**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

**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. lineariae 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 (RGC3) 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
an occur anywhere in the plant, with pH (less than 2008; Nowicki 2016). National strategies of magic, Mountain Merit, Defiant, sported to aptive scale (Santos 2016) identified in the so genes of the S. pimpinellifolium (2013). All genes were identified in wild programs, since many of the late blast resistance QTLs have yet been successfully implemented in breeding Ohlson and St. Clair 2004; Ohlson and St. Clair improvement. The management of late blight is 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 2009). 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. arcuana and S. habrochaites (Dianese et al. 2011). The introgression/inheritance 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 (ToCMoV) 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. habrochaites 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. habrochaites 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

De Almeida et al. (2020)
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 CMPI 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 °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 °C (Jablonska 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 transgenically. 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 thuringiensis (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. corneliiomuelleri 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 pyramiding 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.
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.; Wordragn, M.F.; Lambert, K.N.; Koormneef, 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. Screening of tomato genotypes resistant to early blight by cluster analysis of disease progress curves. *Pesquisa Agropecuária Brasileira* 50: 106-114.

Jones, J.T.; Haegemen, A.; Danchin, E.G.J.; Gaur, H.S.; Helder, J.; Jabeen, N.; Chaudhary, Z.; Gulfraz, M.; Rashid, H.; Mirza, B. 2015. 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, G.; Huang, S.; Guo, S.; Li, Y.; Yang, Y.; Guo, Z.; et al. 2011. Cloning and characterization of the. *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 nematodical 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.; González-Candelas, F.; Carbone, L.; 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.

Maclie-Zambolim, E.; Costa, H.; Capucho, A.; Avila, A.C.; Inoue-Nagata, A.K.; Kitagawa, E. 2004. Surto epidemiológico do vírus do mosaco 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.
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 (Rasitonia 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. *Theoretical 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.; Groenevald, 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 resistance to Colletotrichum 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](https://doi.org/10.33837/msj.v3i3.1287)