

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,100

Open access books available

149,000

International authors and editors

185M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Chapter

Host-Pathogen and Pest Interactions: Virus, Nematode, Viroid, Bacteria, and Pests in Tomato Cultivation

Refik Bozbuga, Songul Yalcin Ates, Pakize Gok Guler, Hatice Nilufer Yildiz, Pinar Aridici Kara, Bekir Bulent Arpaci and Mustafa Imren

Abstract

Several pathogens and pests damage tomato plants, and only one and/or more pathogens and pests can coexist in the same plant at the same time. As several numerous pathogens are found in the same plant, the damage to the tomato plants is higher. Pathogens such as nematodes, viruses, viroids, bacteria, and insects adversely affect the growth and development of tomato plants. They may infect roots or upper part of the plant and can cause not only slow down the growth of plants, but also crop losses and their death. Damaging of plant caused by pathogens and pests reduces the market value of plant products. Those pathogens and pests are also called biotic stress agents. The damage, mode of infection, and the mechanism of infection in each tomato plant and pathogens might be different. This situation is crucially important to understand plant pathogen relationship in detail in terms of controlling pests and pathogen. The effect of each pest/pathogen on tomato plants during the cultivation, the type of damage, and new developments and perspectives on morphological and molecular aspects in tomato-pathogen interactions will be discussed in this chapter.

Keywords: nematode, viroid, bacteria, virus, insects, pathogens, resistance, pest, biotic stress

1. Introduction

Tomato (*Solanum lycopersicum* L.), member of the family Solanaceae, is a cultivated plant with a very large cultivation area in the world. According to 2021 FAO data, the amount of tomatoes produced in the worldwide is 187 million tonnes. The highest production amount is in China, followed by Turkey in the third place [1]. Tomato (*S. lycopersicum* L., family Solanaceae) is one of the most produced crops worldwide, and Turkey is placed in top five countries in terms of the production of Solanaceae family [2, 3]. At least 12% of the world's agricultural products are lost every year due to plant

diseases caused by some pathogenic microorganisms and 20% due to some insect pests. Disease factors, pest organisms, and weeds in agricultural products can cause significant economic losses and damage. If the necessary controls against these factors are not made, crop losses can reach from 35% to 100%. The 60–75% of the diseases observed in plants are caused by fungal and bacterial diseases, 10–15% by viral disease (virus and viroids), and 10% by other pathogens and some environmental stress factors [4].

Viruses are commonly encountered in the living ecosystem. Since it does not have a complete cellular structure, it interacts with prokaryotic and eukaryotic organisms and maintains its own existence [5, 6]. In recent years, plant viruses and their mechanisms of action have been widely studied due to the loss of agricultural products and their effects on fruit-vegetable quality. Plant viruses have either single-stranded RNA (ssRNA) or double-stranded RNA or DNA nucleotides [7].

Nematodes are one of the most abundant multicellular organisms on the earth. They may live as plant and animal parasites and/or free living. Parasitic nematodes may infect humans, plants, and animals [8]. Among nematodes, about 4100 nematode species have been identified as plant-parasitic nematodes [8]. They cause significant crop losses on tomato plants.

Bacterial, viroid diseases, and insect pests give also significant crop losses affecting tomato production in many regions in the world.

In this chapter, the effect of each pest/pathogen (virus, nematode, viroid, bacteria, and pests) on tomato plants during the cultivation, the type of damage, and new developments and perspectives on morphological and molecular aspects in tomato-pathogen interactions are given.

2. Viruses disease

Tomato viruses are transmitted by vector insects, plant material, and seeds [9]. Transmission of tomato viruses is important to determine the plant material used in the diagnosis, to choose the method of diagnosis, to prevent the spread of the virus, and to develop a method of struggle against the virus. In this part, we examine under two subtitles that some viral diseases, the main host of which is tomato, are transmitted only by plant materials including seeds and are transmitted by vector insects and/or plant material together. In addition, in this section, the general information and classification of viruses, their genetic characteristics, symptoms and damage in tomato plants, and preventing against the viruses have been briefly explained.

2.1 Tomato viruses transmitted by plant parts including seeds

2.1.1 *Tomato brown rugose fruit virus*

Tomato brown rugose fruit virus (ToBRFV) was first reported in tomato in Jordan [10]. ToBRFV belongs the family Virgaviridae and genus *Tobamovirus*, has rod-shaped particles with encapsidating a positive-sense single-stranded RNA (ssRNA) [11, 12]. ToBRFV is basically transmitted by mechanical ways as plant-plant contact, workers, tools, equipment, and irrigation water. The virus is also effectively transmitted by seeds [10]. In addition, bumblebees transmit the virus on tomatoes [13]. The virus has severe symptoms as mosaic blotch, narrowing on leaves and brown rugose, yellowing spots on fruits. Moreover, the virus reduces the quality of the fruit and causes the

fruit to be unmarketable [14]. ToBRFV is detected by enzyme-linked immunosorbent analysis (ELISA), polymerase chain reaction (PCR)-based analysis by specific primers, and genome sequencing, NGS (next-generation sequencing) [10, 14–16].

2.1.2 Pepino mosaic virus

Pepino mosaic virus (PepMV) was originally identified in pepino (*Solanum muricatum*) in Peru, in 1974 [17]. Following pepino, the virus was firstly detected in tomato, in Netherlands [18]. PepMV belongs to the family Flexiviridae and genus *Potexvirus*, has a positive-sense ssRNA genome with non-enveloped, flexible, rod-shaped particles [17]. Although PepMV isolates show a high genomic similarity, they differ from the original source isolate that causes disease in tomato [9]. Observing leaf symptoms are yellow and mosaic spots, scorching, and deformations [9]. The common transmission way of PepMV is mechanical basis such as plant sap, contaminated tools, and surfaces [9]. The virus has been also transmitted by recirculating hydroponic system, bumblebees, and the root-infecting fungus *Olpidium virulentus* between tomato plants [19–21]. In addition, conventional polymerase chain reaction (PCR), quantitative PCR (qPCR) methods as TaqMan assays and restriction fragment length polymorphism (RFLP) are also have been used for detection of virus and identification of different genotypes [19, 22].

2.1.3 Tobacco mosaic virus

Tobacco mosaic virus (TMV) was the first virus detected [23], belongs the family Virgaviridae and genus *Tobamovirus* [24, 25]. TMV has rod-shaped and encapsulating particles with a single-stranded RNA (ssRNA) [26–28]. The first viral protein structure sequenced belongs to TMV [29, 30]. TMV is transmitted by mechanically including workers, tools, and propagating materials [31]. Because the virus has oldest genomic information, it has widespread host plants including tomato [32]. TMV has characteristic symptoms on the leaves such as light and dark green spots and malformation. Moreover, TMV infections have also caused necrotic rings, browning, and number and size reducing on fruits [33]. In addition to the serological analysis method for TMV, numerous molecular detection methods and diagnostic studies have been carried out [34]. In general, virus-free seeds, plantlets, and hygienic measures have to be used to prevent from virus like other tobamoviruses.

2.1.4 Tomato mosaic virus

Tomato mosaic virus (ToMV) belongs the family Virgaviridae and genus *Tobamovirus* [12, 35]. The particles of virus are rod-shaped and encapsulating with a genome single-stranded RNA (ssRNA) [26]. ToMV has high rate of infectivity, effective seed transmission, and mechanic transmission easily by working hands, tools, soil, and plants parts [12, 36]. Like as other tobamoviruses, ToMV causes malformation, spotting and clearing on tomato leaves, and malformation on fruit and reducing the yields [36]. As with other tobamoviruses, virus-free seeds, plantlets, and hygiene measures should generally be used to prevent the virus.

2.1.5 Tomato mottle mosaic virus

Tomato mottle mosaic virus (ToMMV) was firstly identified in Mexico in 2013, belongs the family Virgaviridae and genus *Tobamovirus*, has four open reading frames

(ORFs) including the movement protein (MP) and coat protein (CP) in genome [37]. As other tobamoviruses, ToMMV is inclined to mechanical transmission including contacts, hands, tools, the greenhouse structure, and bumblebees. Moreover, seed transmission is also possible with infected seeds [12]. ToMMV causes the mosaic symptoms, chlorosis, and leaf deformation on tomato plants [38]. The virus can be detected by using polymerase chain reaction (PCR) based methods [39]. Management of the ToMMV is possible by using virus-free seeds and plantlets and using hygienic measures [40].

3. Plant-parasitic nematodes

Plant-parasitic nematodes are significant pests and cause crop losses, with an estimated yearly loss of USD 173 billion [41]. It is likely that 10% of world crop production is lost as a result of plant-parasitic nematode damage [42]. Most of the plant-parasitic nematodes feed on roots and decrease the uptake of water and nutrients [43]. Stylets of the plant-parasitic nematodes are important apparatus used to puncture plant cells and uptaking nutrient contents. The main signs shown by plants affected by nematodes are stunted development, wilting, and susceptibility to contamination by other plant pathogens [44]. Although there are many plant-parasitic nematodes, the most vital plant-parasitic nematodes in the USA are *Heterodera glycines*, *Meloidogyne fallax*, *Meloidogyne chitwoodi*, *Globodera pallida*, *Ditylenchus dipsaci*, *Litylenchus crenatae*, *Globodera rostochiensis*, *Meloidogyne enterolobii*, *Pratylenchus fallax* and *Bursaphelenchus xylophilus* [45]. Similarly, *Meloidogyne* spp., *Aphelenchoides besseyi*, *Nacobbus aberrans*, *Pratylenchus* spp., *B. xylophilus*, *Heterodera* and *Globodera* spp, *Xiphinema index*, *Radopholus similis*, *D. dipsaci*, and *Rotylenchulus reniformis* are most important nematodes in terms of plant pathology [46]. Root-knot nematodes: The nematodes belonging to the *Meloidogyne* genus termed root-knot nematodes are polyphagous plant pathogens [47]. They may be found worldwide and parasitize the species of higher plants [47]. Root-knot nematodes, *Meloidogyne* genus, which are obligate plant parasites, are economically important and damage plants. They are found in many parts of the world and have the ability to parasitize any high plants [47]. They disrupt plant physiology and decrease crop quality and yield [9, 48]. Root-knot nematodes have 106 species [47]. *M. hapla*, *M. incognita*, *M. arenaria*, and *M. javanica* are major species; however, *M. fallax*, *M. minor*, *M. chitwoodi*, *M. exigua*, *M. paranaensis*, and *M. enterolobii* (= *M. mayaguensis*) are minor root-knot nematode species [41].

The genus of *Meloidogyne* comprises more than 100 species in the world [46]. Root-knot nematodes are named because of their characteristic features, as they typically cause root galls. While young plants may not survive high infection by a nematode, mature plants often show low yield and growth retardation. Among the root-knot nematodes, *M. graminicola* may cause damage to cereals in South Africa, the USA, Australia, and Mexico [44]. *M. arenaria*, *M. incognita*, and *M. javanica* are good hosts of some cereal cultivars such as rye, barley, oat, and wheat under greenhouse conditions [49]. *M. hapla* is distributed in temperate regions, and yield losses caused by some root-knot nematode species are valued at approximately \$10 billion [50]. Root-knot nematodes cause damage and induce a unique feeding site structure termed giant cells within the plant roots. Cell wall molecular architecture of nematode feeding site is changed [51]. *M. javanica*, *M. arenaria*, *M. graminicola*, *M. incognita*, and *M. hapla* are some of the most damaging species; some species cause more damage to their host than other species. For instance, *M. graminicola* is one of the main



Figure 1.
*The root-knot nematode, *M. incognita*, induced root galls in tomato plants (left) and control-uninfected healthy tomato plant roots (right). The nematode cause galls in tomato roots (right).*

problems in rice fields that develop special hook-like knots on the roots of rice plant roots [52]. Root-knot nematodes induce feeding cells and become sedentary within approximately 48 hours after nematode infection [53]. The second stage juveniles of root-knot nematodes can infect the plant roots. More than one species of root-knot nematodes in the same plant tissues can be found. The nematode causes galls in the root system (**Figure 1**), disrupts the vascular tissues, and restricts the exchange of water and nutrients. Growth slows down, wilting, stunting, and yellowing of leaves are seen. During a severe infection, the plant may completely dry out. The secondary damage of root-knot nematodes is that soil-borne pathogens may enter nematode-induced wounds in plants [54].

4. Tomato pests and their control

4.1 *Tuta absoluta* (Meyrick) (Lepidoptera: Gelechiidae)

Tuta absoluta is the main pest in open field and greenhouse tomato cultivation. Adult butterflies are active at night. They lay their eggs, usually under the leaves, in the lower part of the sepals of buds and immature fruits. Its larvae damage all parts of the tomato plant except the root and in each period. The larva feeds by opening

galleries between the two epidermes on the leaves of the tomato. The plant may dry out completely due to the galleries opened in the green part of the plant. The pest enters under the sepals of immature tomato fruits. The damaged fruit loses its market value, and rots occur when secondary microorganisms settle in the galleries opened in the fruit [55]. As a biotechnical method, pheromone + water trap or pheromone + light + water trap can be used in greenhouse tomato cultivation for mass trapping against tomato moth [55].

4.2 *Bemisia tabaci* (Genn.) and *Trialeurodes vaporariorum* (Westw.) (Hemiptera: Aleyrodidae)

The damage of these pests is important in tomatoes, cucumbers, peppers, beans, and eggplant [56]. Whitefly adults use the underside of leaves for feeding, laying eggs and resting. Larvae and adults feed by sucking plant sap. As a result of suction, yellowing occurs in the form of spots on the leaf. In addition, the pest secretes a sweet substance during feeding, with the development of fumagine fungi on this substance, a black layer forms on the leaves, and these parts cannot assimilate. For this reason, the plant weakens, plant growth is adversely affected, yield and quality decrease. Whiteflies give an average of 9–10 offspring per year, depending on the temperature, and a female lays an average of 200-300 eggs. Whitefly adults also play an important role in the transmission of some viral diseases. Especially *Tomato yellow leaf curl virus* (TYLCV) is carried by Tobacco whitefly [55].

4.3 *Liriomyza trifolii* (Burgess), *L. bryoniae* (Kalt.), *L. huidobrensis* (Blanchard) (Dip.: Agromyzidae)]

Especially tomato, cucumber, and beans are among the important hosts of leaf fly, which is a polyphagous pest. Adults and larvae of the pest cause damage to the plant. Adults lay their eggs between the two epidermes of the leaf [55]. Larvae emerging from the egg feed on the parenchyma tissue between the two epidermes in the leaf, and as a result, galleries are formed. In the following periods, these areas turn yellow, dry, and fall off. It indirectly causes loss of product and value by delaying development in young seedlings and plants [55]. A female can lay about 400 eggs in her lifetime at 30°C. It can give about 10 offspring under greenhouse conditions. In order to obtain healthy plants in the cultural struggle, precautions should be taken against pests, especially during the seedling period, For this purpose, ventilation openings must be covered with gauze. Weeds around and inside the greenhouse must be destroyed. Contaminated plant residues must be destroyed. The soil must be kept moist and the pupae must rot from moisture by mulching, and larvae should be prevented from becoming pupae by passing into the soil. Entry-exit and ventilation openings in greenhouses should be covered with gauze or fine-hole wire to prevent the entry of adults. Yellow sticky traps are used in biotechnical control since planting seedlings. One of the most important parasitoids is *Diglyphus isaea* Walker (Hym.: Eulophidae). In case of 10 larvae per leaf in tomato, chemical control is decided [55].

4.4 Aphids [*Myzus persicae* (Sulz.), *Aphis gossypii* Glov., *A. fabae* Scop., *Macrosiphum euphorbiae* (Thomas) (Hem.: Aphididae)]

Aphids are particularly damaging to tomatoes, peppers, eggplants, cucumbers, and zucchini. Aphids cause damage by sucking plant sap. Due to the suction, the

leaves take a shrivelled, curled appearance. As a result of this suction, the plant weakens, development stops, the yield and quality of the product deteriorate. The sweet substances they secrete cover the plant surface by causing fumagine, and damage occurs as a result of the plant's obstruction to assimilation and respiration. It is also the vector of viral diseases. It is known that only *M. persicae* is the vector of 50 different viruses [55]. Contaminated plants and weeds should be cleaned from inside the greenhouse. Among the predators, especially the species belonging to the Coccinellidae, Chrysopidae, and Syrphidae families and the parasitoids *Aphidius* species are very important in terms of biological control. For chemical control against Aphids in tomato, it is decided to apply if 20 individuals are seen per leaf [55].

4.5 *Tetranychus urticae* Koch. (Acarina: Tetranychidae)

As a polyphagous pest, *T. urticae* is particularly damaging to tomatoes, beans, cucumbers, eggplant, peppers, and zucchini [83]. The females lay their eggs on the underside of the leaves, between the webs they weave along the leaf veins. The larva that emerges from the egg becomes adult by passing the protonymph and deutonymph stages. Larvae change three shirts until they reach adulthood [55]. A female can lay 100–200 eggs. Depending on the climatic conditions and the host, it can produce 10–12 offspring per year in greenhouses [56]. As a cultural precaution in the fight against spider mites, plant residues contaminated with the pest should be removed from the environment. Soil cultivation should be done, and weeds should be combated. In its biological control, especially Phytoseids, Coccinellids, and predatory thrips are the first preferred natural enemies. If five nymphs + adults per leaf are determined in chemical control against spider mites in tomato, the application is decided [55].

4.6 Thrips [*Thrips tabaci* Lind., *Frankliniella occidentalis* Pergande. (Thys.: Thripidae)]

Thrips particularly give damage to tomatoes, cucumbers, peppers, eggplants, and beans. Adults and larvae injure the epidermis layer of leaves, stems, and fruits of plants and feed by absorbing the sap. The cells in the area where the thripsin is fed die and white silvery spots appear. As a result, the assimilation capacity of the leaves decreases and the leaf edges curl. As a result of feeding on fruit or capsules, silvery spots appear, and deformities occur. *T. tabaci* lay 70–100 eggs during their lifetime. It completes one offspring in an average of 14–30 days. It gives 3–10 offspring per year. *F. occidentalis* lays 150–300 eggs during its lifetime. It gives a maximum of 15 offspring per year. As a cultural precaution, plant residues contaminated with pest should be destroyed. Of the natural enemies, especially *Orius* spp., it is important for biological control. In the chemical control of thrips, if 20 nymphs per leaf or three nymphs + adults (adults-larvae) are determined per flower, the application is decided [55].

5. Tomato bacterial diseases

5.1 Bacterial canker *Clavibacter michiganensis* subsp. *michiganensis* (Smith)

Clavibacter michiganensis subsp. *michiganensis* (CMM) is a xylem-inhabiting bacteria [57]. Optimal growth conditions are at 24–38°C and 7 and 8 P. But it found to grow



Figure 2.

Vascular color change of tomato plant by *Clavibacter michiganensis* subsp. *michiganensis* (CMM). The bacteria inhabit in the xylem. The color of the plant vascular tissues is cream-yellow to brown.

in plant xylem at pH 5 [57, 58]. The disease is seed-borne, and bacteria may survive in or on the seed coat. Contaminated soil equipment and other materials serve as inoculum sources for short periods. Infected plant materials and soils with infected plant debris are important inoculum sources by providing long life periods of bacteria. After the plant is infected, bacteria invade xylem vessels, and it moves systemically throughout a plant. Disease causes weak and stunted plants. Infected seedlings may be quickly collapsed. Bacterial canker caused vascular (systemic) and parenchymal (superficial) symptoms. The early symptoms are wilting, curling browning, and wilting of the leaves, especially along one side of the plant. Wilting of the lower leaves can be seen toward the flowering stage. The wilting may progress upward of the plant. The wilted parts can dry out in a short time. As a result of the superficial infections, necrotic or slightly raised spots may appear on the surfaces of leaves, on the stems, and on petioles. In infected plant, cream-yellow to brown coloring of the vascular tissues can be seen (**Figure 2**).

5.2 Bacterial pith necrosis

Bacterial pith necrosis disease is caused by several pathogenic bacteria, *Pseudomonas corrugata* (Scarlett et al.) Roberts and Scarlett, *P. cichorii* (Swingle) Stapp, *P. mediterranea* Catara et al., *P. viridiflava* (Burkholder) Dowson, *P. fluorescens*, *Pseudomonas marginalis* Brown (Stewens), *Dickeya chrysanthemi*, *Pectobacterium carotovorum* subsp. *carotovorum* [59–61]. The disease affects tomato plants (*S. lycopersicum*), especially in greenhouse production. The disease was first described in Britain in 1970 by Scarlett et al. [62]. Disease-causing agents are generally opportunistic bacteria to cause disease when the plant is under stressful conditions. High humidity, high N fertilizer, and low night temperatures encourage rapid plant growth, and the formation of the juicy structure is a disease favorable condition [63]. The major entry place for bacteria is the wounds caused after secondary sprout removal, which is a common practice in staked tomato fields. Disease agents generally survived in seeds, soil, and infected plant debris for 6–8 months [64]. The disease may occur in



Figure 3.
Bacterial pith necrosis: general wilting and stem necrosis by tomato pith necrosis and stem necrosis and vascular coloring of tomato plants caused by tomato pith necrosis. The brown discoloration is seen.

the field and covered greenhouse crops, especially during winter in greenhouse crops. The symptoms are similar to the infections caused by the pathogens *P. viridiflava*, *P. corrugata*, *P. mediterranea*, *P. carotovorum*, or *Pectobacterium atrosepticum* [65–67]. Typical symptoms of pith necrosis on tomato plants consisted of general plant wilting, yellowing, and brown to black spots or lesions developing on the stem, petiole, and fruit stalk (**Figure 3**). Internally, pith tissues developed water-soaking, brown discoloration, hollowing, and soft rotting. In some cases, browning also occurs in the vascular tissues (**Figure 3**).

5.3 Bacterial speck disease *Pseudomonas syringae* pv. *tomato* (Okabe) Young, Dye, Wilkie

Bacterial speck of tomato is a serious problem in many greenhouse and field production areas. Disease can occur at every growing stage of tomato, but it causes severe infections at cool, moist conditions. The optimal growth temperature of the bacteria is 24–30°C. Disease development stops in hot weather conditions. The disease is ubiquitous [68], Bacteria can survive epiphytically on weed hosts [69]. Bacteria can maintain the viability for 1–2 years as saprophytically on diseased plant residues in the soil [70].

The disease is seed-borne. Infection may begin with soil with contaminated seeds or plant debris. Secondary contamination occurs from wounds or natural openings. Water droplets play an important role in the spread of the disease. During the seedling period, brown-black spots sometimes surrounded by chlorotic margin are seen on the leaves and stems of the seedlings, and sometimes these spots spread and cause drying of the seedling. The spots on the leaves are small, round, dark in color, and unlimited. A yellow halo is usually seen around these spots, which are 1–3 mm in diameter. The spots coalesce over time and form large necrotic areas that lead to deformation and drying of the leaf. Superficial large brown spots are seen on the main stem and branches, leaves, and flower stalks (**Figure 4**) [71].



Figure 4.

The symptoms of bacterial speck disease *P. syringae* pv. *Tomato* (*Pst*). Large spots on tomato stems (left), flower spots (middle), spots on fruit stalks and fruits (right) by *Pst*.

5.4 Bacterial spot of tomato *Xanthomonas vesicatoria* Vauterin et al., *Xanthomonas euvesicatoria* (Jones et al.); *Xanthomonas perforans* (Jones et al.).

Bacterial spot of tomato is a worldwide disease. *X. vesicatoria* Vauterin et al., *X. euvesicatoria* *X. perforans* have been identified to cause bacterial spot disease on tomato. The disease was firstly discovered in South Africa in 1914 [72]. High relative humidity and overhead irrigation are optimal conditions for disease development. The optimum growth temperature of these bacteria is 29°C. 20–35°C temperature conditions promote disease development, while night temperatures lower than 16°C suppress disease development. Infected seeds may serve as a major inoculum source. The agent can survive on or in the seed for a year or more. *Xanthomonads* may also survive epiphytically in the tomato phyllosphere. Under favorable conditions, epiphytic populations can cause severe infections or outbreaks, especially in transplants [73]. Tomato bacterial spot caused necrotic lesions on the leaves, stems, petals, and flowers, and fruit [74]. Circular water-soaked lesions appear on seedlings (Figure 5). They later dry and turn dark brown to black [75]. Sometimes, halos are present around the spots. Primary lesions coalesce, resulting in extensive necrosis and a blighted appearance (Figure 5).

5.5 Bacterial wilt of tomato *Ralstonia solanacearum*

Bacterial wilt (BW) is the most important disease affecting tomato production in many regions [76]. It causes severe wilting of economically important crops such as tomato, potato, eggplant, chili, and non-solanaceous crops such as banana and groundnut. *R. solanacearum* is an aerobic obligate organism. It was classified as four races and five biovars. Race 1 has a very wide host range mainly flowering crops. Race 2 attacks bananas, race 3 has worldwide effects on tomatoes, potatoes, and other *Solanaceae* plants, and race 4 infects ginger [77]. *R. solanacearum* can survive on weeds and alternative non-host plants epiphytically. Infected soil and crop residues may serve as important inoculum sources [78]. The pathogen is carried in tomato seeds [79].

Initial symptom of bacteria in tomato is wilting of upper leaves (Figure 6). Complete wilting of the plants is observed in a short time. Brown discoloration of the infected vascular tissues and visible white or yellowish bacterial ooze can be seen [80].



Figure 5.
The symptoms of bacterial spot of tomato *Xanthomonas* spp. Water-soaked lesions of the disease on seedlings (left), leaf spots of *X. euvesicatoria* in greenhouse grown tomatoes (right). N. YILDIZ.



Figure 6.
The symptoms of BW of tomato *R. solanacearum*. Wilting caused by *R. solanacearum* is seen on the leaves of tomato plant.

6. Tomato viroids

Some viroids are pathogenic, some can continue to multiply asymptotically in susceptible plant species. Viroids are classified in two families, Avsunviroidae

and Pospiviroidae. It has been reported that there are eight species in the family Pospiviroidae, which cause symptoms to occur intensely in tomatoes, especially in the Solanaceae family [81].

Common symptoms of viroid infection depending on viroid species and variant (species and strain), variety, temperature, and light conditions include chlorosis, tanning, leaf deformation, reduced plant growth, severe yield loss, and non-marketable fruit symptoms in tomato plants [82].

6.1 Potato spindle tuber viroid

The genus *Pospiviroid* of the family Pospiviroidae; *Potato gothic virus*, *Potato spindle tuber pospiviroid* (PSTVd), *Potato spindle tuber virus*, *Tomato bunchy top viroid* has been named under different names. The PSTVd factor is included in the EPPO A2 list. PSTVd was the first to be identified as a new viroid and is quite different from bacteria and viruses [83]. PSTVd is located in the family Pospiviroidae of the *Pospiviroid* genus [84]. While the main host is potato (*Solanum tuberosum* and other *Solanum* spp.), tomato (*S. lycopersicum*), pepper (*Capsicum* spp.), and other vegetables and ornamental plants and weeds from the Solanaceae family also constitute the host series. Infections in ornamental plants and weeds are generally asymptomatic. It has been determined that many species in the Solanaceae family and a few species in other families can be transmitted experimentally [85].

The type and severity of PSTVd symptoms vary depending on the viroid strain, host species and variety, and environmental conditions. PSTVd infections can be asymptomatic or produce symptoms ranging from mild to severe. PSTVd may cause more severe symptoms at higher temperatures [86]. In tomato, early in infection, infected plants show slow growth and chlorosis in the upper part of the plant, while in advanced stages the growth reduction may become more severe and leaves may turn red and/or purple and become more fragile (Figure 7). At this stage, flowering and fruiting may stop. In advanced stages, plants may die or partially recover.

6.2 *Citrus exocortis* viroid (*Citrus exocortis pospiviroid*) (Indian tomato bunchy top viroid)

The disease agent was observed for the first time with the symptom of bark scaling on the three-leaf rootstock of citrus fruits, and it was revealed that it was transmitted



Figure 7. *Potato spindle tuber pospiviroid* (PSTVd) induced plant symptoms on tomato plants. PSTVd symptoms of tomato plant (*Money maker* cv. (left) and *H5656* cv. Standard cultivar (right)). Control plant represents the uninfected plants.

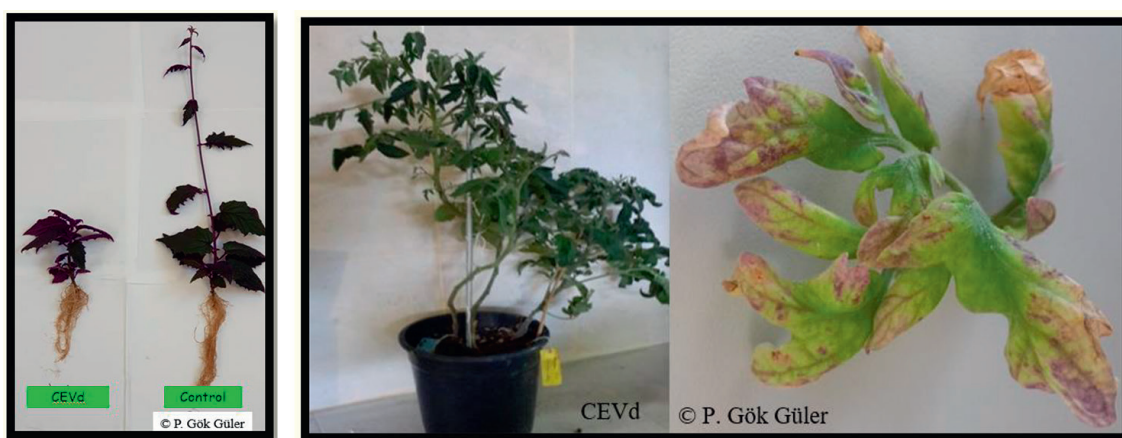


Figure 8.
The symptoms of CEVd (*Citrus exocortis pospiviroid*) (*Indian tomato bunchy top viroid*). CEVd symptoms of *Gynura aurantiaca* indicator plant and tomato plant (H5656 cv. Standart cultivar). Control plant represents the uninfected plants.

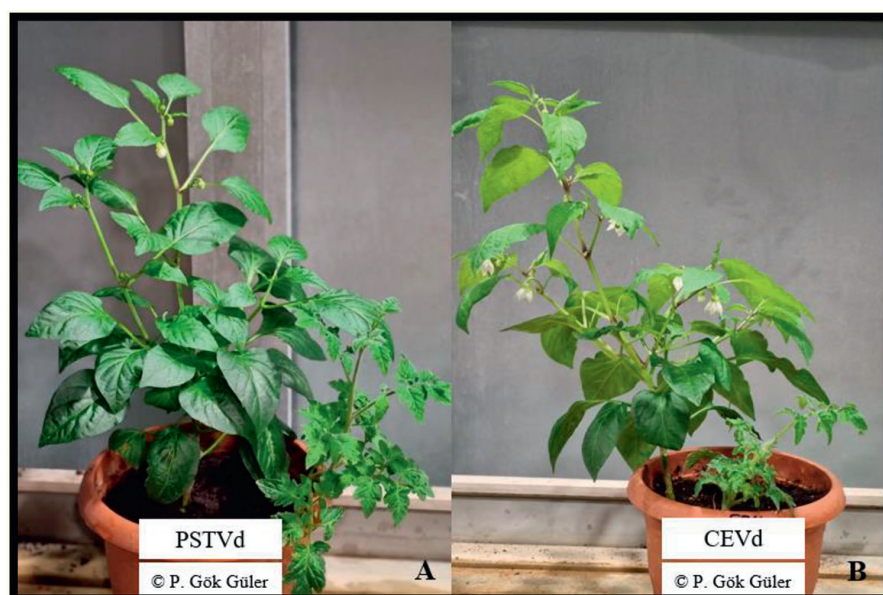


Figure 9.
The symptoms of PSTVd (A) and CEVd (B) in *S. lycopersicum* L. (*Hünkar* cv.) and *C. annuum* L. (*Sunam F1* cv.) plants.

by the bud [87]. In 1972, this factor was determined to be a viroid [88]. The agent is classified as a *Citrus exocortis viroid* (CEVd) species in the *Pospiviroid* genus of the family Pospiviroidae. CEVd is one of the best characterized viroids today. Exocortis disease is called citrus dwarfing viroid disease in our country. CEVd can cause scaling in the bark tissue of citrus trees, peeling and general stunting of the plant [89, 90]. Decreased growth, stunting may occur, chlorosis in leaves may become more severe, turning into reddening, bruising, and/or necrosis (Figures 8 and 9).

6.3 *Columnnea latent viroid*

Columnnea latent viroid (CLVd) agent was first detected in the *Columnnea erythrophaea* plant in the US state of Maryland in 1989, and it was stated that the agent was present asymptotically in this plant [91] but it was later determined that it

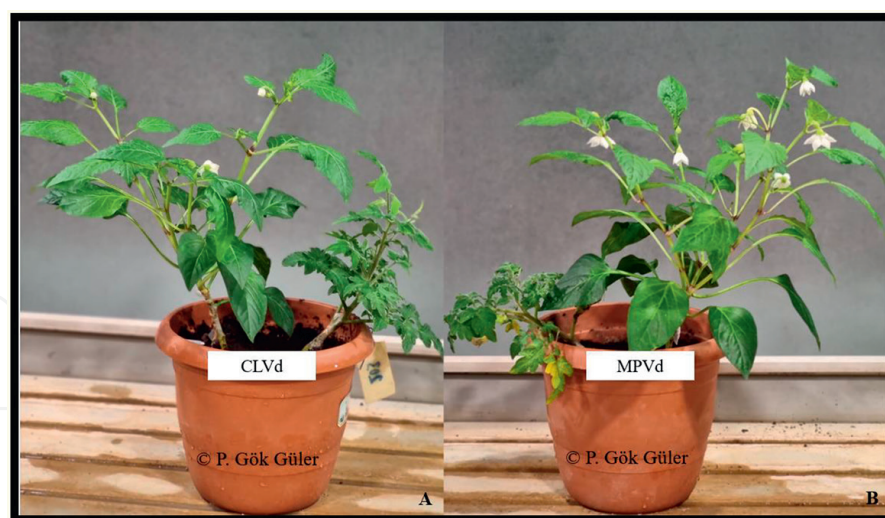


Figure 10. The symptoms of CLVd in pepper plants. CLVd (A) and Mexican papita viroid (MPVd) (B) symptoms of *S. lycopersicum* L. (Hünkar cv.) and *C. annuum* L. (Sunam F1 cv.) plant.

produced PSTVd-like symptoms in potatoes and tomatoes [92]. The agent is *Brunfelsia* spp., *Columnea* spp., *Gloxina* spp. and *Nematanthus* species are generally asymptomatic (latent) in ornamental plants [93, 94]. Both PSTVd and MPVd were found naturally in wild *Solanum* species [95]. In tomatoes, CLVd can cause general stunting, deterioration of leaf structure, formation of thin-stemmed plants, tanning of leaves, chlorosis and leaf epinasticity, as well as necrosis of leaves, stems, and petioles (**Figure 10A**).

6.4 Mexican papita viroid

The MPVd agent was first identified in 1996 in the plant *Solanum cardiophyllum*, a wild *Solanum* species in Mexico [95]. The symptom caused by MPVd in plants is observed as a general stunting and the formation of chlorotic and purplish spots on the leaves (**Figure 10B**). Depending on the severity of the infection, the fruit size decreases and/or no fruit is formed. There are uncertainties about how the agent is transported. The sequence of MPVd was determined to be very similar to that of TPMVd (93%) and PSTVd [95].

6.5 Tomato apical stunt

Tomato apical stunt (TASVd) causes severe symptoms in tomato plants shortening of the internodes, leaf deformation and yellowing, shrinkage, and less coloration of fruits (**Figure 11A**). TASVd has been reported in the Ivory Coast, Tunisia [96], Senegal [97]. TASVd has also been detected asymptotically in some ornamental plants (e.g., *Brugmansia*, *Cestrum*, *Solanum jasminoides*, *S. rantonetii*, *Streptosolen jamesonii*). TASVd is transported by seed, by plant sap during mechanical processes (during pruning, etc.). While it is not carried by pests such as *M. persicae* and *B. tabaci*, it is carried with pollen with the help of bumblebees during pollination. There is insufficient data on the geographical distribution, host range and epidemiology of TASVd, and control of viroids is difficult in practice [98].

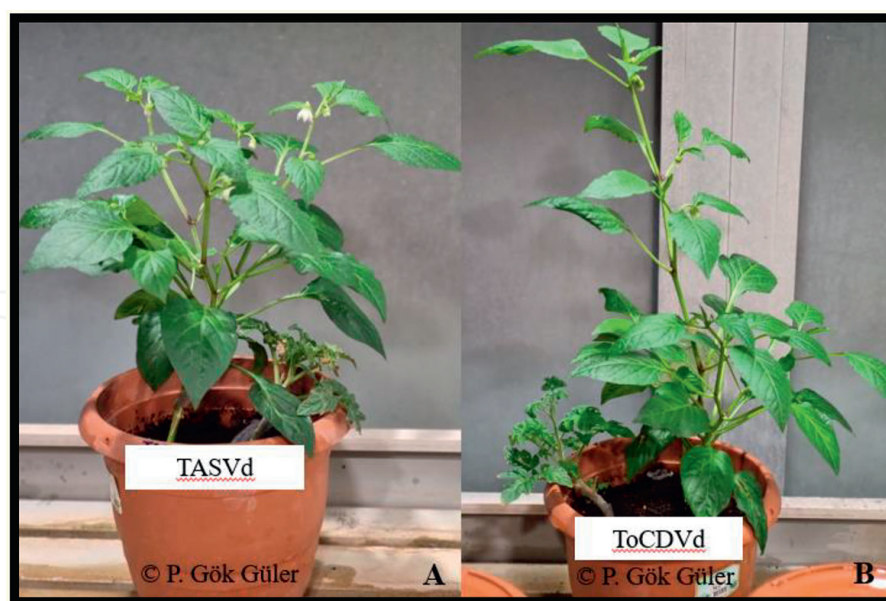


Figure 11.
The symptoms of TASVd in plants. TASVd (A) and ToCDVd (B) symptoms of *S. lycopersicum* L. (Hünkar cv.) and *C. annuum* L. (Sunam F1 cv.) plant.

6.6 Tomato chlorotic dwarf viroid

Tomato chlorotic dwarf viroid (TCDVd) agent was first detected in 1996 in a tomato greenhouse in Manitoba, Canada [99]. As the hosts of the agent; *Brugmansia* spp. and hybrids, *Petunia* spp., *Solanum melongena*, *Verbena* spp., and *Vinca minor* plants have been reported. The agent has been found in Arizona and Hawaii [100, 101], India [102], Slovenia [103]. It has caused disease in tomatoes grown in greenhouses in [104]. General stunting, curling of leaves, chlorosis that may turn bronze or purple in later periods (Figure 11B), necrosis in petioles and veins, leaf epinasticity, apical bunching, small It causes losses in total yield with the appearance of cracked fruit formations [105].

6.7 Tomato planta macho viroid

Tomato planta macho viroid (TPMVd) agent was first detected in the tomato state of Morelos, Mexico, in 1982 [106]. Seven species in the Solanaceae family have been reported as natural hosts of TPMVd to date. Since the fruits of the infected plants are in the size of balls and they are completely unmarketable, great commercial losses have been experienced. Although this factor was initially thought to be a viral disease, it was later determined to have a viroid etiology [107–109]. In infected tomato plants, the first symptoms begin 10–15 days after the infection as growth cessation. Chlorosis, epinasty, wrinkling, wrinkling are seen on the leaves and the leaves become brittle. Later, the leaves shrink and turn yellow and stand upright. Although excessive and early fruit formation is seen, the fruits remain small. No seeds are formed in the fruit or fruits with very few seeds are formed. In general, severe stunting is observed in the plant and the fruits may lose their market value. The main symptom occurring within the cells is necrosis caused by the collapse of the phloem [106]. TPMVd affects plant growth (Figure 12). It has been reported that the agent can be transmitted mechanically and by the vector *M. persicae*, but there is no conclusive evidence of seed transmission [110].



Figure 12. The effect of TPMVd on plants. TPMVd (A (60 days), B (21 days)) symptoms of *S. lycopersicum* L. (Hünkar cv.) and *C. annuum* L. (Sunam F1 cv.) plant.

7. Plant resistance to pathogens

Many devastating diseases widely distribute throughout the world in tomato-growing areas and tomato hosts more than 200 species of pests and pathogens [111]. Bacterial canker caused by seed-borne organism *Clavibacter michiganensis* subsp. *michiganensis* (CMM) is a destructive disease in both field and protected cultivation of tomato crops. *S. hirsutum*, *S. peruvianum*, *S. pimpinellifolium*, and *S. chilense* are the wild relatives to improve resistance source of *S. lycopersicum* [112–115]. Inheritance of the resistance was controlled by four-gene model [116]. Inheritance of the CMM resistance in wild relatives has been explained by at least four genes [117] and quantitative trait loci (QTL) associated with resistance in interspecific cross [118]. Two major loci Rcm 2.0 and Rcm 5.1 introgressed from LA407 (*S. hirsutum*) have been identified on second and fifth chromosome and explained epistatically 68% of the variation [119].

Whitefly transmitted tomato yellow leaf curl virus (TYLCV Genus *Begomovirus*, Family Geminiviridae) has been threatened to tomato production throughout the temperate regions of the world since 1930s [120]. TYLCV and/or TYLCV-like viruses have many strains and genomic recombinants causing similar symptoms [121]. TYLCV-resistant tomato breeding program was initiated in Israel where first symptoms were observed in the world [122]. TY-20 has been improved as the first hybrid variety resistant to TYLCV from *S. peruvianum* (line M-60) and *S. lycopersicum* (line 10) [123]. Cucumber mosaic virus (CMV) has been divided into subgroups (I and II) and generates stunting, filiform leaves, and necrosis. A single dominant resistance gene Cmr derived from chromosome 12 of *S. chilense* accession (LA458) contributes complete or partial resistance to cultivars [124]. Potato virus Y (PVY) and tobacco etch virus (TEV) are two of main viruses belonging potyviridae transmitted by many species of aphids infect to tomato plants. The recessive gene pot-1 sourced from PI 247087 contributes resistance by single recessive genes both TEV and PVY [125, 126]. ToMV and TMV are named synonymously vice versa. Three dominant resistance genes Tm-1, Tm2, and Tm22 are used to improve resistant varieties derived from PI 235673 (*S. lycopersicum*) [127], PI 126926 (*S. peruvianum*) [128], and PI 128650 (*S. peruvianum*) [129], respectively. *S. peruvianum* is the wild relative used as genetic resource for resistance to *Meloidogyne* spp. Resistance is conferred by a single eight

Mi-1 to Mi-8 dominant gene located on chromosome 6 and 12, controls *M. incognita*, *M. arenaria*, and *M. javanica* [130]. Resistance sources to *Meloidogyne* spp. are PI128657 (Mi or Mi-1), PI270435-2R2 (Mi-2) PI126443-1MH (Mi-3), LA1708-1 (Mi-4) PI126443-1MH (Mi-5), PI270435-3MH (Mi-6 and Mi-7) PI270435-2R2 (Mi-8). Mi-3, Mi-7 and Mi-8 genes confer resistance to virulent strain *M. incognita* 557R. Nematode resistance is heat-sensitive in tomato. Mi-4, Mi-5, and Mi-6 genes contribute resistance over 30°C. LA2884 (*S. chilense*) line has heat stable resistance [131]. Potato spindle tuber viroid (PSTVd), tomato chlorotic dwarf viroid, citrus exocortis viroid, Columnea latent viroid, TASVd, tomato planta macho viroid (including Mexican papita viroid), and pepper chat fruit viroid have been identified as causal agents of pospiviroids in tomato. There is no commercial variety resisting to pospiviroids [132]. Potato spindle tuber viroid (PSTVd) causes yield loss, plant stunting, leaf chlorosis, smaller fruits. It is one of the most prevalent viroid species attacked to tomato plants. Four accessions belonging *S. chilense* and *S. habrochaites* have been reported less than 50% of PSTVd infection [133]. *S. pimpinellifolium* (LA0373, LA0411) and *S. chmielewskii* (LA1028) plants reported highly tolerant to PSTVd [134].

8. Conclusions

Plant-pathogens and pests are significantly important and cause an immense amount of crop losses worldwide. Plant-parasitic nematodes, insects, bacteria, viroid, and viruses damage crops at a high rate. Some groups of those diseases and pests parasitize the specific host plant, while others are polyphagous. Identification of plant-parasitic nematodes, insects, bacteria, viroid, and viruses and determination of the parasitism mode of action are important in terms of controlling pests and disease. Plant pathogens and pests show very different symptoms in plants, for example, root knot nematodes cause galls, bacteria cause color changes in plant stems and roots, viruses and viroids cause color changes and deformities in plants. The species of some insects that cause not only their own damage, but also secondary damages due to the fact that some of them carry viruses (for instance *M. persicae* is the vector of numerous viruses). Therefore, in order to grow disease-free plants, it has to be protected of healthy plants from plant-pathogens and pests. In controlling diseases and pests, it is important to have a deep understanding of the host-parasite interactions using cutting-edge technology and techniques. It is also crucially significant for future studies to fully understand host parasite interactions at morphological, molecular, and genetics level.

Conflict of interest

The authors declare no conflict of interest.

IntechOpen

Author details

Refik Bozbuga^{1*}, Songul Yalcin Ates^{2†}, Pakize Gok Guler^{3†}, Hatice Nilufer Yildiz^{3†}, Pinar Aridici Kara^{3†}, Bekir Bulent Arpaci^{4†} and Mustafa Imren^{5†}

1 Faculty of Agriculture, Department of Plant Protection, Eskisehir Osmangazi University, Eskisehir, Turkey

2 İzmir Directorate of Agricultural Quarantine, İzmir, Turkey

3 Biological Control Research Institute, Yuregir, Adana, Turkey


4 Faculty of Agriculture, Department of Horticulture, Cukurova University, Saricam, Adana, Turkey

5 Faculty of Agriculture, Department of Plant Protection, Abant Izzet Baysal University, Bolu, Turkey

*Address all correspondence to: refik.bozbuga@ogu.edu.tr

† Authors contributed equally to this work. Plant parasitic nematodes (by R Bozbuga and M Imren), insects (by PA Kara), viroid diseases (by PG Guler), bacterial diseases (by HN Yildiz), plant resistance to pathogens (by BB Arpaci), virus diseases (by SY Ates) in tomato plants are written in this book chapter.

IntechOpen

© 2022 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] FAOSTAT. Statistics Division of the Food and Agricultural Organization of the United Nations. 2020. Available from: <https://www.fao.org/faostat/en/#data/QCL>. [Accessed 16 May 2022]
- [2] Abak K, Düzyaman F, Şeniz V, Gülen H, Pekşen A, ve Kaymak HÇ. Sebze Üretimini Geliştirme Yöntem ve Hedefleri. VII. Ziraat Kongresi, 11-15 Ocak 2010, Ankara; 2010, 477-492 s
- [3] Hammond RW. Economic significance of viroids in vegetable and field crops. In: Hadidi A, Flores R, Palukaitis P, Randles J, editors. Viroids and Satellites. Cambridge, MA: Academic Press; 2017. DOI: 10.1016/B978-0-12-801498-1.00001-2
- [4] Agrios GN. Control of plant diseases. In: Plant Pathology. 4th ed. San Diego: Academic Press; 1997. pp. 200-216
- [5] Roossinck MJ. Lifestyles of plant viruses. Philosophical Transactions of the Royal Society Biological Sciences. 2010;365:1899-1905
- [6] Scholthof K-BG. Making a virus visible: Francis O. Holmes and a biological assay for tobacco mosaic virus. Journal of the History of Biology. 2014;47:107-145
- [7] Wang M-B, Masuta C, Smith NA, Shimura H. RNA silencing and plant viral disease. American Phytopathological Society. 2012;25:1275-1285
- [8] Decraeamer W, Hunt DJ. Structure and classification. In: Perry RN, Moens M, editors. Plant Nematology. Wallingford, Oxfordshire, UK: CABI Publishing; 2006. pp. 4-32
- [9] Choi H, Jo Y, Cho WK, Yu J, Tran P-T, Salaipeh L, et al. Identification of viruses and viroids infecting tomato and pepper plants in Vietnam by metatranscriptomics. International Journal of Molecular Sciences. 2020;21(20):7565. DOI: 10.3390/ijms21207565
- [10] Salem N, Mansour A, Ciuffo M, Falk B, Turina M. A new tobamovirus infecting tomato crops in Jordan. Archives of Virology. 2016;161:503-506
- [11] Oladokun JO, Halabi MH, Barua P, Nath PD. Tomato brown rugose fruit disease: Current distribution, knowledge and future prospects. Plant Pathology. 2019;68:1579-1586. DOI: 10.1111/ppa.13096
- [12] Dombrovsky A, Smith E. Seed transmission of tobamoviruses aspects of global disease distribution. In: Jimenez-Lopez JC, editor. Advances in Seed Biology. London, UK: IntechOpen; 2017. pp. 233-260
- [13] Levitzky N, Smith E, Lachman O, Luria N, Mizrahi Y, Bakelman H. The bumblebee *Bombus terrestris* carries a primary inoculum of tomato brown rugose fruit virus contributing to disease spread in tomatoes. PLoS One. 2019;14(1):e0210871. DOI: 10.1371/journal.pone.0210871
- [14] Luria N, Smith E, Reingold V, et al. A new Israeli tobamovirus isolate infects tomato plants harboring Tm-22 resistance genes. PLoS One. 2017;12:e0170429
- [15] Alkowni R, Alabdallah O, Fadda Z. Molecular identification of tomato brown rugose fruit virus in tomato in Palestine. Journal of Plant Pathology. 2019;101(3):719-723
- [16] Menzel W, Knierim D, Winter S, Hamacher J, Heupel M. First report

of tomato brown rugose fruit virus infecting tomato in Germany. *New Disease Reports*. 2019;**39**:1

[17] Jones RAC, Koenig R, Lesemann D-E. Pepino mosaic virus, a new potexvirus from pepino (*Solanum muricatum*). *Annals of Applied Biology*. 1980;**94**:61

[18] Van der Vlugt RAA, Stijger CCMM, Verhoeven JTJ, Lesemann DE. First report of Pepino mosaic virus on tomato. *Plant Disease*. 2000;**84**:103

[19] Alfaro-Fernández A, Córdoba-Sellés MC, Herrera-Vásquez JA, Cebrián MC, Jordá C. Transmission of Pepino mosaic virus by the fungal vector *Olpidium virulentus*. *Journal of Phytopathology*. 2010;**158**:217-226

[20] Schwarz D, Paschek U, Bandte M, Büttner C, Obermeier C. Detection, spread, and interactions of Pepino mosaic virus and *Pythium aphanidermatum* in the root environment of tomato in hydroponics. *Acta Horticulturae*. 2009;**808**:163-170

[21] Shipp JL, Buitenhuis R, Stobbs L, Wang K, Kim WS, Ferguson G. Vectoring of Pepino mosaic virus by bumble-bees in tomato greenhouses. *The Annals of Applied Biology*. 2008;**53**:149-155

[22] Gutiérrez-Aguirre I, Mehle N, Delic D, Gruden K, Mumford R, Ravnkar M. Real time quantitative PCR based sensitive detection and strain discrimination of Pepino mosaic virus. *Journal of Virological Methods*. 2009;**162**:46-55

[23] Scholthof K-BG. Tobacco mosaic virus. *The Plant Health Instructor*. 2000. DOI: 10.1094/PHI-I-2000-1010-01. Updated 2005. Available from: <https://www.apsnet.org/edcenter/disandpath/viral/pdlessons/Pages/TobaccoMosaic.aspx>

[24] Carstens EB. Ratification vote on taxonomic proposals to the International Committee on Taxonomy of Viruses (2009). *Archives of Virology*. 2010;**155**:133-146

[25] Lewandowski DJ. Genus *Tobamovirus*. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, editors. *Virus Taxonomy*. 8th Report of the International Committee on Taxonomy of Viruses. San Diego, USA: Elsevier/Academic Press; 2005. pp. 1009-1011

[26] Dawson WO, Beck DL, Knorr DA, Grantham GL. cDNA cloning of the complete genome of tobacco mosaic virus and production of infectious transcripts. *Proceedings of the National Academy of Sciences of the United States of America*. 1986;**83**:1832-1836

[27] Goelet P, Lomonosoff GP, Butler PJG, Akam ME, Gait MJ, Karn J. Nucleotide sequence of tobacco mosaic virus RNA. *Proceedings of the National Academy of Sciences of the United States of America*. 1982;**79**:5818-5822

[28] Green SK, Sulyo Y, Lesemann DE. Leaf curl virus on tomato in Taiwan province. *FAO Plant Protection Bulletin*. 1987;**35**:62

[29] Anderer FA, Uhlig H, Weber E, Schramm G. Primary structure of the protein of tobacco mosaic virus. *Nature*. 1960;**186**:922-925

[30] Tsugita A, Gish DT, Young J, Fraenkel Conrat H, Knight CA, Stanley WM. The complete amino acid sequence of the protein of tobacco mosaic virus. *Proceedings of the National Academy of Sciences of the United States of America*. 1960;**46**:1463-1469

[31] Mirones E, Luis-Arteaga M. Viral diseases. In: *Integrated Pest and Disease*

- Management in Greenhouse Crops. R. Albajes, M. L. Gullino, J. C. van Lenteren, and Y. Elad, editors. Kluwer Academic Publishers, Dordrecht, Netherlands; 1999. pp. 16-33
- [32] Creager AN, Scholthof KB, Citovsky V, Scholthof HB. Tobacco mosaic virus. Pioneering research for a century. *The Plant Cell*. 1999;**11**:301-308
- [33] Caciagli P. Vegetable viruses. In: BWJ Mahy and MHV van Regenmortel, editors. *Encyclopedia of Virology*, 3rd ed. Oxford: Academic Press; 2008. pp. 2860
- [34] Conacalla NC, Nitin M, Kaldis A, Masarapu H, Carpentier S, Voloudakis A. dsRNA molecules from the tobacco mosaic virus p126 gene counteract TMV-induced proteome changes at an early stage of infection. *Frontiers in Plant Science*. 2021;**12**. DOI: 10.3389/fpls.2021.663707
- [35] Broadbent L. Epidemiology and control of tomato mosaic-virus. *Annual Review of Phytopathology*. 1976;**14**:75-96
- [36] Petrov N, Stoyanova M, Gaur RK. Plant virus-host interaction: molecular approaches and viral evolution. In: *Ecological methods to control viral damages in tomatoes*. 2nd ed. 2021. pp. 469-488. Chapter 19. DOI: 10.1016/B978-0-12-821629-3.00005-1
- [37] Li R, Gao S, Fei Z, Ling K-S. Complete genome sequence of a new tobamovirus naturally infecting tomatoes in Mexico. *Genome Announcements*. 2013;**1**:e00794-e00713
- [38] Webster CG, Roskopf EN, Lucas L, Mellinger HC, Adkins S. First report of tomato mottle mosaic virus infecting tomato in the United States. *Plant Health Progress*. 2014;**15**:151-152
- [39] Ambrós S, Martínez F, Ivars P, Hernández C, de la Iglesia F, Elena SF. Molecular and biological characterization of an isolate of tomato mottle mosaic virus (ToMMV) infecting tomato and other experimental hosts in Eastern Spain. *European Journal of Plant Pathology*. 2017;**149**:261-268
- [40] Liqin T, Shuhua W, Gao D, Liu Y, Zhu Y, Ji Y. Synthesis and characterization of a full-length infectious cDNA clone of tomato mottle mosaic virus. *Viruses*. 2021;**13**:1050. DOI: 10.3390/v13061050
- [41] Elling AA. Major emerging problems with minor *Meloidogyne* species. *Phytopathology*. 2013;**103**:1092-1102. DOI: 10.1094/PHYTO-01-13-0019-RVW
- [42] Whitehead AG. *Plant Nematode Control*. Wallingford, UK: CABI; 1998
- [43] Mojtahedi H, Balali G, Akhiani A, et al. Tylenchorhynchid nematodes of Iran (Tylenchoidea: Nematoda). *Iranian Journal of Plant Pathology*. 1983;**19**:11-14
- [44] Williamson MW, Hussey RS. Nematode pathogenesis and resistance in plants. *The Plant Cell*. 1996;**8**(10):1735-1745. DOI: 10.1105/tpc.8.10.1735
- [45] Kantor M, Handoo Z, Kantor C, Carta L. Top ten most important U.S.-regulated and emerging plant-parasitic nematodes. *Horticulturae*. 2022;**8**(3):208. DOI: 10.3390/horticulturae8030208
- [46] Jones JT, Haegeman A, Danchin EG, Gaur HS, Helder J, Jones MG, et al. Top 10 plant-parasitic nematodes in molecular plant pathology. *Molecular Plant Pathology*. 2013;**14**(9):946-961. DOI: 10.1111/mpp.12057
- [47] Karssen G, Moens M. *Root-knot Nematodes*, Plant Nematology. Wallingford, Oxfordshire, UK: CABI Publishing; 2006. pp. 59-90

- [48] Eisenback JD, Triantaphyllou HH. Root-knot nematodes: meloidogyne species and races. Manual of Agricultural Nematology. 1991;1:191-274
- [49] Johnson AW, Motsinger RE. Suitability of small grains as hosts of *Meloidogyne* species. Journal of Nematology. 1989;21(4S):650
- [50] Chitwood DJ. Research on plant-parasitic nematode biology conducted by the United States Department of Agriculture–Agricultural Research Service. Pest Management Science. 2003;59(6-7):748-753
- [51] Bozbuga R, Lilley JL, Knox JP, Urwin PE. Host-specific signatures of the cell wall changes induced by the plant parasitic nematode, *Meloidogyne incognita*. Scientific Reports. 2018;8:17302. DOI: 10.1038/s41598-018-35529-7
- [52] Pankaj Sharma HK, Prasad JS. The rice root knot nematode, *Meloidogyne graminicola*: an emerging problem in rice-wheat cropping system. Indian Journal of Nematology. 2010;40(1):1-11
- [53] Bartlem CJ, Jones MGK, Hammes UZ. Vascularisation and nutrient delivery at root-knot nematode feeding sites in host roots. Journal of Experimental Botany. 2014;65:1789-1798. DOI: 10.1093/jxb/ert415
- [54] Anonymous. Baę Entegre M¼cadele Teknik Talimati (in Turkish). Tarımsal Arařtırmalar ve Politikalar Genel M¼d¼rl¼ę¼, Gıda ve Kontrol Genel M¼d¼rl¼ę¼, 2017. pp. 1-120. Ankara, Turkey
- [55] Anonymous. Ört¼altı Entegre M¼cadele Teknik Talimati (in Turkish). Tarımsal Arařtırmalar ve Politikalar Genel M¼d¼rl¼ę¼, Gıda ve Kontrol Genel M¼d¼rl¼ę¼, 2017. pp. 1-137. Ankara, Turkey
- [56] Anonymous. Available from: https://www.turktob.org.tr/dergi/makaleler/dergi17/TTOB_Dergi17_WEB-60_65.pdf [Accessed: 26 May 2022]
- [57] Cass Smith WP, Goss M. Bacterial canker of tomatoes. Journal of the Department of Agriculture of Western Australia. 1946;23:147-156
- [58] Eichenlaub R, Gartemann KH, Burger A. *Clavibacter michiganensis*, a group of gram-positive phytopathogenic bacteria. In: Plant-Associated Bacteria. The Netherlands: Springer; 2006. pp. 385-421
- [59] Pekhtereva ES, Kornev KP, Matveeva EV, Polityko VA, Budenkov NI, Ignatov AN, et al. Pith necrosis of tomato in Russia. Acta Horticulturae. 2009:251-253. Available from: <http://www.actahort.org>
- [60] Molan YY, Ibrahim YE, Al-Masrahi AA. Identification in Saudi Arabia of *Pseudomonas corrugata*, the tomato pith necrosis pathogen, and assessment of cultivar resistance and seed treatment. Journal of Plant Pathology. 2010;92:213-218
- [61] Baki D. Domates (*Solanum lycopersicum* L.) bakteriyel öz nekrozu hastalık etmenleri *Dickeya chrysanthemi*, *Pectobacterium carotovorum* subsp. *carotovorum*, *Pseudomonas cichorii*, *Pseudomonas corrugata*, *Pseudomonas fluorescens*, *Pseudomonas mediterranea* ve *Pseudomonas viridiflava*'nın LNA probe kullanılarak real-time PCR tanısı ve hastalıklı bitki dokularından tespiti [PhD thesis]. 2014
- [62] Scarlett CMJT, Roberts FP, Lelliott RA. Tomato pith necrosis caused by *Pseudomonas corrugata* sp. Annals of Applied Biology. 1978;88(1):105-114

- [63] Ustun N, Saygili H. Pith necrosis on greenhouse tomatoes in Aegean region of Turkey. In: Proceedings 11th Congress of the Sociedade Portuguesa de Fitopatologica, University of Evora (Portugal), 17-20 September. Portugal. 2001. pp. 70-73
- [64] Yildiz HN, Aysan Y, Sahin F, Cinar O. Potential inoculum sources of tomato stem and pith necrosis caused by *Pseudomonas viridiflava* in the Eastern Mediterranean Region of Turkey/Mögliche Inokulum-Quellen der Stamm-und Gefäßnekrose an Tomaten verursacht durch *Pseudomonas viridiflava* in der östlichen Mittelmeerregion der Türkei. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz/Journal of Plant Diseases and Protection. 2004;**111**:380-387
- [65] Kolomiiets Y, Grygoryuk I, Butsenko L, Bohoslavets V, Blume Y, Yemets A. Identification and biological properties of the pathogen of soft rot of tomatoes in the greenhouse. Open Agriculture Journal. 2020;**14**(1):290-298. DOI: 10.2174/1874331502014010290
- [66] Trantas EA, Sarris PF, Mpalantinaki EE, Pentari MG, Ververidis FN, Goumas DE. A new genomovar of *Pseudomonas cichorii*, a causal agent of tomato pith necrosis. European Journal of Plant Pathology. 2013;**137**(3):477-493
- [67] Wick RL, Shrier R. Tomato pith necrosis caused by *Erwinia chrysanthemi*. Plant Disease. 1990;**74**(8):615. DOI: 10.1094/PD-74-0615F
- [68] Schneider RW, Grogan RG. Bacterial speck of tomato: sources of inoculum and establishment of a resident population. Phytopathology. 1977;**67**(3):388-394
- [69] Jones JB, Pohronezny KL, Stall RE, Jones JP. Survival of *Xanthomonas campestris* pv. *vesicatoria* in Florida on tomato crop residue, weeds, seeds, and volunteer tomato plants. Phytopathology. 1986;**76**(4):430-434
- [70] Devash Y, Bashan Y, Okon Y, Henis Y. Survival of *Pseudomonas tomato* in soil and seeds. Journal of Phytopathology. 1980;**99**:175-185
- [71] Preston GM. *Pseudomonas syringae* pv. *tomato*: the right pathogen, of the right plant, at the right time. Molecular Plant Pathology. 2000;**1**(5):263-275
- [72] Doidge EM. A tomato canker. The Annals of Applied Biology. 1921;**7**:407-430
- [73] McGuire RG, Jones JB, Stanley CD, Cszinsky AA. Epiphytic populations of *Xanthomonas campestris* pv. *vesicatoria* and bacterial spot of tomato as influenced by nitrogen and potassium fertilization. Phytopathology. 1991;**81**:656-660
- [74] Jones JB, Stall RE, Scott JW, Somodi GC, Bouzar H, Hodge NC. A third tomato race of *Xanthomonas campestris* pv. *vesicatoria*. Plant Disease. 1995;**79**:395-398
- [75] Vallad GE, Goodman RM. Systemic acquired resistance and induced systemic resistance in conventional agriculture. Crop Science. 2004;**44**(6):1920-1934
- [76] Opena RT, Hartman GL, Chen JT, Yang CH. Breeding for Bacterial Wilt Resistance in Tropical Tomato. Malaysian Plant Protection Society; 1992. pp. 44-50
- [77] Denny TP, Hayward AC. Ralstonia. 2001:151-174
- [78] Granada GA, Sequeira L. A new selective medium for *Pseudomonas solanacearum*. Plant Disease. 1983;**67**:1084-1088

- [79] Shakya DD. Occurrence of *Pseudomonas solanacearum* in tomato seeds imported into Nepal. Bacterial Wilt ACIAR. 1993;**45**:371-372
- [80] Vanitha SC, Niranjana SR, Mortensen CN, Umesha S. Bacterial wilt of tomato in Karnataka and its management by *Pseudomonas fluorescens*. BioControl. 2009;**54**(5):685-695
- [81] Di Serio F, Owens RA, Li LF, Matoušek J, Pallás V, Randles JW, et al. ICTV virus taxonomy profile: poospiviroidae. Journal of General Virology. 2020. DOI: 10.1099/jgv.0.001543
- [82] Singh RP, Singh M, Boucher A, Owens RA. A mild strain of potato spindle tuber viroid from China is similar to North American isolates. Canadian Journal of Plant Pathology. 1993;**15**:134-138
- [83] Diener TO. Potato spindle tuber "virus": IV. A replicating, low molecular weight RNA. Virology. 1971;**45**:411-428
- [84] Di Serio F, Li S-F, Pallás V, Owens RA, Randles JW, Sano T, et al. Viroid taxonomy. In: Hadidi A, Flores R, Randles JW, Palukaitis P, editors. Viroids and Satellites. Oxford, UK: Academic Press; 2017. pp. 135-146
- [85] Singh RP. Experimental host range of the potato spindle tuber "virus". American Potato Journal. 1973;**50**:111-123
- [86] Harris PS, Browning IA. The effects of temperature and light on the symptom expression and viroid concentration in tomato of a severe strain of potato spindle tuber viroid. Potato Research. 1980;**23**:85-93
- [87] Fawcett HS, Klotz LJ. Exocortis on trifoliolate orange. Citrus Leaves. 1948;**28**:8
- [88] Semancik JS, Weathers LG. Exocortis disease: evidence for new species of infectious low molecular weight RNA in plants. Nature: New Biology. 1972a;**237**:242-244
- [89] Semancik JS, Weathers LG. Exocortis disease: an infectious free-nucleic acid plant virus with unusual properties. Virology. 1972b;**47**:456-566
- [90] EFSA. Pest risk assessment made by France on citrus exocortis viroid (CEVd) considered by France as harmful in French overseas department of reunion. EFSA Journal. 2008;**685**:1-17
- [91] Owens RA, Smith DR, Diener TO. Measurement of viroid sequence homology by hybridization with complementary DNA prepared in vitro. Virology. 1978;**89**:388-394
- [92] Hammond R, Smith DR, Diener TO. Nucleotide sequence and proposed secondary structure of Columnea latent viroid: a natural mosaic of viroid sequences. Nucleic Acids Research. 1989;**17**:10083-10094
- [93] Singh RP, Boucher A, Somerville TH. Detection of potato spindle tuber viroid in the pollen and various parts of potato plant pollinated with viroid-infected pollen. Plant Disease. 1992;**76**:951-953
- [94] Spieker RL. A viroid from Brunfelsia undulata closely related to the Columnea latent viroid. Archives of Virology. 1996;**141**:1823-1832
- [95] Martinez-Soriano JP, Galindo-Alonso J, Maroon JM, Yucel I, Smith DR, Diener TO. Mexican papita viroid: putative ancestor of crop viroids. Proceedings of the National Academy of Sciences of the United States of America. 1996;**93**:9397-9401
- [96] JTJ V, CCC J, Roenhorst JW. First report of tomato apical stunt viroid

- (TASVd) in tomato in Tunisia. Plant Disease. 2006;**90**(4):528
- [97] Candresse T, Marais A, Ollivier F, Verdin E, Blancard D. First report of the presence of tomato apical stunt viroid on tomato in Senegal. Plant Disease. 2007;**91**(3):330
- [98] EPPO. 2017. EPPO Global Database. Available from: <https://gd.eppo.int/>
- [99] Singh RP, Nie X, Singh M. Tomato chlorotic dwarf viroid (TCDVd): an evolutionary link in the origin of pospiviroids. Journal of General Virology. 1999;**80**(11):2823-2828
- [100] Verhoeven JTJ, Jansen CCC, Werkman AW, Roenhorst JW. First report of tomato chlorotic dwarf viroid in *Petunia hybrida* from the United States of America. Plant Disease. 2007;**91**(3):324
- [101] Ling KS, Verhoeven JTJ, Singh RP, Brown JK. First report of tomato chlorotic dwarf viroid in greenhouse tomatoes in Arizona. Plant Disease. 2009;**93**(10):1075
- [102] Singh RP, Dilworth AD, Baranwal VK, Gupta KN. Detection of citrus exocortis viroid, iresine viroid, and tomato chlorotic dwarf viroid in new ornamental host plants in India. Plant Disease. 2006;**90**(11):1457-1457
- [103] Viršček Marn M, Mavrič Pleško I. First report of tomato chlorotic dwarf viroid in *Petunia* spp. in Slovenia. Plant Disease. 2010;**94**(9):1171
- [104] Fox A, Daly M, Nixon T, Brurberg MB, Blystad D, Harju V, et al. First report of tomato chlorotic dwarf viroid (TCDVd) in tomato in Norway and subsequent eradication. New Disease Reports. 2013;**27**:8
- [105] Belalcazar CS, Galindo AJ. Estudio sobre el virus de la “planta macho” del jitomate. Agrociencia. 1974;**18**:79
- [106] Galindo AJ, Rodriguez MR. Rectificación del agente causal de la planta macho del jitomate. Resúmenes VIII Congr. Soc. Mex. Fitopatol. 1978
- [107] Galindo J, Smith DR, Diener TO. Etiology of planta macho, a viroid disease of tomato. Phytopathology. 1982;**72**:49-54
- [108] Hadidi A, Flores R, Randles JW, Semancik JS. Viroids. Collingwood, Australia: CSIRO Publishing; 2003. p. 370
- [109] Galindo J, Lopez M, Aguilar T. Significance of *Myzus persicae* in the spread of tomato planta macho viroid. Fitopatologia Brasileira. 1986;**11**:400-410
- [110] Heuvelink E, editor. Tomatoes. Vol. 27. Wallingford, UK: CABI Publishing; 2018
- [111] Yordanov M, Stamova L. A new source of resistance to *Corynebacterium michiganense* (E.F. Sm) Jensen. Report of the Tomato Genetics Cooperative. 1977;**27**:26
- [112] Lindhout P, Purimahua C, van der Giessen A. New resistance to bacterial canker in the wild tomato species *Lycopersicon peruvianum*. Prophyta. 1987;**41**(5):100-102
- [113] Berry SZ, Madumadu GG, Uddin MR, Coplin DL. Virulence studies and resistance to *Clavibacter michiganensis* ssp. *michiganensis* in tomato germplasm. Horticultural Science. 1989;**24**:362-365
- [114] Francis DM, Kabelka E, Bell J, Franchino B, St. Clair D. Resistance to bacterial canker in tomato (*Lycopersicon hirsutum* LA407) and its progeny derived from crosses to *L. esculentum*. Plant Disease. 2001;**85**:1171-1176

- [115] Elenkov E. Die Selektion von tomaten auf resistenz gegen die bakterienwelke. Internationale Zeitschrift der Landwirtschaft. 1965;594-597
- [116] De Jong J, Honma S. Inheritance of resistance to *Corynebacterium michiganense* in the tomato. Journal of Heredity. 1976;67:79-84
- [117] Thyr BD. Inheritance of resistance to *Corynebacterium michiganense* in tomato. Phytopathology. 1976;66:1116-1119
- [118] Van Heusden AW, Koornneef M, Voorrips RE, Brüggemann W, Pet G, Virelink-van Ginkel R, et al. Three QTLs from *Lycopersicon peruvianum* confer a high level of resistance to *Clavibacter michiganensis* ssp. *michiganensis*. Theoretical and Applied Genetics. 1999;99:1068-1074
- [119] Coaker GL, Francis DM. Mapping, genetic effects, and epistatic interaction of two bacterial canker resistance QTLs from *Lycopersicon hirsutum*. Theoretical and Applied Genetics. 2004;108:1047-1055
- [120] Lefeuvre P, Martin DP, Harkins G, Lemey P, Gray AJ, Meredith S, et al. The spread of tomato yellow leaf curl virus from the Middle East to the world. PLoS Pathogens. 2010;6(10):e1001164
- [121] Czosnek H, editor. Tomato Yellow Leaf Curl Virus Disease: Management, Molecular Biology, Breeding for Resistance. Springer Science & Business Media. Dordrecht: Springer; 2007. DOI: 10.1007/978-1-4020-4769-5_7
- [122] Picó B, Ferriol M, Diez MJ, Nuez F. Developing tomato breeding lines resistant to tomato yellow leaf curl virus. Plant Breeding. 1999;118(6):537-542
- [123] Pilowsky M, Cohen S. Tolerance to tomato yellow leaf curl virus derived from *Lycopersicon peruvianum*. Plant Disease. 1990;74(3):248-250
- [124] Stamova BS, Chetelat RT. Inheritance and genetic mapping of cucumber mosaic virus resistance introgressed from *Lycopersicon chilense* into tomato. Theoretical and Applied Genetics. 2000;101:527-537
- [125] Legnani R, Gognalons P, Selassie KG, Marchoux G, Moretti A, Laterrot H. Identification and characterization of resistance to tobacco etch virus in *Lycopersicon* species. Plant Disease. 1996;80:306-309
- [126] Ruffel S, Moretti A, Morel C, Palloix A, Caranta C. Recessive resistance genes against potyviruses are localized in colinear genomic regions of the tomato (*Lycopersicon* spp.) and pepper (*Capsicum* spp.) genomes. Theoretical and Applied Genetics. 2002;105:855-861
- [127] Pelham J. Resistance in tomato to tobacco mosaic virus. Euphytica. 1966;15(2):258-267
- [128] Laterrot H, Pecaut P. Tm-2: new source. Report of the Tomato Genetics Cooperative. 1969;19:13-14
- [129] Alexander LJ. Host-pathogen dynamics of tobacco mosaic virus on tomato. Phytopathology. 1971;61(61):1-617
- [130] Blancard D. Tomato Diseases. San Diego, CA, USA: Academic Press; 2012. pp. 17-34
- [131] Williamson VM. Root-knot nematode resistance genes in tomato and their potential for future use. Annual Review of Phytopathology. 1998;36(1):277-293

[132] Ling KS. Decontamination measures to prevent mechanical transmission of viroids. In: *Viroids and Satellites*. Oxford, UK: Academic Press; 2017. pp. 437-445

[133] Li R, Ling KS. Screening tomato germplasm for resistance to potato spindle tuber viroid. In: *Meeting Abstract*. 2014. p. 8. Available from: <https://www.ars.usda.gov/research/publications/publication/?seqNo115=306499>

[134] Naoi T, Hataya T. Tolerance even to lethal strain of potato spindle tuber viroid found in wild tomato species can be introduced by crossing. *Plants*. 2021;**10**(3):575