;126:369-381. English.

13. Carmo CAS, Caliman LF. Climate, planting season and cultivating. In: INCAPER, editors. Tomato. Vitória: INCAPER; 2010. English.

14. Bradford KJ, Chen F, Cooley MB, Dahal P, Downie B, Fukunaga KK, et al. Physiology of tomato development Yang H, Yim KO Gene expression prior to radicle emergence in imbibed tomato seeds. In: Black M, Bradford KJ, Vazquez-Ramos, editors. Seed Biology: Advances and Applications. New York: CAB International; 2000. English.

15. Kinet JM, Peet MM. Tomato. In: Wien HC, editors. The physiology of vegetables crops. New York: CAB International; 1997. English.

16. Maluf WR. Tomato genetic improvement tool. Lavras: UFLA; 2000. English.

17. Smith EF. A bacterial disease of tomato, pepper, eggplant and Irish potato (*Bacillus solanacearum* nov. sp.). United States Department of Agriculture: Division of Vegetable Physiology and Pathology. 1896;12:1-28. English.

18. Chester FD. Report of the mycologist: bacteriological work. Del. Agric. Exp. Stn. Bull. 1898;10:47-137. English.

19. Smith EF. Bacteria in relation to plant disease. Washington: Carnegie Institution; 1914. English.

20. Bergey DH. Manual of systematic bacteriology: the Proteobacteria. 1ª ed. New York: Springer-Verlag; 1923. English.

21. Yabuuchi E, Kosaro Y, Oyizu H, Yano I, Hotta H, Hashimoto Y, et al. Proposal of *Burkholderia* gen. nov. and transfer of seven species of the genus *Pseudomonas* homology group II to the new genus, with the type species *Burkholderia cepacia* (Palleroni and Holmes, 1981) comb. nov. Microb. and Imm. 1992;36(12):1251-1275. English.

22. Yabuuchi E, Kosako Y, Oyaizu H, Yano I, Hotta H, Hashimoto Y, et al. Transfer of two *Burkholderia* and an *Alcaligenes* species to *R.* gen. nov. – Proposal of *R. pickettii* (Ralston, Palleroni and Doudoroff, 1973) com. nov., *R. solanacearum* (Smith, 1896) comb. nov. and *R. eutropha* (Davis, 1969) comb. nov. Microb. and Imm. 1995;39(11):897-904. English.

23. Fegan M, Prior P. How complex is the *Ralstonia solanacearum* species complex. In: Allen C, Prior C, Hayward AC, editors. Bacterial wilt disease and the *Ralstonia solanacearum* species complex. 2ª ed. Saint Paul: APS Press; 2005. English.

24. Silva JR. Diversity of isolates of R. solanacearum from the North and Northeast regions of Brazil. Recife, Rural Federal University of Pernambuco; 2014. English.

25. Wicker E, Lefeuvre P, Cambiaire JC, Poussier S, Prior P. Contrasting recombination patterns and demographic histories of the plant pathogen *R. solanacearum* inferred from MLSA. Inter. Soc. for Microb. Ecol. Jour. 2012;6(5):961-974. English.

26. Safni I, Cleenwerck I, De-Vos P, Fegan M, Sly L, Kappler U. Polyphasic taxonomic revision of the *R. solanacearum* species complex: proposal to emend the descriptions of *R. solanacearum* and *R. syzygii* and reclassify current *R. syzygii* strains as *R. syzygii* subsp. *syzygii*, *R. solanacearum* phylotype IV strains as *R. syzygii* subsp. *indonesiensis* subsp. nov., banana blood disease bacterium strains as *R. syzygii* subsp. *celebesensis* subsp. nov. and *R. solanacearum* phylotypes I and III strains as *R. pseudosolanacearum* sp. nov. Inter. Jour. of Syst. and Evol. Microb. 2014;64(9):3087-103. English.

27. Agrios GN. Plant pathology. 5ª ed. San Diego: Elsevier; 2005. English.

28. Liu HL, Zhang SP, Schell MA, Denny TP. Pyramiding, unmarked deletions in *R. solanacearum* shows that secreted proteins in addition to plant cell-wall degrading enzymes contribute to virulence. Mol. Plant-Microb. Inter. 2005;18(12):1296-1305. English.

29. Hikichi Y, Yoshimochi T, Tsujimoto S, Shinohara R, Nakaho K, Kanda A, et al. Global regulation of pathogenicity mechanism of *R. solanacearum*. Plant Biot. 2007;24(1):149-154. English.

30. Amorim L, Rezende MAJ, Bergamin Filho A. Manual of phytopathology: principles and concepts. 1ª ed. Agronomic Ceres: Ouro Fino; 2011. English.

31. Oliveira WF, Giordano LB, Lopes CA. Inheritance of resistance in tomato to withered bacterial. Fitop. Bras. 1999;24:49-53. English.

32. Lopes CA, Quezado-Soares AM. Diseases caused by bacteria in tomato. In: Zambolim L, Vale FXR, Costa H, editors. Control of plant diseases: vegetables. Viçosa: UFV; 2000. English.

33. Lopes CA, Mendonça JL. Enxertia in tomato for control of bacterial withered. Brasília: EMBRAPA; 2014. English.

34. Santiago TR, Lopes CA, Caetano-Anolles G, Mizubuti ESG. Phylotype and sequevar variability of *R. solanacearum* in Brazil, an ancient centre of diversity of the pathogen. Plant Pathol. 2016;66:383-392. English.

35. Mariano RLR, Melo RAG, Holanda VT, Cabral GB, Silva MSSG. Survey of the phytobacterioses of the state of Pernambuco in the 1987-1988 biennium. Braz. Phyto. 1989;14(2):158-169. English.

36. Filgueira FAR. Solanaceae: modern agro-technology in tomato, potato, pepper, eggplant and jiló production. Lavras: UFLA; 2003. English.

37. Lopes CA, Boiteux LS. Breeding for resistance to bacterial diseases. In: Fritse-Neto R, Borém A, editors. Plant breeding for biotic stress conditions. Viçosa: UFV; 2012. English.

38. Rodrigues LMR, Destefano SAL, Silva MJ, Costa GGL, Maringoni AC. Characterization of *R. solanacearum* from Brazil using molecular methods and pathogenicity tests. Jour. of Plant Pathol. 2012;94(3):505-516. English.

39. Albuquerque GMR. Resistance to bacterial wither in tomato: diversity of Ralstonia spp. in Pernambuco, selection of wild accesses and genetic characterization of resistance. Recife, Rural Federal University of Pernambuco; 2017. English.

40. Huet G. Breeding for resistances to *R. solanacearum*. Mini review article. In: Allen C, Prior P, Hayward AC, editors. Bacterial wilt disease and the *R. solanacearum* species complex. 2ª ed. Saint Paul: APS Press; 2014. English.

41. Egashira H, Kuwashima A, Imanishi S, Ishiguro H, Fukushima K, Kaya T. Screening of wild accessions resistant to gray mold (*Botrytis cinerea* Pers.) in Lycopersicon. Acta Phys. Plant. 2000;22:324-326. English.

42. Pico B, Sifres A, Elia M, Diez MJ, Nuez F. Searching for new resistance sources to tomato yellow leaf curl virus within a highly variable wild Lycopersicon genetic pool. Acta Phys. Plant. 2000;22:344-350. English.

43. Scott JW, Wang JF, Hanson P. Breeding tomatoes for resistance to bacterial wilt, a global view. Acta Hortic. 2005;695:161-168. English.

44. Thoquet PJ, Olivier C, Sperisen P, Rogowsky H, Laterrot H, Grimsley N. Quantitative trait loci determining resistance to bacterial wilt in tomato cultivar Hawaii7996. Mol. Plant-Mic. Int. 1996;9(9):826-836. English.

45. Yuliar YAN, Toyota K. Recent trends in control methods for bacterial wilt diseases caused by *R. solanacearum*. Microb. Envir. 2015;30:1-11. English.

46. Hanson PM, Wang JF, Licardo O, Hanudin SYM, Hartman GL, Lin YC. Variable reaction of tomato lines to bacterial wilt evaluated at several locations in Southeast Asia. Hortsc. 1996;31:143-146. English.

47. Lopez MM, Biosca EG. Potato bacterial wilt management: new prospects for an old problem. In: Allen C, Prior P, Hayward AC, editors. Bacterial Wilt Disease and the *R. solanacearum* Species Complex. Saint Paul: APS Press; 2005. English.

48. Wang JF, Hanson P, Barnes JA. Worldwide evaluation of an international set of resistance sources to bacterial wilt in tomato. In: Prior P, Allen C, Elphinstone J, editors. Bacterial Wilt Disease. Molecular and Ecological Aspects. Berlin: Springer-Verlag; 1998. English.

49. Prior P, Steva H, Cadet P. Aggressiveness of strains of *Pseudomonas solanacearum* from the French West Indies (Martinique and Guadeloupe) on tomato. Plant Dis. 1990;74:962-965. English.

50. Camargo LEA. Genetic analysis of resistance and pathogenicity. In: Bergamin Filho A, Kimati H, Amorim L, editors. Manual of phytopathology: Principles and concepts. São Paulo: Agronômica Ceres; 1995. English.

51. Borém A, Miranda GV. Plant breeding. 6ª ed. Viçosa: UFV; 2013. English.

52. Ramalho APR, Abreu AFB, Santos JB, Nunes JAR. Applications of quantitative genetics in the improvement of autogamous plants. Lavras: UFLA; 2012. English.

53. Lima Neto AFL, Silveira MA, Souza RM, Nogueira SR, André CMG. Inheritance of bacterial wilt resistance in tomato plants cropped in naturally infested soils of the state of Tocantins. Crop Breed. and Appl. Biot. 2002;2(1): 2002. English.

54. Menezes D. Genetic Analysis of a Dialelic Crossing in Tomatoes (*Lycopersicon esculentum* Mill). Recife, Rural Federal University of Pernambuco; 1998. English.

55. Viana JMS, Cruz CD, Barros EG. Genetics: Fundamentals. Viçosa: UFV; 2012. English.

56. Siegel S, Castellan Júnior NJ. Nonparametric Statistics for Behavioral Sciences. São Paulo: Artmed-Bookman; 2008. English.

57. Allard RW. Principles of plant breeding. 2ª ed. New York: John Willey e Sons; 1999. English.

58. Falconer DS. Introduction to quantitative genetics. Viçosa: UFV; 1987. English.

59. Mather K, Jinks JL. Biometrical genetics. 3ª ed. Cambridge: University Press; 1982. English.

60. Ferreira DF, Zambalde AL. Simplification of the analysis of some special techniques of agricultural experimentation in Mapgen and related software. In: Congress of the Brazilian society of informatics applied to agriculture and agroindustry. Belo Horizonte: Annals; 1997. English.

61. Cruz CD. GENES - A software package for analysis in experimental statistics and quantitative genetics. Acta Sci. 2013; 35(3): 271-276. English.

62. Persley GJ, Batugal P, Gaparin D, Vander PZ. Sumary odf discussion and recommendations. Bacterial Wilt Disease in Asia and the South Pacifc. ACIAR. 1985;13:7-13. English.

63. Grimault V, Prior P, Anais GA. A monogenic dominant resistance of tomato to bacterial wilt in Hawaii 7998 is associated with plant colonization by *Pseudomonas solanacearum*. Jour. of Phyt. 1995;143:349-352. English.

64. Ferrer ZA. The nature of resistance in a tomato tolerant to *Pseudomonas solanacearum.* Phytopathology. 1984;74:1014. English.

65. Hayward AC. Biology and Epidemiology of Bacterial Wilt Caused by Pseudomonas solanacearum. Ann. Rev. of Phyt. 1991;29:65-87. English.

66. Monma S, Sakata Y, Matsunaga H. Inheritance and selection efficiency of bacterial wilt resistance in tomato. JARQ. 1997;31:195-204. English.

67. Acosta JC, Gilbert JC, Quinon VL. Heritability of bacterial wilt resistance in tomato. Proc. of Amer. Soc. for Hort. Sc. 1964;84:455-462. English.

68. Digat B, Derieux MA. Study of the varietal resistance of tomato to bacterial wilt II. The practical value of F1 hybrids ans their contribuition to the genetic basis of resistance. In: Proceedings of the annual meeting caribbean food crops society. Mayaguez: Augustine; 1968. English.

69. Graham KM, Yap TC. Studies on bacterial wilt. I. Inheritance of resistance to Pseudomonas solanacearum in Tomato. Mal. Agr. Res. 1976;5:1-8. English.

70. Mew TW, Ho WC. Varietal resistance to bacterial wilt in tomato. Plant Dis. 1976;60:264-268. English.

71. Tikoo SK, Anand N, Ramkrishna. Presence of two independet genetic systems for resistance to bacterial wilt (*Pseudomonas solanacearum*) in tomato. Int. gen. cong. 1983;15:12-23. English.

72. Scott JW, Somodi GC, Jones JB. Bacterial spot resistance is not associated to bacterial wit resistance in tomato. Proc. of the Flor. St. Hort. Soc. 1988;101:390-392. English.

73. Somodi GC, Jones JB, Scoot JW. Comparison of inoculation techniques for screening tomato genotypes for bacterial wilt resistance. Bacterial Wilt. ACIAR. 1992;45:120-123. English.

74. Peter KV, Gopalakrishnam TR, Rajan S, Kumar PGS. Breeding for resistance to bacterial wilt in tomato, eggplant and pepper. Bacterial Wilt. ACIAR. 1992;45:183-190. English.

75. Scott JW, Somodi GC, Jones JB. Testing tomato genotypes and breeding for resistance to bacterial wilt in Florida. In: Hartman GL, Hayward AC, editors. Bacterial wilt. Canberra: ACIAR; 1993. English.

76. Thakur AK, Kohli UK, Kumar M. Inheritance of resistance to bacterial wilt in tomato (*Lycopersicon esculentum Mill*.). Ind. Jour. of Gen. and Plant Breed. 2004;64(1):79-80. English.

77. Sharma KC, Sharma LK. Genetic studies of bacterial wilt resistance in tomato crosses under mid-hill conditions of Himachal Pradesh. Jour. of Hil. Agr. 2015;6(1):136-137. English.

78. Danesh D, Aarons S, Mcgill GE, Young ND. Genetic dissection of oligogenic resistance to bacterial wilt in tomato. Mol. Plant-Mic. Int. 1994;7:464-471. English.

79. Mangin B, Thoquet P, Olivier J, Grimsley NH. Temporal and multiple quantitative trait loci analyses of resistance to bacterial wilt in tomato permit the resolution of linked loci. Genetics. 1999;151:1165-1172. English.

80. Cruz CD. Principles of quantitative genetics. Viçosa: UFV; 2012. English.

81. Cruz CD, Carneiro PCS, Regazzi AJ. Biometric models applied to genetic breeding. 3ª ed. Viçosa: UFV; 2014. English.

82. Fiorini CVA, Gomes LAA, Libânio RA, Maluf WR, Campos VP, Licursi V, et al. Identification of progenies F2:3 of homozygous lettuce resistant to gnats nematodes. Hort. 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