Review article

Tomato breeding for disease resistance

Gabriella Queiroz de Almeida [1*], Juliana de Oliveira Silva[2], Mariane Gonçalves Ferreira Copati[1], Felipe de Oliveira Dias[1], Manoel Coelho dos Santos[1]

[1] *Departamento de Fitotecnia, Universidade Federal de Viçosa (UFV), CEP 36570-900, Viçosa, Minas Gerais, Brasil.*
[2] *Department of Nematology, University of California Riverside, 92521, Riverside, California, USA.*

**Corresponding author: Email: gabriellaqueirozalmeida@hotmail.com*

ABSTRACT. In the genetic breeding of tomatoes, not only productivity, but also factors related to fruit quality and pest and disease management are taken into account. In this context, diseases stand out, since they are the main bottlenecks for successful cultivation. Currently, the search for more sustainable crops has demanded from producers' alternatives to disease control to reduce the use of pesticides. Among the diseases that most reduce tomato production in Brazil, whether for table or industry, we can mention late blight, black spot, fusarium wilt, viruses, bacterial and nematode diseases. Genetic resistance, obtained by genetic breeding programs, is one of the best tools to deal with diseases to depend less on pesticides. Thus, this review aims to provide an overview of tomato breeding programs in terms of resistance to the main diseases that affect this crop.

Keywords: *Solanum lycopersicum;* genetic resistance; pathogens; tomato crop.

DOI: https://doi.org/10.33837/msj.v3i3.1287

Received August 7, 2020. Accepted September 10, 2020.
Associate editor: Ana Siqueira

INTRODUCTION

The tomato (*Solanum lycopersicum* L.) is a species native to South America, being a fruit vegetable that has economic, nutritional importance and as a model organism for biotechnological research. In tomato production, whether for industry or table, diseases are a major concern worldwide. Diseases cause great economic losses due to reduced yields resulting from these crop diseases. Thus, breeding aimed at resistance is an important management tool, which aims to eliminate or reduce the use of pesticides in tomato production through the use of resistant genotypes (Foolad 2007).

It is desirable that the selection regarding resistance is made within the cultivated species (*Solanum lycopersicum* L.) or derived from intra-specific crosses (within the *lycopersicon* group), so that the selection of characters of agronomic interest and fruit quality is not lost with the link drag of interspecific intersections. When the source of resistance is found in wild materials outside the species *S. lycopersicon* it is recommended that an adaptation be made to the germplasm (pre-breeding) (Nick *et al*. 2013).

The selection of resistant genotypes can be done using biotechnology, but it can also be done using the area under the disease progress curve (AUDPC). The format of the disease progress curve presents important information about the dynamics of the disease, allowing its use for the selection of the best genotypes. In this situation, the genotypes with the least damaged leaf area in the last days of evaluation are considered to be the best, and priority is given to the selection of genotypes whose injured leaf area is smaller in the initial evaluation periods, which can provide greater efficiency of control methods. in the initial period of disease progression and reduce risks of damage to the crop (Laurindo *et al*. 2015).

In tomato, resistance mechanisms include qualitative inheritance (monogenic or oligogenic) and quantitative inheritance (polygenic), which have been the focus of breeding programs for years. In breeding programs, the introgression of this qualitative inheritance gene can be accomplished through the use of backcross. However, plants with resistance to specific breeds by means of main genes (vertical resistance) are effective in protecting only at the beginning of the infection (Fry *et al*. 1993; Klarfeld *et al*. 2009). For quantitative inheritance genes, a recurrent selection program can be used. Furthermore, selection assisted by molecular markers is an extremely efficient

strategy to assist in the pyramid process of these genes. The gene pyramiding will guarantee the new cultivar more time on the market due to the greater difficulty of the pathogen in overcoming this resistance (Young and Kelly 1996). Thus, this review aims to provide an overview of tomato breeding programs in terms of resistance to the main diseases that affect this crop.

Resistance to black spot

Black spot is one of the main leaf diseases of tomato culture, with yield reductions of up to 78% (Upadhyay *et al*. 2016). It is caused by the fungus *Alternaria* spp. (Lourenço Jr. *et al*. 2009; Rodrigues *et al*. 2010), mainly for the species *A. solani* Sorauer (Rotem 1994) and *A. tomatophila* (Simmons 2000). Because they are morphologically very similar, they are part of the *A. linearіae* species (Woudenberg *et al*. 2014).

Black spot epidemics can occur at any time of the year, however it is more severe in regions with high relative humidity (wetting time above 4h) combined with high temperatures (optimal temperature range between 25 and 30 ºC) (Batista *et al* 2006; Salustiano *et al*. 2006). This broad development of the disease is due to the genetic variability of the populations of the pathogens, which can adapt to different environmental conditions (Foolad *et al*. 2008). The black spot causes a reduction of the photosynthetic area and, in severe cases, can cause defoliation of the plants. As the disease progresses, elongated and circular necrotic lesions form, forming concentric rings, in leaves, stems and fruits, it can be observed at any stage of plant development, but it is more severe in mature and senescent tissues (Agrios 2005).

The black spot is difficult to control, as the pathogen is highly virulent and has a short life cycle, also its conidia are disseminated by the wind, and can reach long distances (Kemmitt 2002). The control is based almost exclusively on intensive fungicide applications, as until now, there are no commercial tomato cultivars resistant to black spots in Brazil (Catão *et al*. 2013). In the USA there are already resistant cultivars, such as the F1 hybrids: Mountain Magic, Iron Lady and Jasper.

Resistance to black spot is a complex characteristic (non-additive interaction) of quantitative inheritance (governed by several genes where the plant produces physical and chemical barriers that partially prevent penetration, infection and colonization by the pathogen) and of low heritability. Thus, the launch of a resistant cultivar is more difficult, especially when marker-assisted breeding is not used. This difficulty may be related to the genotype x environment interaction, which can mask the responses of the evaluated genotypes and, also, because it requires successive evaluations of a large number of individuals, which reduces the accuracy of the responses (Foolad *et al*. 2008).

There are reports of resistance to black spots in the wild species *S. habrochaites*, *S. arcanum*, *S. peruvianum*, *S. neorickii* and *S. chilense* (Foolad et al. 2008), but most accessions resistant to black spots belong to the species *S. habrochaites* which is not considered to be a good parent due to the link drag (Kumar and Srivastava 2013). With this in mind, work on the identification of resistant germplasms within the cultivated species (*S. lycopersicum*) has been carried out, such as: Grigolli *et al*. (2011) and Laurindo *et al*. (2015), who identified in the Vegetable Germplasm Bank (BGH) of the Federal University of Viçosa (UFV) accesses with high levels of resistance to *A. solani* and *A. tomatophila* respectively, which presented reduced values of AUDPC (area under the disease progress curve); Catão *et al*. (2017) identified, based on the AUDPC, a commercial strain (CH152) of *Solanum lycopersicum* var. *cerasiforme* resistant to *A. tomatophila*.

To understand the genetic control of this trait and facilitate its introgression in tomato cultivars, molecular markers (SNPs) and QTL (quantitative trait loci) mapping have been carried out. These studies compare the host's genome (tomato introgression lines) with the pathogen's genome, to determine the molecular basis of the infection, as well as the host's response, but stable QTL's have not yet been validated (great additive and independent effects of epistasis) to facilitate improvement. In the field of transgenics, the rice chitinase (defense mechanism) gene (RCG3) was identified, which confers resistance to black spot in genetically modified strains of S. lycopersicum (Jabeen *et al*. 2015).

Resistance to late blight

Late blight is caused by oomycete *Phytophthora infestans* Bary (Mont.), this pathogen is one of the most destructive of the cultivated tomato, which results in significant losses in crop production, since most tomato cultivars are susceptible to late blight. (Nowicki *et al*. 2012). The first reports of late blight were recorded in the United States, in 1843. Subsequently, the pathogen spread to Europe through an expedition of infested seed potatoes (Nowicki *et al*. 2013). The main causes of the rapid destruction caused to culture by late blight are: i) high rate of disease progression; ii) difficult to detect low levels of the pathogen in the field; iii) high rate of production of new sporangia in each injury; and iv) low time to complete an asexual cycle of the pathogen (Fry and Goodwin 1997; Foolad *et al*. 2008).

The success of *P. infestans* as a phytopathogenic agent is related to its reproductive cycle, which can occur asexual or sexually (Nowicki *et al*. 2012). In the asexual cycle, sporangia are produced in sporangiophores that grow from infected tissue (Fry

2008). Sporangia can germinate directly forming a germ tube at high temperatures (about 20-25°C optimal) and high relative humidity (above 90%), or indirectly, forming zoospores at lower temperatures (optimal between 10 and 15°C) (Lima *et al*. 2009).

Symptoms can occur anywhere in the plant, but the leaves are more intense; irregularly shaped, brownish-olive colored lesions appear on the leaflets, with the presence of a lighter green halo around the leaf spots and a wet appearance is observed in the lesions. In conditions of high relative humidity, there is the growth of sporangiophores and sporangia on the abaxial part of the leaves, giving a whitish color around the lesion, very similar to a white, thin mold (Bosco *et at*. 2009).

The management of late blight is done mainly by the principle of protection of the host, through the application of fungicides (Fiorini *et al*. 2010). However, the indiscriminate use of fungicides has resulted in the development of resistant forms of the pathogen. Thus, the most promising approach to achieve control is the use of resistant genotypes (Park *et al*. 2005). In tomato, the mechanisms of resistance to late blight include qualitative (monogenic) and quantitative (polygenic) inheritance, which have been the focus of breeding programs for many years.

Studies on qualitative resistance in this culture are related to the discovery of resistance alleles in wild species such as *S. pimpinellifolium* L. Five resistance genes have been found, mapped on chromosomes 7 (Ph-1), 10 (Ph-2), 9 (Ph-3) (Nowicki *et al*. 2012), 2 (Ph-4) (Li *et al*. 2011) and 1 (Ph-5) (Foolad *et al*. 2008). The Ph-1 gene is the only one that has complete dominance providing resistance against the pathogen's T-0 race, but it has been quickly supplanted by new breeds. The Ph-2 gene provides only partial resistance against several isolates of the pathogen. Ph-3 has incomplete dominance for several isolates of P. infestans, being a stronger resistance gene (Chen *et al*. 2008; Nowicki *et al*. 2012). Currently, the use of Ph-2 together with Ph-3 is sought, since when used together, a high level of resistance was observed (Panthee and Gardner 2010; Foolad *et al*. 2014). F1 hybrids with the combined Ph-2 and Ph-3 genes are commercialized in the USA, for example: Mountain Magic, Mountain Merit, Defiant, Cherry Bomb, Iron Lady and Jasper.

Quantitative resistance in tomato was found in *S. habrochaites* (Brouwer and St. Clair 2004; Brouwer *et al*. 2004; Abreu *et al*. 2008) and *S. pennellii* (Smart *et al*. 2007). However, these sources of resistance have not yet been successfully implemented in breeding programs, since many of the late blast resistance QTLs identified in *S. habrochaites* are linked to undesirable characteristics, which leads to the dragging of link when employing traditional strategies of improvement. (Brouwer and St. Clair 2004; Ohlson and Foolad 2016).

With this in mind, work to identify resistant germplasms within the cultivated species (*S. lycopersicum*) has been carried out, such as Fiorini *et al*. (2010) and Laurindo *et al*. (2016) identified in the Vegetable Germplasm Bank (VGB) of the Federal University of Viçosa (UFV) accesses with high levels of resistance to late blight, which presented reduced values of AUDPC (area under the disease progress curve).

Resistance to fusarium wilt

Tomato fusarium wilt is a soil disease caused by the fungus *Fusarium oxysporum* f. sp. *lycopersici* (FOL), which has three physiological races and is one of the main phytosanitary problems of culture in Brazil (Alexander and Tucker 1945; Booth 1971; Grattidge and Obrien 1982). The pathogen can infect plants at different stages, as the disease progresses, the foliage and branches turn yellow and gradually wilt and die. The best way to visualize and identify the infection is through the cross-section of the tomato stem, where an intense brown color is observed in the xylem region (Gonzalez-Cendales *et al*. 2016).

With the death of the plant, the fungus continues to colonize the cultural remains where the chlamydospores form. These can be transported to other areas through seeds, soil, water, contaminated material and agricultural machinery (Ajilogba and Babalola 2013). The occurrence of infection is favored by temperatures between 21 and 33°C combined with high humidity. Factors such as low soil pH (less than 5.5) and use of ammonium-based fertilizers also contribute to increasing the severity of the disease (McGovern 2015). The most used methods for controlling fusarium wilt are: the use of resistant cultivars (even if they are as rootstock) and cultural practices. Although quantitative and qualitative resistance against fusarium wilt have been identified, qualitative resistance is the most used in breeding programs for the development of commercial cultivars (McGovern 2015).

Four resistance genes of the so-called series I (Immunity) confer specific resistance to the race of FOL isolates. The gene designated I 1 confers resistance to race 1, I 2 to race 2, and I 3 and I 7 to race 3 (Ma *et al*. 2013). All genes were identified in wild species (*S. pimpinellifolium* and *S. pennellii*) and introgressed by backcross in commercial cultivars. Thus, Carrer Filho *et al*. (2016) suggest the joint use of the molecular markers SSR - 67, TFusrr and SSRD1, using multiplex PCR, for the simultaneous selection of tomato accessions resistant to races 1, 2 and 3 of *F. oxysporum* f. sp. *lycopersici*. The phenotypic selection of tomato accessions for resistance to FOL breeds has been done in tests with seedling inoculation and evaluations with the aid of a descriptive scale (Santos 1996), with notes based on external symptoms and

vascular discoloration, as well as the use of differentiating witnesses of the races (Reis *et al*. 2004; Santos-Júnior *et al*. 2009).

The identification of sources of resistance to FOL is essential for use in breeding programs, since new breeds of the pathogen may arise due to the selection pressure generated by the extensive use of a specific resistant cultivar (Reis *et al*. 2004). Research that seeks to identify sources of resistance has been carried out, such as Reis *et al*. (2004) found sources of multiple resistance in tomato accessions in the wild species of *S. habrochaites*, *S. chilense*, *S. pennellii* and *S. peruvianum*; Carrer Filho *et al*. (2015) identified resistance to the three breeds in accessions of *S. peruvianum* and *S. corneliomuelleri*; Santos-Júnior *et al*. (2009) identified ten experimental tomato hybrids from the Embrapa Tomato Germplasm Bank resistant to race 3. Today in Brazil there are already many tomato cultivars resistant to the three FOL breeds, including BRS Imigrante launched by Embrapa Hortaliças (Embrapa Hortaliças 2013), in addition to the F1 hybrids launched by the companies VILMORIN, TSVsementes, SAKATA, FELTRIN among others.

Resistance to viruses

Among the main viral diseases that attack tomatoes are those caused by species classified in the genera *Tospovirus*, *Begomovirus*, *Potyvirus* and *Crinivirus*. These viruses can lead to total crop loss and there is no chemical control for them, what is done is the control of the vector with insecticides, the uprooting of plants with symptoms (roughing) and sanitary voids to reduce the incidence in the region. Therefore, the use of resistant cultivars is the most promising method for controlling these diseases (Becker *et al*. 2016).

The main viruses that affect the tomato has as a vector the thrips (*Frankliniella schultzei*), and it is caused mainly by the species *Tomato spotted wilt virus* (TSWV), *Groundnut ringspot virus* (GRSV), *Tomato chlorotic spot virus* (TCSV) and *Chrysanthemum stem necrosis virus* (CSNV) that are of the genus *Tospovirus* (Dianese et al. 2011). The first gene identified with a "broad" spectrum of resistance to tospovirus was called Sw-5 and originally described in *S. peruvianum*, and later alternative sources of resistance were identified in accessions of *S. chilense*, *S. corneliomuelleri*, *S. lycopersicum*, *S. pimpinellifolium*, *S. arcanum* and *S. habrochaites* (Dianese *et al*. 2011). The introgression/incorporation of these resistance alleles in commercial varieties can allow the development of materials with broad resistance (quantitative resistance).

Different vectors are responsible for the spread of viruses in the tomato crop. Due to the increase in whitefly populations, viruses caused by the genus *Begomovirus* have grown a lot in Brazil and the main species found are *Tomato severe rugose virus* (ToSRV), *Tomato yellow vein streak virus* (ToYVSV), *Tomato common mosaic virus* (ToCmMV) and *Tomato chlorotic mottle virus* (ToCMoV) (Aguilera *et al*. 2014). Resistance genes have already been identified in wild species, some of these described genes include Ty-1, Ty-2, Ty-3 and Ty-4. Currently, the commercialized hybrids have the Ty-1 gene that gives tolerance to these species, but there are no resistant hybrids. With that in mind, Aguilera *et al*. (2014) found tolerant accessions in *S. lycopersicum* in the VGB of Federal University of Viçosa in field or greenhouse conditions, but resistance was found in an accession of *S. peruvianum* L., which had an excellent adhesion in both conditions, attributed to the presence of Ty-2 and Ty-3 alleles of the heterosis resistance gene.

There are two described species of potyvirus that affect tomato in Brazil: *Potato virus Y* (PVY) causal agent of tomato streak (Zerbini and Maciel-Zambolim 1999) and *Pepper yellow mosaic virus* (PepYMV) (Maciel-Zambolim *et al*. 2004), with aphids as vectors that in the tomato especially induce the mosaic symptom (Dianese *et al*. 2008). At the moment, commercial cultivars are apparently all susceptible to PepYMV, so losses in productivity reach large proportions as reported in producing regions in Espírito Santo (Maciel-Zambolim *et al*. 2004). Two wild accessions of *S. habrochaites* were identified as potential sources of resistance, both to PepYMV and to PVY (accessions 'CNPH 1121' and 'CNPH 1122'). The resistance of both viruses is due to the presence of the pot-1 gene or an allele, once *S. habrochaties* is the source of this resistance character (Ruffel *et al*. 2005).

The emerging disease known as yellowing is caused by *Tomato chlorosis virus* (ToCV, genus *Crinivirus*, family Closteroviridae), is limited to phloem and transmitted by the whitefly complex (*Bemisia tabaci*) in a semi-persistent manner (Brown, 1994; Bedford et al. , 1994; Faria et al., 2000). An alternative to combat this disease is genetic improvement that reduces the impact of criniviruses on tomato production (direct to the virus and indirect resistance to the whitefly vector). Resistance to ToCV in accessions of *S. pennellii* and *S. habrochaitese* is related to resistance to whitefly mediated by acyl sugars present in leaf trichomes. One of the sources of resistance are lines 802-11-1 and 821-113-1, derived from the IAC CN RT population (from the interspecific hybridization of *Solanum lycopersicum* x *S. peruvianum*) and LA1028 (*S. chmielewskii*), respectively (Garcia-Cano *et al*. 2010). Inheritance studies with these sources are necessary to better understand their respective mechanism(s) of resistance.

Resistance to bacterial diseases

The bacterial leaf spot in tomato, which is caused by four species of *Xanthomonas*: *X. euvesicatoria* (race T1), *X. vesicatoria* (race T2), *X. perforans* (races T3, T4 and

T5) and *X. gardneri* (race T2), has already been reported in Brazil (Potnis *et al*. 2015). This disease causes severe losses of yield and quality due to defoliation and formation of necrotic lesions in the fruits. The conditions conducive to the development of the disease are high temperature and humidity. Bhattarai *et al*. (2017) evaluated 63 advanced tomato strains, with several genetic origins, in greenhouse and field, for resistance to the T4 race, which was found to be prevalent in North Carolina. Race T4 isolated 9 was used to inoculate plants by spraying and disease severity was measured using the Horsfall-Barratt scale. The resistant strains were derived from a specific strain of *S. pimpinellifolium*, demonstrating that within this wild species there are sources of resistance for the control of bacterial spot.

The bacterial speck disease of tomato is caused by *Pseudomonas syringae* pv. *tomato* (Pst), which proliferates mainly in leaves and fruits, suffers necrotic lesions and consequently decreases production yield (Thapa *et al*. 2015). It occurs predominantly in regions with mild temperatures and high humidity. Genetic studies have identified dominant resistance genes, called Pto-1/Pto-4 and Prf. These genes were used in *S. pimpinellifolium* and were rapidly introgressed in commercial cultivars, and for years they were resistant to a race 0 of Pst, but currently, the problem is a race 1 that has not grown resistant. Thapa *et al*. (2015) working with interspecific cross lines, between *S. peruvianum*×*S. lycopersicum* and *S. habrochaites*×*S. lycopersicum* mapped QTLs applied for resistance to Pst.

Bacterial wilt is caused by *Ralstonia solanacearum*, which is a soil bacterium, which infects the plant through injuries to root tissues (Nakaho *et al*. 2017). The only effective method to avoid the damage caused by this disease is to use plants grafted with tolerant cultivars or with quantitative resistance, as rootstocks. The initial symptoms are characterized by wilt of the terminal leaves, darkening of the vascular region, wilt of leaflets and leaf epinastia. With the progression of the disease, the wilt affects the entire plant, occurring at the death of the plant. In highly resistant tomato cultivars derived from the wild tomato *S. pimpinellifolium*, the growth of *R. solanacearum* in intercellular spaces is suppressed and the invasion of bacteria in vascular tissues is inhibited. The resistance to *R. solanacearum* in tomato rootstock cultivars is genetically controlled by several loci of quantitative traits (QTL). Nakaho *et al*. (2017) suggest that the response of the LS-89 cultivar is a true hypersensitive response, and the induction of this vascular response in xylem parenchyma and marrow cells surrounding the xylem vessels seems to be associated with quantitative resistance to *R. solanacearum*.

Bacterial wilt is one of the factors limiting the performance of tomatoes in regions predominantly hot and humid, such as in the Amazon. Thinking about it Souza *et al*. (2013) evaluated the behavior of F12, F13 and F14 strains of the Yoshimatsu HT-16 crossing in soils naturally infested by the pathogen, comparing it with other commercial varieties under cultivation conditions in the municipality of Parintins - AM. In this work, the authors concluded that all the progenies evaluated and the resistant cultivar Y-4-1 used as a control, presented higher levels of genetic resistance and fruit yield in relation to the susceptible control Santa Cruz Kada, recommending the lines derived from this group Yoshimatsu for free-standing cultivation or for rootstock in that region.

Bacterial wilt and canker of tomato is one of the most destructive bacterial diseases in tomatoes (Wittmann *et al*. 2016). It is caused by the gram-positive bacterium *Clavibacter michiganensis* subsp. *michiganensis* (Cmm), which enters the cell through wounds and colonizes the xylem vessels, causing necrotic lesions on the sides of the leaves and the stems, leading to low productivity and even the death of the plant. Antimicrobials, such as copper compounds, hydroxyquinoline, streptomycin or tetracycline, reduce the incidence of the disease, but do not control the bacteria. Of the wild tomato species, *L. hirsutum* was identified as resistant to Cmm, however there are no resistant cultivars found. The bacteriophage CMP1 endolysin (Lys) gene, which encodes a peptidase responsible for reducing *C. michiganensis*, specifically hydrolyzing its murein, was transferred to tomato plants by mediated *Agrobacterium*. The presence of the gene was verified by PCR and the product of the gene was confirmed in immunoblots and stably expressed in three generations. Transgenic tomato plants did not show symptoms of disease after infection with *C. michiganensis* subsp. *michiganensis*, although small amounts of bacteria can still be identified in xylem and leaf extracts, although in significantly reduced amounts (Wittmann *et al*. 2016).

Resistance to nematodes

The root-knot nematode (*Meloidogyne* spp.), commonly known for its main symptom, which is the deformation of the root system of plants, forming structures called galls, causes serious losses in tomato culture. The development of the plant is reduced, as it is unable to absorb water and nutrients efficiently. Its control is challenging for two reasons, because it has a high number of species (around 98) and because it parasitizes a wide range of hosts, which makes it difficult to use crop rotation (Abad *et al*. 2003; Jones *et al*. 2013).

The control of the root-knot nematode in tomato cultivation is very problematic, since these organisms are inhabitants of the soil and under favorable environmental conditions they multiply

quickly and are protected from the action of pesticides or antagonistic organisms (Pinheiro *et al*. 2014), due to this, several control methods, aiming at the suppression of this nematode, have been researched in an attempt to decrease the nematode population density to a level that does not cause economic damage to the culture, trying to make this process more efficient and economical. Genetic resistance is one of the best ways to control nematodes, and one of the most sought after as well, as it is easily assimilated by farmers, does not increase production costs, minimizing risks to human health and does not cause damage to the environment.

Resistance to root-knot nematodes was identified more than 70 years ago, when the *Mi*-1 gene located on chromosome 6 of the wild species *S. peruvianum* L. (Ho *et al*. 1992) was identified. This gene confers resistance to the three main species of root-knot nematode, *M. incognita*, *M. javanica* and *M. arenaria*. Resistance is associated with a hypersensitive response, characterized by cell death located in the host's tissue near the site of the invasive nematode's establishment. However, this gene is not always efficient in suppressing the reproduction of gall nematodes, since it can become inactive at soil temperatures above 28 ^{o}C or may not include certain breeds or species, such as *M. enterolobii* (Pinheiro *et al*. 2014; Rosa *et al*. 2014).

Although the only commercially available source of resistance to root-knot nematodes in tomato is in the dominant *Mi*-1 gene, other resistance genes have already been identified (*Mi*-2, *Mi*-3, *Mi*-4, *Mi*-5, *Mi*-6, *Mi*-7, *Mi*-8, *Mi*-9 and *Mi*-HT). The *Mi*-2, *Mi*-3, *Mi*-4, *Mi*-5, *Mi*-6, *Mi*-9 and Mi-HT genes are stable at high temperatures, however, due to the incompatibility of crossing, it has not been possible to transfer these genes from wild species for cultivated tomatoes (El-Sappah *et al*. 2019). The *Mi*-3 and *Mi*-5 genes were mapped on chromosome 12, the *Mi*-9 gene is homologous to the *Mi*-1 gene, mapped on the shortest arm of chromosome 6, as well as the *Mi*-HT gene, the other resistance genes have not yet been mapped (Wang *et al*. 2013; El-Sappah *et al*. 2019).

More recently, the *Mi*-9 gene (introgressed from the wild species *S. arcanum* LA2157) has been cloned and characterized, demonstrating its resistance to the three species *M. incognita*, *M. javanica* and *M. arenaria*, as well as the *Mi*-1 gene, however, with the thermal stability differential, being effective up to 32 ^{o}C (Jablosnka *et al*. 2007). Six markers were used to map *Mi*-9, two of them based on RFPL (C32.1 and C264.2) and four based on PCR (CT119, REX-1, APS-1 and C&B) (Ammiraju *et al*. 2003; Jablonska *et al*. 2007).

Studies have shown, through crosses and phenotypic analyzes, that heat-stable resistance is a characteristic inherited independently as a single dominant gene which has been designated as the *Mi*-9 gene. This gene was mapped in a region very close to the site of the *Mi*-1 gene, indicating that it may be a member of the *Mi*-1 family that has evolved to provide resistance to heat. Molecular markers such as REX1 and C8B can be used to select the *Mi*-9 gene and incorporate it into cultivated tomatoes (Ammiraju *et al*. 2003).

There are several possibilities for root-knot nematode resistance genes in wild tomato species, the current challenge is to isolate these genes and introgress them in tomato plants grown using modern biotechnology and analyzing them via conventional breeding approaches, such as crossbreeding, or transgenetically. Studies with transgenic tomatoes have already shown some results in reducing the population of root-knot nematodes, such as those carried out by Li *et al*. (2007) who observed that tomatoes with the Cry6A gene from *Bacillus thuringiencis* (Bt) reduced the reproduction of *M. incognita* and by Chan *et al*. (2015) who presented the delay in embryogenesis of nematode eggs when inoculated in tomato plants with the CeCPI + PjCHI-1 genes from Taro and *Paecilomyces javanicus*.

The newest technology studied aiming the resistance of tomato to root-knot nematodes is the CRISPR-Cas9 strategy. Researchers at the University of California are developing a project that aims at the genetic characterization and biological variation of commercially relevant nematodes (National Institute of Food and Agriculture 2019). The greater the knowledge on the nematode-tomato genetic interaction, the greater the progress in obtaining and controlling genes linked to resistance to these nematodes.

CONCLUSION

The wild species of *S. habrochaites*, *S. chilense*, *S. pennellii*, *S. peruvianum* and S. corneliomuelleri are the main sources of resistance to the numerous diseases that affect tomatoes. In genetic improvement programs, the introgression of the qualitative inheritance genes can be successfully accomplished through the use of backcross. Within the cultivated group, there are already genotypes identified as possible sources of quantitative resistance that can be used in recurrent selection programs. Furthermore, selection assisted by molecular markers is an extremely efficient strategy to assist in the pyramidation process of these genes. The gene pyramiding will guarantee the new cultivar more time on the market due to the greater difficulty of the pathogen in overcoming this resistance.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

Abad, P.; Favery, B.; Rosso, M.N.; Castagnone-Sereno, P. 2003 Root-knot nematode parasitism and host response: Molecular basis of a sophisticated interaction. *Molecular Plant Pathology* 4: 217–224.

Abreu, F.F.B.; Silva, D.J.H.; Cruz, C.D.; Mizubuti, E.S.G. 2008. Inheritance of resistance to *Phytophththora infestans* (Peronnosporales, Pythiaceae) in a new source of resistance in tomato (*Solanum* sp. (formerly *Lycopersicon* sp.), Solanales, Solanaceae). *Genetics and Molecular Biology* 31: 493-497.

Agrios, G.N. 2005. *Plant pathology*. 5th ed. New York: Academic, 922 p.

Aguilera, J.G.; Hurtado, F.D.; Ramos Sobrinho, R.; Almeida, V. de S.; Tavares, S.S.; Nick, C.; *et al*. 2014. Response of tomato (*Solanum* L. section *Lycopersicon* Mill.) germplasm to begomovirus inoculation under controlled and field conditions. *Genetic Resource Crop Evolution* 61:435-450.

Ajilogba, C.F.; Babalola, O.O. 2013. Integrated management strategies for tomato *Fusarium* wilt. *Biocontrol Science* 18: 117-127.

Alexander, L.J.; Tucker, C.M. 1945. Physiologic specialization in the tomato wilt fungus *Fusarium oxysporum* f. sp. *lycopersici*. *Journal of Agricultural Research* 70: 303-313.

Ammiraju, J.S.S.; Veremis, J.C.; Huang, X.; Roberts, P.A.; Kaloshian, I. 2003. The heat-stable, root-knot nematode-resistance gene *Mi*-9 from *Lycopersicon peruvianum* is localized on the short arm of chromosome 6. *Theorical and Applied Genetics* 106: 478-484.

Batista, D.C.; Lima, M.A.; Haddad, F.; Maffia, L.A.; Mizubuti, E.S.G. 2006. Validation of decision support systems for tomato early blight and potato late blight, under Brazilian conditions. *Crop Protection* 25: 664-670.

Becker, W.F.; Wamser, A.F.; Feltrim, A.L.; Suzuki, A.; Santos, J.P.; Valmorbida, J.; Hahn, L.; Marcuzzo, L.L; Mueller, S. 2016. *Sistema de produção integrada para o tomate tutorado em Santa Catarina*. Florianópolis, SC: Epagri, 149p.

Bedford, I.D.; Briddon, R.W.; Brown, J.K.; Rosell, R.C.; Markham, P.G. 1994. Geminivirus transmission and biological characterization of *Bemisia tabaci* (Gennadius) biotypes from different geographic regions. *Annals of Applied Biology* 125: 311-325.

Bhattarai, K.; Louws, F.J.; Williamson, J.D.; Panthee, D.R. 2017. Resistance to *Xanthomonas perforans* race T4 causing bacterial spot in tomato breeding lines. *Plant Pathology* 66: 1103-1109.

Booth, C. 1971. *The Genus Fusarium*. Kew: England**.** Common-wealth Mycological Institute, 237p.

Bosco, L.; Heldwein, A.; Lucas, D. 2009. Sistema de previsão de ocorrência de requeima em clones de batata suscetíveis e resistentes. *Ciência rural* 39: 1024-1031.

Brouwer, D.J.; St. Clair, D.A. 2004. Fine mapping of three quantitative trait loci for late blight resistance in tomato using near isogenic lines (NILs) and sub-NILs. *Theoretical and Applied Genetics* 108: 628-638.

Brouwer, D.J.; Jones, E.S.; Clair, D.A.S. 2004. QTL analysis of quantitative resistance to *Phytophthora infestans* (late blight) in tomato and comparisons with potato. *Genome* 47: 475–492.

Brown, J.K. 1994. Current status of *Bemisia tabaci* as a plant pest and virus vector in agroecosystems worldwide. *FAO Plant Protection Bulletin* 42: 3-32.

Carrer Filho, R.; Dias, V.D.; Oliveira, R.M.; Dianese, E.C.; Boiteux, L.S. Cunha, M.G. 2016. Detecção simultânea de fatores de resistência à murcha de fusário do tomateiro por meio de PCR multiplex. *Pesquisa Agropecuária Brasileira* 51: 925-932.

Carrer Filho, R.; Oliveira, R.M.; Dias, V.D.; Boiteux, L.S.; Dianese, E.C.; Cunha, M.G. 2015. Fontes de resistência múltipla à murcha de fusário em tomateiro. *Pesquisa Agropecuária Brasileira* 50: 1225-1231.

Catão, H.C.R.M.; Sales, N.L.P.; Azevedo, D.M.Q.; Flávio, N.S.D.S.; Menezes, J.B.C.; Barbosa, L.V.; Martinez, R.A.S. 2013. Fungicides and alternative products in the mycelial growth and germination control of *Alternaria tomatophila*. *Idesia* 31: 21-28.

Catão, H.C.R.; Sales, N.L.P.; Menezes. J.B.C.; Caixeta, F.; Costa, C.A. 2017. Productive potential of the cherry tomato genotype group before infection by *Alternaria tomatophila*. *Revista Caatinga* 30: 296-302.

Chan, Y.-L.; He, Y.; Hsiao, T.-T.; Wang, C.-J.; Tian, Z.; Yeh, K.-W. 2015. Pyramiding taro cystatin and fungal chitinase genes driven by a synthetic promoter enhances resistance in tomato to root-knot nematode *Meloidogyne incognita*. *Plant Science* 231: 74-81.

Chen, C.-H.; Sheu, Z.-M.; Wang, T.-C. 2008. Host Specificity and Tomato-Related Race Composition of *Phytophthora infestans* Isolates in Taiwan During 2004 and 2005. *Plant Disease* 92: 751-755.

Dianese, E.C.; Fonseca, M.E.N.; Inoue-Nagata, A.K.; Resende, R.O.; Boiteux, L.S. 2011. Search in *Solanum* (section *Lycopersicon*) germplasm for sources of broad-spectrum resistance to four Tospovirus species. *Euphytica* 180: 307-319.

Dianese, E.C.; Resende, R.O.; Inoue-Nagata, A.K. 2008. Alta incidência de *Pepper yellow mosaic virus* em tomateiro em região produtora no Distrito Federal. *Tropical Plant Pathology* 33: 67-68.

El-Sappah, A.H.; Islam, M.M.; El-Awady, H.H.; Yan, S.; Qi, S.; Liu, J.; Gheng, G.; Liang, Y. 2019. Tomato natural resistance genes in controlling the root-knot nematode. *Genes* 10: 1-19.

Embrapa Hortaliças. 2013. *BRS-Imigrante: híbrido tipo salada tolerante à begomovírus e Fusarium raça 3*. Available from: https://www.embrapa.br/en/busca-de-publicacoes/-/publicacao/982292/brs-imigrante-hibrido-tipo-salada-tolerante-a-begomovirus-e-fusarium-raca-3. Accessed on 10/04/2019.

Faria, J.C.; Bezerra, I.C.; Zerbini, F.M.; Ribeiro, S.G.; Lima, M.F. 2000. Situação atual das geminiviroses no Brasil. *Fitopatologia Brasileira* 25: 125-137.

Fiorini, C.V.A.; Silva, D.J.H.; Silva, F.F.; Mizubuti, E.S.G.; Alves, D.P.; Cardoso, T.S. 2010. Agrupamento de curvas de progresso de requeima, em tomateiro originado de cruzamento interespecífico. *Pesquisa Agropecuária Brasileira* 45: 1095-1101.

Foolad, M.R. 2007. Genome mapping and molecular breeding of tomato. *International Journal of Plant Genomics* Doi:10.1155/2007/64358.

Foolad, M.R.; Sullenberg, T.; Ohlson, E.W.; Gugino, B.K. 2014. Response of accessions within tomato wild species, *Solanum pimpinellifolium* to late blight. *Plant Breeding* 133: 401-411.

Foolad, M.R.; Merk, H.L.; Ashrafi, H. 2008. Genetics, genomics and breeding of late blight and early blight resistance in tomato. *Critical Reviews in Plant Sciences* 27: 75-107.

Fry, W. 2008. *Phytophthora infestans*: the plant (and R gene) destroyer. *Molecular plant pathology* Doi.org/10.1111/j.1364-3703.2007.00465.x.

Fry, W.; Goodwin, S.B.; Dyer, A.T.; Matuszak, J.M.; Drenth, A.; Tooley, P.W.; *et al*. 1993. Historical and Recent Migrations of Phytophthora-Infestans - Chronology, Pathways, and Implications. *Plant Disease* 77: 653-661.

Fry, W.E.; Goodwin, S.B. 1997. Re-emergence of Potato and Tomato Late Blight in the United States. *Plant Disease* 81: 1349-1357.

Garcia-Cano, E.; Navas-Castillo, J.; Moriones, E.; Fernândez-Munõz, R. 2010. Resistance to *Tomato chlorosis virus* in wild tomato species that impair virus accumulation and disease symptom expression. *Phytopathology* 100: 582-592.

García-Cano, E.; Resende, R.O.; Fernández-Muñoz, R.; Moriones, E. 2006. Synergistic interaction between *Tomato chlorosis virus* and *Tomato spotted wilt virus* results in breakdown of resistance in tomato. *Phytopathology* 96: 1263-1269.

Gauffier, C.; Lebaron, C.; Moretti, A.; Constant, C.; Moquet, F.; Bonnet, G.; Caranta, C.; Gallois, J.L. 2016. A TILLING approach to generate broad-spectrum resistance to potyviruses in tomato is hampered by eIF4E gene redundancy**.** *The Plant Journal* 85: 717-729.

Gonzalez-Cendales, Y.; Catanzariti, A.M.; Baker, B.; Mcgrath, D.J.; Jones, D.A. 2016. Identification of *I-7* expands the repertoire of

genes for resistance to *Fusarium* wilt in tomato to three resistance gene classes. *Molecular Plant Pathology* 17: 448-463.

Grattidge, R.; Obrien, R.G. 1982. Occurrence of a 3rd race of *Fusarium*-wilt of tomatoes in Queensland. *Plant Disease* 66: 165-166.

Grigolli, J.F.J.; Kubota, M.M.; Alves, D.P.; Rodrigues, G.B.; Cardoso, C.R.; Silva, D.J.H.; Mizubuti, E.S.G. 2011. Characterization of tomato accessions for resistance to early blight. *Crop Breeding and Applied Biotechnology* 11: 174-180.

Ho, J.-Y.; Weide, R.; Ma, H.M.; Wordragen, M.F.; Lambert, K.N.; Koornneef, M.; Zabel, P.; Williamson, V.M. 1992. The root-knot nematode resistance gene (*Mi*) in tomato: Construction of a molecular linkage map and identification of dominant cDNA markers in resistant genotypes. *The Plant Journal* 2: 971–982.

Jabeen, N.; Chaudhary, Z.; Gulfraz, M.; Rashid, H.; Mirza, B. 2015. Expression of rice chitinase gene in genetically engineered tomato confers enhanced resistance to fusarium wilt and early blight. *Plant Pathology Journal* 31: 252-258.

Jablonska, B.; Ammiraju, J.S.; Bhattarai, K.K.; Mantelin, S.; Martinez De Ilarduya, O.; Roberts, P.A.; Kaloshian, I. 2007. The Mi-9 gene from *Solanum arcanum* conferring heat-stable resistance to root-knot nematodes is a homolog of *Mi*-1. *Plant Physiology* 143: 1044-1054.

Jones, J.T.; Haegemen, A.; Danchin, E.G.J.; Gaur, H.S.; Helder, J.; Jones, M.G.K.; Kikuchi, T.; Palomares-Rius, J.E.; Wesemael, W.M.L.; Perry, R.N. 2013. Top 10 plant-parasitic nematodes in molecular plant pathology. *Molecular Plant Pathology* 14: 946-961.

Kemmitt, G. 2002. Early blight of potato and tomato. *The Plant Health Instructor* Doi: 10.1094/PHI-I-2002-0809-01.

Klarfeld, S.; Rubin, A.; Cohen, Y. 2009. Pathogenic Fitness of Oosporic Progeny Isolates of *Phytophthora infestans* on Late-Blight-Resistant Tomato Lines. *Plant Disease* 93: 947-953.

Kumar, S.; Srivastava, K. 2013. Screening of tomato genotypes against early blight (*Alternaria solani*) under field condition. *The Bioscan* 8: 189-193.

Laurindo, B.S.; Laurindo, R.D.F.; Azevedo, A.M.; Nick, C.; Silva. D.J.H.; Mizubuti, E.S.G. 2015. Selection of tomato accessions resistant to early blight by cluster analysis of disease progress curves. *Pesquisa Agropecuária Brasileira* 50: 106-114.

Laurindo, B.S.; Laurindo, R.D.F.; Nick, C.; Carneiro, P.C.S.; Mizubuti, E.S.G.; Silva, D.J.H. 2016. Potencial de hibridação entre acessos de tomateiro para pre-melhoramento quanto à resistência à requeima. *Pesquisa Agropecuária Brasileira* 51: 27-34.

Li, G.; Huang, S.; Guo, S.; Li, Y.; Yang, Y.; Guo, Z.; *et al*. 2011. Cloning and characterization of r3b; members of the r3 superfamily of late blight resistance genes show sequence and functional divergence. *Molecular plant-microbe interactions* 24: 1132-1142.

Li, X.Q.; Wei, J.Z.; Tan, A.; Aroian, R.V. 2007. Resistance to root-knot nematode in tomato roots expressing a nematicidal *Bacillus thuringiensis* crystal protein. Plant Biotechnology Journal 5: 455-464.

Lima, M.A.; Maffia, L.A.; Barreto, R.W.; Mizubuti, E.S.G. 2009. *Phytophthora infestans* in a subtropical region: survival on tomato debris, temporal dynamics of airborne sporangia and alternative hosts. *Plant Pathology* 58: 87-99.

Lourenço Jr., V.; Moya, A.; Gonzáles-Candelas, F.; Carbone, I.; Maffia, L.A.; Mizubuti, E.S.G. 2009. Molecular diversity and evolutionary processes of *Alternaria solani* in Brazil inferred using genealogical and coalescent approaches. *Phytopathology* 99: 765-774.

Ma, L.-J.; Geiser, D.M.; Proctor, R.H.; Rooney, A.P.; O'Donnell, K.; Trail, F.; Gardiner, D.M.; Manners, J.M.; Kazan, K. 2013. *Fusarium* Pathogenomics. *Annual Review Microbiology* 67: 399-416.

Maciel-Zambolim, E.; Costa, H.; Capucho, A.; Ávila, A.C.; Inoue-Nagata, A.K.; Kitagima, E. 2004. Surto epidemiológico do vírus do mosaico amarelo do pimentão em tomateiro na região serrana do Espírito Santo. *Fitopatologia Brasileira* 29: 325-327.

Mcgovern, R. J. 2015. Management of tomato diseases caused by *Fusarium oxysporum*. *Crop Protection* 73: 78-92.

Nakaho, K.; Seo, S.; Ookawa, K.; Inoue, Y.; Ando, S.; Kanayama, Y.; Miyashita, S.; Takahashi, H. 2017. Involvement of a vascular hypersensitive response in quantitative resistance to *Ralstonia solanacearum* on tomato rootstock cultivar LS-89. *Plant Pathology* 66: 150-158.

National Institute of Food and Agriculture. Variability, Adaptation and Management of Nematodes Impacting Crop Production and Trade; University of California. 2019. (https://portal.nifa.usda.gov/web/crisprojectpages/1018832-variability-adaptation-and-management-of-nematodes-impacting-crop-production-and-trade.html). Accessed on 05/03/2020.

Nick, C.; Laurindo, B.S.; Almeida, V.S.; Laurindo, R.D.F.; Aguilera, J.G.; Silva, E.C.F.; Cruz, D.C.; Silva, D.J.H. 2013. Seleção simultânea para qualidade do fruto e resistência à requeima em progênies de tomateiro. *Pesquisa Agropecuária Brasileira* 48: 59-65.

Nowicki, M.; Foolad, M.R.; Nowakowska, M.; Kozik, E.U. 2012. Potato and tomato late blight caused by *Phytophthora infestans*: an overview of pathology and resistance breeding. *Plant Disease* 96: 4-17.

Nowicki, M.; Kozik, E.U.; Foolad, M. 2013. Late Blight of Tomato. *Translational Genomics of Crop Breeding* 1: 241-265.

Ohlson, E.W.; Foolad, M.R. 2016. Genetic analysis of resistance to tomato late blight in *Solanum pimpinellifolium* accession PI 163245. *Plant Breeding* 398: 391-398.

Panthee, D.R.; Gardner, R.G. 2010. 'Mountain Merit': a late blight-resistant large-fruited tomato hybrid. *HortScience* 45: 1547-1548.

Park, T.-H.; Vleeshowers, V.G.A.A.; Hutten, R.C.B.; van Eck, H.J.; van der Vossen, E.; Jacobsen, E.; Visser, R.G.F. 2005. High-resolution mapping and analysis of the resistance locus rpi-abpt against *Phytophthora infestans* in potato. *Molecular Breeding* 16: 33-43.

Potnis, N.; Timilsina, S.; Strayer, A.; Shantharaj, D.; Jeri, D.; Barak, J.D.; Paret, M.L.; Vallad, G.E.; Jones, J.B. 2015. Bacterial spot of tomato and pepper: diverse *Xanthomonas* species with a wide variety of virulence factors posing a worldwide challenge. *Molecular Plant Pathology* 16: 907-920.

Pinheiro, J. B.; Pereira, R. B.; Suinaga, F. A. 2014. *Manejo de nematoides na cultura do tomate*. Embrapa Circular Técnica.

Reis, A.; Giordano, L.B.; Lopes, C.A.; Boiteux, L. 2004. Novel sources of multiple resistance to three races of *Fusarium oxysporum* f. sp. *lycopersici* in *Lycopersicon* germplasm. *Crop Breeding and Applied Biotechnology* 4: 495-502.

Rodrigues, T.T.M.S.; Berbee, M.L.; Simmons, E.G.; Cardoso, C.R.; Reis, A.; Maffia, L.A.; Mizubuti, E.S.G. 2010. First report of *Alternaria tomatophila* and *A. grandis* causing early blight on tomato and potato in Brazil. *New disease Reports* 22: 28.

Rosa, J.M.O; Westerich, J.N.; Wilcken, S.R.S. 2014. Reação de genótipos e híbridos de tomateiro à *Meloidogyne enterolobii*. *Ciência Rural* 44: 1166-1171.

Rotem, J. 1994. *The genus Alternaria biology, epidemiology, and pathogenicity*. APS Press St. Paul 326p.

Ruffel, S.; Gallois, J.L.; Lesage, M.L.; Caranta C. 2005. The recessive potyvirus resistance gene *pot-1* is the tomato orthologue of the pepper *pvr2-eIF4E* gene. *Mol Genet Genomics* 274: 346–353.

Salustiano, M.E.; Vale, F.X.R.; Zambolim, L.; Fontes, P.C.R. 2006. Tomato blight management in low temperature periods. *Summa Phytopathologica* 32: 353-359.

Santos Júnior, W.N.; Carvalho, M.R.M.; Cabral, C.S.; Reis, A. 2009. Selection of tomato hybrids and acessions for resistance to *Fusarium oxysporum* f. sp. *lycopersici* race 3. *Boletim de Pesquisa e Desenvolvimento*. Embrapa Hortaliças 19p.

Santos. J.R.M. 1997. Methodology for screening tomato for *Fusarium* wilt. *Verticillium* wilt. gray leaf spot. Early blight and Septoria leaf blight. In: International conference on the processing tomato; International symposium on tropical tomato diseases, Recife. *Proceedings*... Alexandria: ASHS: IPA 164-166.

Simmons, E.G. 2000. Alternaria themes and variations (244-286) species on Solanaceae. *Mycotaxon* 75: 1-115.

Smart, C.D.; Tanksley, S.D.; Mayton, H.; Fry, W.E. 2007. Resistance to *Phytophthora infestans* in *Lycopersicon pennellii*. *Plant Disease* 91: 1045-1049.

Souza, N.M.; Blind, A.D.; Silva Filho, D.F.; Rodrigues, H.S.; Noda, H. 2013. Avaliação de linhagens e cultivares de tomate resistentes à murcha bacteriana (*Rasltonia solanacearum*) desenvolvidas na amazonia. *Enciclopédia Biosfera Centro Científico Conhecer* 9: 400-410.

Thapa, S.P.; Miyao, E.M.; Davis, R.M.; Coaker, G. 2015. Identification of QTLs controlling resistance to *Pseudomonas syringae* pv. *tomato* race 1 strains from the wild tomato, *Solanum habrochaites* LA1777. *Theorical and Applied Genetics* 128:681-692.

Upadhyay, P.; Ganie, S.H.; Rai, A.; Singh, M.; Sinha, B. 2016. Identification of transcription factors in tomato, potentially related to early blight resistance at invasion in host tissue, using microarray expression profiling. *South African Journal of Botany* 106: 165-173.

Wang, Y.; Yang, W.; Zhang, W.; Han, Q.; Feng, M.; Shen, H. 2013. Mapping of a heat-stable gene for resistance to southern root-knot nematode in *Solanum lycopersicum*. *Plant Molecular Biology Reports* 31: 352-362.

Wittmann, J.; Brancato, C.; Berendzen, K.W.; Dreiseikelmann, B. 2016. Development of a tomato plant resistant to *Clavibacter michiganensis* using the endolysin gene of bacteriophage CMP1 as a transgene. *Plant Pathology* 65: 496-502.

Woudenberg, J.H.C.; Truter, M.; Groenewald, J.Z.; Crous, P.W. 2014. Large-spored *Alternaria* pathogens in section Porri disentangled. *Studies in Mycology* 79: 1-47.

Young, R.A; Kelly, J.D. 1996. Characterization of genetic resistence to *Colletotrochum lindemuthianum* in common bean differential cultivars. *Plant Disease* 80: 650-654.

Zerbini, F.; Maciel-Zambolim, E. 1999. A família Potyviridae–Parte I. *Revisão Anual de Patologia de Plantas* 7: 1-67.

To cite this paper:

De Almeida, G. Q., Silva, J. O., Copati, M. G. F., Dias, F. O., Santos, M. C. (2020). Tomato breeding for disease resistance. *Multi-Science Journal*, 3(3): 8-16. DOI: https://doi.org/10.33837/msj.v3i3.1287