

Review

A Review of the Most Common and Economically Important Diseases That Undermine the Cultivation of Tomato Crop in the Mediterranean Basin

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Abstract: Tomato (*Solanum lycopersicum* L.), family *Solanaceae*, has become in the past fifty years one of the most important and extensively grown horticultural crops in the Mediterranean region and throughout the world. In 2019, more than 180 million tonnes of tomato have been produced worldwide, out of which around 42 million tonnes in Mediterranean countries. Due to its genetic properties, tomato is afflicted by numerous plant diseases induced by fungal, bacterial, phytoplasma, virus, and viroid pathogens. Not only is its genetic inheritance of great importance to the management of the numerous tomato pathogens, but equally as important are also the present climate changes, the recently revised phytopathological control measures, and the globalization of the seed industry. Thus, the recognition of symptoms and the knowledge of the distribution and spread of the disease and of the methods for early detection of the pathogens are the major prerequisites for a successful management of the disease. In this review, we will describe the main tomato pathogens in the Mediterranean area that impact mostly the tomato yield and provide the current and perspective measures necessary for their successful management.

Keywords: tomato diseases; fungi; bacteria; phytoplasmas; viruses; viroids; detection; crop management

1. Introduction

Tomato (*Solanum lycopersicum* L.), family *Solanaceae*, originated in the Andean region of South America, is the second most cultivated vegetable crop throughout the world following the potato, with approximately 181 million tonnes from 5 Mha, according to the Food and Agriculture Organization Statistics (FAOSTAT) [1]. In Southern Europe, it ranks as the highest yielding vegetable with 0.2 Mha, and the major producers in the Mediterranean basin are Turkey, Egypt, Italy, Spain, and Morocco [1].

The production of fresh market and processed tomatoes is hampered by numerous diseases caused by fungi, bacteria, phytoplasmas, viruses and viroids (Table 1).

Table 1. List of tomato plant pathogens present in the Mediterranean basin.

Pathogen Group	Pathogen Name	Reference
Fungi	<i>Alternaria solani</i> , <i>Botrytis cinerea</i> , <i>Cladosporium fulvum</i> , <i>Colletotrichum coccodes</i> , <i>Fusarium oxysporum</i> , <i>Fusarium clavum</i> , <i>Leveillula taurica</i> , <i>Oidium lycopersici</i> , <i>Pseudoidium neolycopersici</i> , <i>Pyrenochaeta lycopersici</i> , <i>Rhizoctonia solani</i> , <i>Septoria lycopersici</i> , <i>Sclerotinia sclerotiorum</i> , <i>Sclerotium rolfsii</i> , <i>Stemphylium spp.</i> , <i>Verticillium dahliae</i>	[2,3]
Oomycetes	<i>Phytophthora infestans</i> , <i>Phytophthora nicotianae</i> , <i>Phytophthora cryptogea</i> , <i>Pythium debaryanum</i> , <i>Pythium sylvaticum</i>	[4]
Bacteria	<i>Clavibacter michiganensis</i> subsp. <i>michiganensis</i> , <i>Erwinia carotovora</i> subsp. <i>carotovora</i> , <i>Pseudomonas corrugata</i> , <i>Pseudomonas mediterranea</i> , <i>Pseudomonas syringae</i> pv. <i>tomato</i> , <i>Ralstonia solanacearum</i> , <i>Xanthomonas axonopodis</i> pv. <i>vesicatoria</i>	[4]
Phytoplasma	<i>Candidatus Phytoplasma solani</i>	[5]
Viruses	Alfalfa mosaic virus (AMV), Chickpea chlorotic dwarf virus (CpCDV), Cucumber mosaic virus (CMV), Eggplant mottled dwarf virus (EMDV), Parietaria mottle virus (PMoV), Pelargonium zonate spot virus (PZSV), Pepino mosaic virus (PepMV), Potato virus Y (PVY), Southern tomato virus (STV), Tobacco mosaic virus (TMV), Tomato brown rugose fruit virus (ToBRFV), Tomato chlorosis virus (ToCV), Tomato infectious chlorosis virus (TICV), Tomato leaf curl New Delhi virus (ToLCNDV), Tomato mosaic virus (ToMV), Tomato spotted wilt virus (TSWV), Tomato torrado virus (ToTV), Tomato yellow leaf curl virus (TYLCV), Tomato yellow leaf curl Sardinia virus (TYLCSV)	[2,6]
Viroids	Potato spindle tuber viroid (PSTVd), Tomato apical stunt viroid (TASVd)	[5]

The cultivated tomato has a low genetic diversity due to its intensive selection and severe genetic bottlenecks that arose during evolution and domestication [7–9]. For these reasons, the tomato is more prone to a high disease incidence, and during cultivation and post-harvest period, it can be affected by more than 200 diseases caused by different pathogens throughout the world [10,11]. In this review, we present the most important pathogens affecting tomato in the Mediterranean area (Table 2), with the objective to describe their distribution, symptoms, way of transmission, the most favourable environmental conditions for their development, and their impact on crop yield, highlighting the influence of climate change on disease severity. In addition, we provide a state-of-the-art of the most recent, sophisticated, and sensitive detection methods and of the control measures currently authorized in EU that allow growers to achieve a successful and eco-sustainable disease management of this vegetable crop, fundamental for the Mediterranean diet.

Table 2. List of the tomato pathogens described in this review, their geographical distribution and impact of climate change on the disease severity.

Tomato Pathogen	Tomato Disease	Geographical Distribution *	Maximal Yield Loss (%)	Impact of Climate Change on Disease Severity on Tomato		
				Temperature and CO ₂ Concentration	Relative Humidity	
Fungi	<i>Alternaria solani</i>	Early blight of tomato	Portugal, Spain, France, Italy, Greece, Turkey, Cyprus, Malta, Israel, Lebanon, Egypt, Lybia, Morocco	80 [12]	Decrease at low and high T; Optimal T at 25 °C [13]	Increase with free moisture or near-saturated RH [14]
	<i>Septoria lycopersici</i>	<i>Septoria</i> leaf spot	Portugal, Spain, France, Italy, Greece, Turkey, Cyprus, Israel, Lebanon, Lybia, Morocco	50 [15]	No germination of conidia at ≥30 °C [16,17]	Increase with high RH [16]
	<i>Botrytis cinerea</i>	Grey mould	Spain, Italy, Turkey, Egypt	40 [18]	Increase at high T (25 °C) [19,20]; Decrease at high CO ₂ (800 ppm) [21]	Increase with nearly 90% RH [18]
	<i>Fusarium oxysporum</i> f. sp. <i>lycopersici</i>	Fusarium wilt of tomato	Spain, France, Italy, Albania, Turkey, Cyprus, Israel, Egypt, Lybia, Morocco	70 [22–25]	Conidial germination decreases at low T (<20 °C) and high T (>35 °C) [26]	Increase with high RH [27]
	<i>Fusarium oxysporum</i> f. sp. <i>radicis-lycopersici</i>	Crown and root rot	Spain, Italy, Greece, Turkey, Israel, Egypt, Tunisia, Malta	90 [28–30]	Increase at high T (27 °C) [31]	Decrease at high humidity in first stages of the disease [31]
	<i>Verticillium dahliae</i>	Verticillium wilt of tomato	Portugal, Spain, France, Italy, Greece, Turkey, Cyprus, Malta, Israel, Lebanon, Syria, Egypt, Algeria, Morocco	50 [32,33]	Increase at medium to high T (21–30 °C) [34]	n.a.
Bacteria	<i>Clavibacter michiganensis</i> subsp. <i>michiganensis</i>	Bacterial canker	Portugal, Spain, France, Italy, Greece, Turkey, Cyprus, Malta, Israel, Lebanon, Syria, Egypt, Tunisia, Morocco	84 [35–37]	Decrease at low T (<18 °C) and high T (>28 °C) [38]	Increase with high RH (80%) [39]
	<i>Pseudomonas syringae</i> pv. <i>tomato</i>	Bacterial speck	Portugal, Spain, France, Italy, Greece, Turkey, Israel, Lebanon, Tunisia, Morocco	75 [40,41]	Decrease at high T (28 °C) [42]; Decrease at high CO ₂ (800 ppm) [43]	Increase with high RH [40]
Phytoplasma	<i>Candidatus</i> Phytoplasma solani	Stolbur	Spain, France, Italy, Greece, Albania, Montenegro, Croatia, Turkey, Israel, Lebanon, Syria	80 [5,44]	n.a.	Increase with high RH [45]

Viruses	Tomato spotted wilt virus	Spotted wilt disease of tomato	Portugal, Spain, France, Italy, Greece, Croatia, Albania, Montenegro, Cyprus, Malta, Turkey, Israel, Lebanon, Egypt, Lybia, Algeria, Tunisia	95 [46,47]	Increase at medium to high T [48]	n.a.
	Cucumber mosaic virus	Tomato fern leaf	Portugal, Spain, France, Italy, Greece, Croatia, Albania, Montenegro, Cyprus, Malta, Turkey, Israel, Lebanon, Egypt, Algeria, Tunisia, Morocco	100 [49]	Decrease at high T (32 °C) [50]	n.a.
	Tomato yellow leaf curl virus	Tomato yellow leaf curl disease	Portugal, Spain, France, Italy, Greece, Cyprus, Malta, Turkey, Turkey, Israel, Lebanon, Egypt, Algeria, Lybia, Tunisia, Morocco	100 [51,52]	Decrease at elevated CO ₂ (750 ppm) [53]	n.a.
	Tomato yellow leaf curl Sardinia virus	Tomato yellow leaf curl disease	Spain, Italy, Greece, Tunisia, Morocco	100 [51,52]	n.a.	n.a.
	Tomato brown rugose fruit virus	Tomato brown rugose fruit disease	Spain, Italy, Greece, Cyprus, Malta, Turkey, Israel, Palestine, Egypt	100 [54]	n.a.	n.a.
	Tomato mosaic virus	Tomato mosaic disease	Spain, France, Italy, Turkey, Syria, Egypt, Algeria	70 [55,56]	Increase at medium to high T (20–31 °C) [57]	Decrease with high RH (>70%) [57]
	Parietaria mottle virus	n.a.	Spain, France, Italy, Greece	30 [58]	n.a.	n.a.
	Pepino mosaic virus	n.a.	Spain, France, Italy, Greece, Cyprus, Turkey, Israel, Syria, Egypt, Morocco	80 [59,60]	Increase or decrease at high T (30 °C) depending on the virus genotype [61]	n.a.
	Viroids	Potato spindle tuber viroid	Bunchy top of tomato	Spain, Italy, Croatia, Montenegro, Greece, Malta, Turkey, Israel, Egypt	90 [62]	Decrease at low T (15 °C); Increase at high T (31 °C) [63,64]

* Data obtained from <https://www.cabi.org/> and from <https://gd.eppo.int/> (accessed on 10 June 2021); T = temperature; RH = relative humidity; n.a. = not available.

2. Fungal Diseases

2.1. *Alternaria solani*

Alternaria solani (Ellis & Martin) Jones & Grout (*As*; see Table S1 for the abbreviations and acronyms used in this review) is the large-spored fungal species belonging to the phylum *Ascomycetes* and to the largest *Alternaria* section *Porri* [66]. *Alternaria* species, considered asexual fungi, are important pathogens of plants and animals [67]. Among them, *As* is one of the causative agents of the early blight disease of tomato, an important foliar disease present throughout the Mediterranean basin [4,68,69]. Favourable conditions for *As* growth and spread include frequent rainfalls, high humidity, and medium-to-high

temperatures (24–29 °C) [14]; under severe infections, it may cause up to 80% yield loss [12].

This necrotrophic fungus causes typical dark concentric spots on leaves, often surrounded by a chlorotic halo (Figure 1).



Figure 1. Early blight symptoms caused by *Alternaria solani*, with target-like concentric rings.

As disease develops, defoliation occurs, starting from older leaves and moving towards younger ones; necrotic lesions may be observed on flowers and stems [70]. Berries may also exhibit necrotic lesions that expand from the peduncle insertion point. The disease can lead to complete defoliation, strongly influencing photosynthetic efficiency of the plant and yield and quality of the fruits [71,72].

As is a seed-borne pathogen, and the use of healthy seeds is of primary importance for disease prevention [73]. However, *As* also spreads through alternative ways, as it may survive in plant debris by way of conidia and mycelia and in soil by way of chlamydo-spores [74].

Efficient disease management can be achieved through the use of sensitive and sophisticated detection methods. Thus, in-field loop-mediated isothermal amplification (LAMP), hyperspectral measurements, and ensemble machine learning methods have been recently developed, which are suitable for early and mass detection [75,76]. In addition, species-specific polymerase chain reaction (PCR) and multilocus sequencing are highly recommended techniques to differentiate *A. solani* from other *Alternaria* species infecting tomato [66,77].

For the successful management of this disease, rotation of fungicides with different modes of actions (multi-site and eco-sustainable fungicides in particular) is crucial due to the onset of resistant strains, particularly against respiration inhibitor fungicides (quinone outside inhibitors (QoIs) and succinate dehydrogenase inhibitors (SDHIs)) [78,79]. Additional strategies to effectively manage the early blight disease include preventive agronomic methods, starting from the choice of resistant cultivars, the use of healthy seeds or transplant material, the adoption of forecasting models, the control of above-ground plant humidity via soil-directed irrigation, crop alternation, the elimination of weeds and plant

residues, and the increase of plant vigour through an appropriate fertilization management [80–82].

2.2. *Septoria lycopersici*

Septoria leaf spot, caused by the fungus *Septoria lycopersici* Speg. (*Sl*), is another important foliar disease of tomato widely distributed in the Mediterranean basin. *Sl* belongs to the *Septoria* genus of asexual morphs of ascomycota, causing leaf spot diseases on numerous horticultural and wild plants [83]. Yield reductions in tomato due to *Septoria* leaf spot disease may reach around 50% when up to 75% defoliation is visible on plants [15]. Yield may be also considerably affected in the context of climate change, particularly with high humidity and temperature conditions [84–86]. In general, the growth of this fungus is favoured by medium temperatures (20–25 °C) with high humidity and by rainfalls or over-head irrigation with wetting of leaves for extended periods [16,87,88].

Septoria leaf spot symptoms consist of circular to oval necrotic lesions with a light grey colour surrounded by dark margins; on older leaves, necrotic spots may be surrounded by a chlorotic halo, and pycnidia may be observed in the central part of the lesions (Figure 2). Lesions may coalesce, leading to leaf chlorosis, defoliation, with consequent excessive sun exposure of the fruits [16,89].



Figure 2. Leaf spot disease caused by *Septoria lycopersici*.

The spread of the disease is favoured by the overwintering of the pathogen in alternative solanaceous hosts, such as the weeds *Solanum nigrum*, *S. carolinense*, and *Datura stramonium*, as well as in the debris of tomato plants [4,68,90]. Moreover, *Sl* is seed-transmitted [86], a feature additionally complicating its containment.

New approaches in fungal detection have been experimented for *Sl* on tomato, through multilocus sequence typing [83]. In general, management strategies and forecasting disease-warning models for this pathogen are similar to those of *A. solani* [78,91]. In addition, implementation of cultural practices including row spacing, staking and defoliation of plants, and intercropping were shown to have a positive impact on disease management [92].

2.3. *Botrytis cinerea*

Under favourable conditions, *Botrytis cinerea* Pers. (*Bc*), the anamorph of *Botryotinia fuckeliana* (de Bary) Whetzel, causes the devastating grey mould disease on tomato. This ascomycete belongs to the cosmopolitan *Botrytis* genus (family *Sclerotiniaceae*), which infects over 200 host plants cultivated both in greenhouse and field, including ornamental and wild species [93]. *Bc* may cause yield losses of tomato in some countries of the Mediterranean region, both under protected cultivation and in open field, although the damages occurring inside greenhouses are more serious [94–96]. Tomato yield loss accounts to around 20% [20,97], but it may reach up to 40% under optimal environmental conditions (mild temperatures between 15 and 20 °C and relative humidity above 90%) [18]. Severe infections might also develop under protected conditions when combinations of high temperature (25 °C), stem wounds, and high concentration of air-borne conidia occur [20].

The typical symptoms caused on tomato by *Bc* are soft rots, water soaking of parenchyma tissues, and grey masses of conidia (Figure 3), while stem lesions may lead to complete plant collapse [98,99].



Figure 3. Grey mould on tomato petioles caused by *Botrytis cinerea*.

The most sensitive organs, such as stems, flowers, and fruits, may be infected through cultivation and pruning practices or by direct fungus penetration [100–102]. Conversely, soft rots of ripe tomato fruits occur mainly in post-harvest, while “ghost spot” symptoms related to a host defence response against this pathogen can arise on unripe fruits, making them hardly marketable [99].

Transmission of this necrotrophic fungus occurs mainly through air-borne conidia originating from infected plants of tomato or other hosts, or through plant debris [101], while no evidence of seed-transmission has been reported. *Bc* may overwinter as sclerotia in the soil and in decayed plant residues that will last till the following growing season [103].

For the detection of *Bc*, sensitive molecular (e.g., LAMP) and multilocus sequence typing methods are preferable, in order to obtain a precise species identification [104] and to identify the fungus at early disease stages or in latent infections [105].

The current management disease strategies include the use of conventional fungicides and of resistant cultivars. Additional strategies against this disease rely on agronomic measures, such as ventilation of the greenhouse, appropriate distancing between plants, prudent use of irrigation and nitrogen fertilization, removal of infected debris, and caution during agricultural operations in order to avoid plant wounding. Physical measures such as heat treatment of tomato seeds may also be effective for the disease management. Additionally, different measures that stimulate plant defence mechanisms have been described, such as the treatments with the defence elicitor chitosan, with plant-growth-promoting steroids, and with beneficial bacteria acting as inducers of plant resistance or as biological control agents (e.g., *Pythium oligandrum*, *Bacillus licheniformis*, *B. amyloliquefaciens*, *Pseudomonas* strain QBA5, *Streptomyces* spp., *Trichoderma atroviride*) [106–109].

2.4. *Fusarium oxysporum*

Fusarium oxysporum (Schlectend. Fr.) is a species complex composed of asexual ascomycetes, which includes soil-borne plant pathogenic fungi comprising more than 150 host-specific *formae speciales* (f. sp.) but also non-pathogenic strains [110,111]. Noteworthy, this complex comprises species that can also infect humans and other animals [112]. Beside their negative impact on plant yield, it is relevant to note that few *Fusarium* species are important mycotoxin producers in fresh and processed foods [113]. Among the numerous existing *formae speciales*, two affect mostly tomato, i.e., *F. oxysporum* f. sp. *lycopersici* Snyder and Hansen (*Fol*) and *Fusarium oxysporum* f. sp. *radicis-lycopersici* Jarvis & Shoemaker (*Forl*). Interestingly, these two tomato f. sp. have been recently assigned to the phylogenetic species *Fusarium languescens* due to epitypification of *Fusarium oxysporum* [114].

2.4.1. *Fusarium oxysporum* f. sp. *lycopersici*

Fol causes the *Fusarium* wilt, one of the most common soil-borne diseases of tomato, and is widely spread throughout the Mediterranean basin [115–118]; it infects the tomato transplants and plants cultivated both in greenhouse and open-field conditions. Until now, three physiological races of *Fol* are distinguished, namely 1, 2, and 3, based on their pathogenicity on tomato cultivars containing race-specific resistance genes [119]. Races 1 and 2 are the main ones present in the Mediterranean area and throughout the world, while race 3 has a more limited distribution, mainly in Turkey, Egypt and Algeria [115,118,120,121]. Yield losses due to *Fol* may reach 45–55% and extend till 70% in case of favourable environmental conditions (27–30 °C) [22–25].

The initial symptoms of the disease appear as yellowing of the lower leaves, a symptom that may be confined to only one part of the plant due to the sectorial invasion of the pathogen [122]. After root penetration, the fungus colonizes intercellularly the root cortical cells and invades the vascular tissue through the xylem pits, causing a typical dark brown colour that can extend throughout the upper stems, leading to wilting, collapse and death of the plant [113] (Figure 4). The pathogen is host-specific and may infect only few solanaceous plants [122,123].



Figure 4. *Fusarium* wilt caused by *Fusarium oxysporum* f. sp. *lycopersici* causing typical dark brown colour on plant vessels (A), leading to plant collapse (B).

Being a soil-borne pathogen, *Fol* may survive as chlamydospores in the soil for long periods and also in plant debris. It is also a seed- and air-borne pathogen, spreading its conidia easily by air currents [124,125].

Fol disease management includes the use of fungicides (i.e., bromuconazole) and biological control agents (*B. subtilis*, *B. amyloliquefaciens*, *Streptomyces griseoviridis*, *Trichoderma harzianum*) [126–130]. Additional strategies encompass standard agricultural practices (plant rotation and elimination of infected plants, plant debris and weeds), use of resistant cultivars carrying immunity (*I*) genes derived from wild relatives (*I*-2, *I*-3, and *I*-7) that provide resistance against all the three pathogen races, use of *Fol*-resistant rootstocks, organic substrates, silicon amendments, soil solarization, soil fumigation in combination with metham sodium, and treatment with plant essential oils and plant extracts. These strategies, together with rapid and sensitive *Fol* detection (e.g., LAMP) and specific-race PCR detection (amplification of the *secreted in xylem* (*SIX*) genes, *SIX3* and *SIX4*, as an example) may be implemented for a successful disease management [122,126,130–135].

2.4.2. *Fusarium oxysporum* f. sp. *radicis-lycopersici*

Forl is the causal agent of the tomato crown and root rot, one of the most destructive soil-borne diseases of tomato. Nowadays, this pathogen is commonly present in most of the Mediterranean countries, infecting tomato plants during the transplantation phase or during cultivation [28,118,136,137]. *Forl* causes around 20–60% yield loss, possibly reaching up to 90% under optimal pathogen growth conditions, i.e., cold weather (<20 °C), sterilised or fumigated soils, or cultivation in soilless systems [28–30]. However, high disease incidence on tomato plants under high-temperature regime (27 °C) has also been reported [31]. No physiological races of *Forl* have been recognized, and its genetic diversity has been documented by the existence of several vegetative compatibility groups (VCGs); until now, six VCGs (0090, 0091, 0092, 0093, 0094, 0096) have been identified in the Mediterranean countries, including Italy, France, Greece, Turkey, Tunisia, and Morocco [138–140].

Forl causes vascular and substantial cortical discolourations that extend from 20–30 cm above the ground. Necrotic brownish lesions can develop on the main root and on lateral roots, which may lead to root rot and to the development of adventitious roots on the stems above the ground [122] (Figure 5). In case of severe attacks, the fungus may cause complete wilting and death of plants. In contrast to *Fol*, this pathogen may infect

few solanaceous and apiaceous plants and a wide range of leafy vegetable and leguminous plants [141–143].



Figure 5. Crown and root rot caused by *Fusarium oxysporum* f. sp. *radices-lycopersici*. (A) Necrotic brownish lesions on the stem. (B) Adventitious roots on the stem.

Like *Fol*, this fungus is a soil-borne pathogen, surviving in the soil and in plant debris in the form of chlamydospores. It is also an air-borne pathogen, easily spreading its conidia by air currents and, to a lesser extent, a seed-borne pathogen.

A multilocus sequence-typing technique is necessary for precise species identification, a prerequisite for a successful management of tomato crown and root rot [114]. The disease management includes the use of eco-sustainable fungicides, including chemical (i.e., hymexazol) and biological control agents (*B. amyloliquefaciens*, *Pseudomonas chlororaphis*, *T. gamsii*, *Pythium oligandrum*) [144–147]. Standard agricultural practices (plant rotation, use of uninfected seed and soil, elimination of propagation material with suspicious crown and root rot symptoms, elimination of weeds and plant debris immediately after harvest, avoidance of plant wounds) and use of resistant cultivars carrying the single dominant *Frl* locus and of *Forl*-resistant vegetable rootstocks, combined use of soil solarization with metam sodium or with hydrogen peroxide and benzoic acid, use of elicitors inducing resistance (chitosan), and plant extracts represent additional techniques implemented for a successful disease management [122,148–150].

2.5. *Verticillium dahliae*

The asexual ascomycete *Verticillium dahliae* Kleb. (*Vd*) is a soil-borne fungal pathogen causing the *Verticillium* wilt disease on tomato and on many other host plants in the Mediterranean countries and worldwide [151–154]. There are two races of *Vd*-infecting tomato, race 1 carrying the avirulence gene *VdAve1* and race 2 which lacks it [155], and both are present in the Mediterranean region [156–158]. Yield reduction in tomato may reach 20–50% [32,33] with a higher impact in the temperature range of 21–30 °C, optimal for *Vd* growth [34].

The infection of plants starts with the penetration of the fungus into the roots, followed by the colonization of the xylem vessels. The fungus spreads acropetally through the water-conducting vessels to the aboveground parts of the plant, leading to vascular wilt disease; brown discoloration is manifest when the plant is incised. Symptoms are initially visible on older leaves and then progress to younger leaves, with the desiccation extending from the tip towards the petiole (Figure 6). The plant reacts to the infection by forming barriers in the vessels, but such obstructions lead to withering and death [159].



Figure 6. Wilting symptoms caused by *Verticillium dahliae* (Courtesy of Aida Kohnić, Dušica doo, Čapljina, Bosnia and Herzegovina).

Management of the *Verticillium* wilt is difficult due to the long survival in the soil of the fungal resting structures (microsclerotia) and to the wide range of plants hosting this pathogen [160,161]. *Verticillium* wilt management primarily requires a precise species and race identification, for which the multilocus sequence technique and race-specific PCR are recommended [154,155,162]. Soil fumigation with chloropicrin, dazomet, dimethyl disulfide, and metam (including sodium and potassium) are efficacious control measures on tomato [163–167]. Elimination of plant debris, plant rotation, use of resistant tomato cultivars and the rootstocks mediated by tomato *Ve1* gene, non-chemical soil disinfestation practices (e.g., soil heating, anaerobic soil disinfestation, biofumigation), use of organic amendments, and supplementation with biocontrol agents (*T. harzianum*, *S. griseoviridis*) are other methods useful for a successful management of the disease [126,168,169].

3. Bacterial Diseases

3.1. *Clavibacter michiganensis* subsp. *michiganensis*

Clavibacter michiganensis subsp. *michiganensis* (Smith) Davis, Gillaspies, Vidaver & Harris (*Cmm*) is a gram-positive, non-motile actinomycete belonging to the *Microbacteriaceae* family, phylum *Actinobacteria* [170]. It is the causal agent of the bacterial canker of tomato, the most important bacterial disease of this crop, causing considerable losses throughout the world [171]. This pathogen is mainly restricted to the Mediterranean area, with the exception of Greece and Turkey, where it has a widespread distribution. This bacterium is currently classified as an EPPO (European Plant Protection Organization) A2 quarantine pathogen [172]. Beside tomato, *Cmm* can attack other solanaceous plants [173,174]. The optimal temperature for the disease development is 26 °C, leading to an extremely rapid plant death [38] with yield reduction ranging from 46 to 84% [35–37].

Since the bacterium colonizes vascular tissue, symptoms are visible on the whole plant (Figure 7).



Figure 7. Initial symptoms of bacterial canker caused by *Clavibacter michiganensis* subsp. *michiganensis*.

Symptoms develop slowly and are rarely visible in the nursery but become clearly manifest between fruit set and the beginning of ripening. Leaf margins roll upward and begin to wilt and dry up, and such wilting may affect individual parts or the whole plant. Longitudinal yellowish or brown streaks may appear on the stem, in correspondence of which tumour structures may form [175]. Tiny, dark lesions surrounded by bright halos resembling characteristic birds-eye spots develop on infected berries [176], and in later infection stages, extensive wilting and plant death are observed [177].

Cmm is a seed-borne pathogen, and the use of healthy seeds is elementary to prevent its diffusion [178,179]. External seed contamination with *Cmm* may also contribute to pathogen transmission [180]. However, besides seeds, the bacterium may also survive in plant debris [181].

Copper-based treatments may reduce the epiphytic presence of bacteria but are not effective against systemic infection [37]. Biological control and treatment with compost, elicitors inducing resistance, plant essential oils or plant extracts, and resistance obtained through traditional breeding or genetic engineering have been employed against the bacterial canker disease but only with moderate effectiveness [170,181]. The most recommended techniques for a timely detection of *Cmm* directly in the field, even at early stages of infection, are based on LAMP [182,183] and ImmunoStrip® visual end-point assay commercialized by Agdia (<https://orders.agdia.com/agdia-immunostrip-for-cmm-isk-44001>) (accessed on 10 June 2021), forming the basis for prompt disease management strategies. Additional measures for the disease management of *Cmm* include the thermotherapy of seeds (48–52 °C for 20 min) or seed treatment with acidified nitrite or 1% hydrochloric acid, crop rotation every 4–5 years, and the eradication of infected plants. It is advised to avoid sprinkler irrigation and excess nitrogen fertilization. In addition, frequent disinfection of cutting tools and hail protection are suggested as well [170].

3.2. *Pseudomonas syringae* pv. *tomato*

Pseudomonas syringae pathovar (pv.) *tomato* Okabe (*Pst*) is a motile, gram-negative bacterium of the phylum *Proteobacteria* (family *Pseudomonadaceae*), which causes the bacterial speck of tomato, an occasionally destructive disease of tomato cultivated both in greenhouse or open field [184]. *Pst* is genetically monomorphic with a recent evolutionary origin on tomato. It is present in different Mediterranean countries where the T1-like strains are the predominant strains, as in the rest of the world [185–187]. Two *Pst* races (0 and 1) have been identified so far, based on the different responses of tomato cultivars with or without resistance gene to *Pseudomonas syringae* pv. *tomato* (*Pto*) and based on the *Pst* expression of the avirulence (*avr*) genes (*avrPto* and *avrPtoB*) [188]; so far, the more

virulent race 1 has been reported in Italy and Portugal and Tunisia [189–192]. *Pst* is favoured by a temperature range of 13–28 °C, with a high humidity and even with free water on leaves [40]. Bacterial speck outbreaks may cause 20–25% seedling losses, while tomato yield losses may reach 75% in case of early-infection [40,41].

Initially, hydropic, angular spots become visible on leaves of infected plants; these spots can further converge, affecting larger portions of the leaf and assuming a dark colour surrounded by a chlorotic halo. Symptoms on stems include elongated necrotic spots (Figure 8), while fruits show tiny, rounded dots of few millimetres, surrounded by a green halo [193], thereby reducing their marketable value. *Pst* may survive on tomato seeds, in particular within the cavities present on the seed surface [194,195].



Figure 8. Bacterial speck disease caused by *Pseudomonas syringae* pv. *tomato*.

The principal dissemination of *Pst* occurs through infected seeds, but long-distance spread through the atmosphere is also documented, due to its presence in rain and snow [196].

The detection of *Pst* can be achieved by LAMP assays or real-time PCR in the early phases of the disease or directly on contaminated seeds, respectively [197,198].

Currently, the disease management is based on the chemical control of *Pst* in the field through the application of copper-based bactericides. However, *Pst* copper-resistant populations have arisen worldwide, urging the necessity to adopt alternative control strategies [199–201]; for example, the use of pathogen-free seeds and the elimination of plant debris are of utmost importance. Ventilation in greenhouses aimed at maintaining the leaves in dry conditions and treatments with plant extracts or essential oils may be useful for the management of the bacterial speck [202–204]. Moreover, a few biological control agents have been reported to efficiently counteract the bacterial speck disease, e.g., *Azospirillum brasilense*, *P. syringae* Cit7, *P. fluorescens*, *P. aeruginosa*, *B. stratosphericus*, *B. pumilus*, plant growth-promoting rhizobacteria, and bacteriophages, but none of them is currently commercialized or approved in EU [202,205–208].

4. Phytoplasma Diseases

4.1. *Candidatus Phytoplasma solani*

Candidatus Phytoplasma solani (CPs) is a wall-less, non-helical, non-culturable, and phloem-limited prokaryote belonging to the class *Mollicutes* in the taxonomic phytoplasma group 16SrXII [209,210]. CPs represents the most important phytoplasma of tomato, on which it causes the stolbur disease. Beside tomato, CPs infects also other cultivated, spontaneous herbaceous and woody plants, such as grapevine, on which it causes the Bois Noir disease [210]. CPs is present in different European and West-Asian Mediterranean countries and is currently listed as an EPPO A2 quarantine pathogen [172]. During the outbreaks, CPs can provoke considerable yield reductions that may reach up to 80% [5,44].

CPs causes a change in the distribution of iron in tomato leaves, influences the photosynthesis and compromises the organization of root-mediated transport functions [211]. Initial infection may result in an erect stand growth of the plants which assume gradually a bushy appearance due to the stem enlargement and to an increased number of axillary stems and shoots with shortened internodes. Leaves are reduced in size, distorted and curled, showing yellowing (Figure 9) and in some cases reddening.



Figure 9. Stolbur disease symptoms induced by *Candidatus Phytoplasma solani*. (A) Reduced size and yellowing of leaves. (B) Distorted and curled leaves.

The upper part of the plant may eventually become defoliated. Flowers exhibit virescence and phyllody, with a reduced number of anthers and ovary and enlarged calyces (hence the English name of the disease, “tomato big bud”). A reduced number of fruits is also observed, and fruits are smaller, with a paler colour and a harder pulp consistency compared to healthy fruits [212–214].

CPs is transmitted by the polyphagous planthopper *Hyalesthes obsoletus* (Signoret 1865; family *Cixiidae*), which transmits the phytoplasma on tomato during vegetation from infected bindweed (*Convolvulus arvensis* L.) or stinging nettle (*Urtica dioica* L.), on which the insects overwinter [215–217].

Modern diagnostic techniques for CPs are available, such as a stolbur-specific PCR protocol [218] and a rapid LAMP assay [219]. The main management measures of the stolbur disease include the use of healthy propagation material, a constant monitoring and control of insect vectors and of symptom onset, timely detection, and elimination of infected plants and of reservoirs of alternative spontaneous host plants [220]. The use of

more tolerant tomato cultivars may be an alternative route of control, while bactericides, such as antibiotics, against phytoplasma are not allowed in EU [221].

5. Viral Diseases

5.1. Tomato Spotted Wilt Virus

Tomato spotted wilt virus (TSWV) is the type member of the genus *Orthospovirus* in the family *Bunyaviridae*. TSWV particles are spherical or pleomorphic, with a diameter of about 80–120 nm, enveloped by a double-layer lipoprotein membrane; the membrane presents numerous pyriform protrusions of 5–10 nm, which are composed of glycoproteins. Regarding its genomic composition, TSWV presents a multipartite genome composed by three negative/ambisense single-stranded RNA molecules [222].

TSWV is a temperature-sensitive virus; in fact, depending on the isolate, the thermal inactivation points vary from 40 to 46 °C, with an exposure time of approximately 10 min [223]. TSWV infects a wide range of agricultural crops, including tomato [224,225], where it is responsible for the spotted wilt disease. TSWV is spread in all Mediterranean countries apart from Morocco [226] and may cause from 40–90% and up to 95% loss in yield and marketable value of tomato, respectively [46,47].

The initial symptoms on leaves can be mistaken with cold injury, since young leaves show a purple colouration on the lower leaf laminas. As the disease progresses, chlorotic spots are visible on leaves, which later become necrotic. These gradually merge, giving a brown-violet colour to the leaf, known as “leaf bronzing” [2]. Necrosis can extend onto petioles, stems, and flowers and with spots with chromatic alterations on the berry. These symptoms are the most frequent and typical at the early infection stages and can lead to plant death before the production of the first berries. Fruits exhibit light green spots of around 1 cm that later become necrotic, depressed, and brownish (Figure 10) [2].



Figure 10. Necrotic and brownish ring spots on immature fruits caused by Tomato spotted wilt virus.

A suberose consistency of the spots with deep longitudinal cracks formed near the peduncle leads to early fruit drops. Infected plants have a reduced size, but if infection occurs in the nursery before transplantation, plant death can rapidly occur in a few days [2,227]. Symptoms can vary due to the high number of strains and the frequency of genetic recombination and reassortment among them, thus generating new variants and conferring the ability to overcome the genetic resistance that has been introgressed into commercial cultivars [228]. Therefore, symptoms can vary depending on the time of infection, the host species, and also environmental factors.

TSWV is transmitted in a circulative and persistent manner exclusively by thrips, namely the western flower thrip, *Frankliniella occidentalis* (Pergande, 1895), the most efficient vector [229].

TSWV infection can be visually identified thanks to its characteristic symptomatology, but it is always advised to confirm visual inspection with laboratory assays. These consist in serological assays based on enzyme-linked immunosorbent assay (ELISA) using TSWV-specific antibodies. More sensitive molecular techniques are available, such as reverse transcription-polymerase chain reaction (RT-PCR) or real-time RT-PCR [230]. Moreover, quick isothermal amplification methods, including reverse transcription-helicase-dependent amplification (RT-HDA) and reverse transcription-loop-mediated isothermal amplification (RT-LAMP) assays were developed for specific virus detection [231].

TSWV disease management is difficult due to the biological and molecular characteristics of the pathogen, the wide diffusion of thrips vectors, and to its extremely wide host range. Therefore, as for most virus diseases, it is necessary to deploy preventive and integrated control strategies using healthy plant material and resistant tomato cultivars with the gene locus *Sw-5*, particularly the homolog *Sw-5b* allowing broad and durable resistance against TSWV [228]. Under protected cultivation systems, it is fundamental to reduce the access of insect vectors using close mesh nets (40 mesh or greater). Biological or chemical control strategies of insect vectors can be adopted, taking care to alternate different methods or chemical substances in order to delay, if not prevent, the onset of resistance to insecticides. Additionally, if TSWV-positive plants are found, it is essential to remove and destroy all symptomatic and neighbouring plants, including weeds [2]. Finally, strategies based on RNAi have been experimented, using either transgenic plants or the exogenous application of double stranded RNA molecules.

5.2. Cucumber Mosaic Virus

Cucumber mosaic virus (CMV) is the type member of the genus *Cucumovirus* of the family *Bromoviridae* [232]. The virions are constituted of three icosahedral particles with a diameter of about 29 nm. The CMV genome is composed of three segments of positive single-stranded RNA, called RNA-1, RNA-2, and RNA-3. Another RNA molecule, called RNA-4, is generated by RNA-3. RNA1 encodes the 1a protein, which, together with the RNA-2-encoded 2a protein, forms the replicase complex; RNA-2 also encodes a second protein, 2b, which is involved with the long-distance movement. RNA-3 encodes two proteins, 3a, a cell-to-cell movement protein (MP), and 3b, the coat protein (CP), which facilitates cell-to-cell movement and virus transmission [233]. Some particular virus strains support the replication of a fifth non-genomic RNA segment called Carna 5, a satellite RNA that can modify symptom expression [234]. The presence of numerous CMV strains with a high genetic variability is thought to explain the extremely wide variety of symptoms that can make diagnosis difficult in some cases.

Currently, according to serological and molecular characteristics, all strains are grouped into two subgroups: Cucumber mosaic virus subgroup I–strain Fny (CMV-Fny) and subgroup II–strain Q (CMV-Q). Furthermore, subgroup I is divided into IA and IB [235]. Specifically, subgroup IA includes isolates that induce a generalized mosaic, while subgroup IB includes all the Asian isolates that induce necrotic lesions on leaves. Being a very adaptable virus with high evolutionary capacity, CMV may infect more than 1200 plant species and may be transmitted by more than 80 aphid species in a circulative, non-

persistent manner [236,237]. Transmission efficiency varies according to the aphid species, virus strain, host species, and environmental conditions. In particular, the green peach aphid (*Myzus persicae* Sulzer), cotton or melon aphid (*Aphis gossypii* Glover), black bean aphid (*A. fabae* Scop.), cowpea aphid (*A. craccivora* Koch.), and potato aphid (*Macrosiphum euphorbiae* Thomas) are the most efficient vectors. Moreover, CMV can also be transmitted by seeds [238] and through ten species of dodder.

CMV is present in all Mediterranean countries except for Libya [226]. In the most serious cases, losses of tomato production can reach up to 100% [49].

Symptoms caused by CMV infection may vary depending on the host, the environment, and the age of the plant at the time of infection but also the virus strain and the presence of the virus satellite [2,237,239]. In the case of early infections (within 15–20 days from transplanting), leaf malformations, stunted growth, and apical dwarfism can occur, leading to a bushy appearance of the plant. In severe infections, the leaves show curled and upward-rolled margins and filimorphism. The fruit ripening process is slowed down, and fruits often cannot reach full ripeness. Fruits can show a characteristic necrosis, with internal hardening in the proximity of the pedicel and darkened areas, which makes the product completely unmarketable (Figure 11) [2].

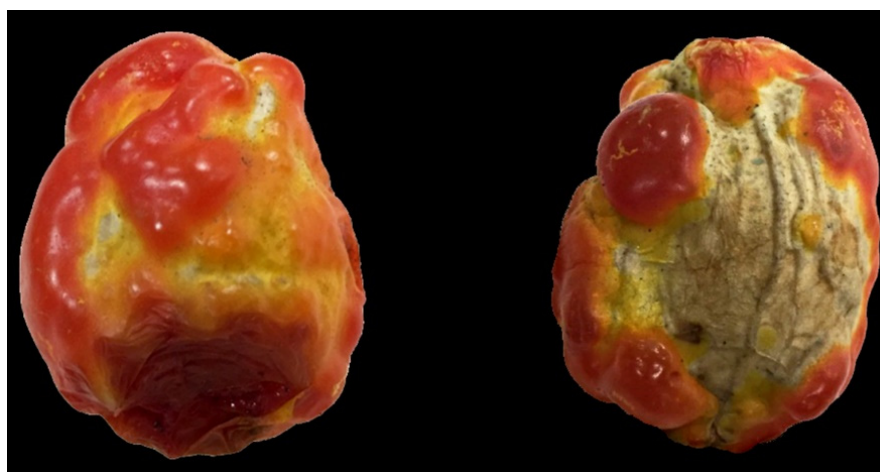


Figure 11. Large necrotic and depressed areas on fruits caused by Cucumber mosaic virus.

The most destructive symptom is lethal necrosis, caused by the necrogenic strain (CMV^N), which leads to the decay and death of the plant within 2–3 weeks from infection. In this case, yellow areas between the secondary veins of the young leaves appear, which rapidly necrotize; then, leaves appear rolled downwards, leading to whole plant wilting starting from the vegetative apex [2,239].

In the past, the pathogen was diagnosed using indicator plants, but this method has been replaced by simpler and faster procedures, such as serological and molecular tests. In the first case, the double antibody sandwich enzyme-linked immunosorbent (DAS-ELISA) technique relies on commercial polyclonal or monoclonal antibodies, that allow to identify and distinguish the different CMV isolates, but most sensitive and faster techniques are based on real-time RT-PCR and RT-LAMP [240,241].

Due to the commercial unavailability of CMV-resistant tomato varieties, use of CMV-free seeds and transplant material is of crucial importance for the control of this virus. Moreover, as the virus is very polyphagous, preventive measures to minimize the natural inoculum must be taken, for example, the elimination of contaminating weeds. Greenhouses must be kept free from weeds, both internally and externally, and sufficient distance, especially from solanaceous and cucurbit crops, should be adopted. Reflective mulch, change of transplanting date of tomato during the year, and biological control using plant growth-promoting rhizobacteria are additional practices useful for the management of CMV under greenhouse conditions [242]. Monitoring and treatments of aphid

vectors might be used in protected conditions. Protecting greenhouse openings with tightly meshed nets is a good preventive rule, even if it hinders the growth of seedlings, along with the adoption of hygiene procedures to prevent the mechanical transmission of the virus by agricultural tools and workers. Recently, treatment with chitosan has been reported as useful means to reduce the CMV titre in infected tomato plants [243].

5.3. Tomato Yellow Leaf Curl Virus and Tomato Yellow Leaf Curl Sardinia Virus

Several studies conducted in some Mediterranean countries, including Italy, have shown that there are at least two viruses responsible for the Tomato yellow leaf curl disease (TYLCD) [244,245], i.e., Tomato yellow leaf curl Sardinia virus (TYLCSV) and Tomato yellow leaf curl virus (TYLCV), and that genetic recombination between members of these species occurs frequently, giving rise to a wide range of new hybrid genomes [246,247].

TYLCV and TYLCSV, presently assigned as EPPPO A2 quarantine pathogens [172], belong to the genus *Begomovirus* of the family *Geminiviridae* [232]. Their genome consists of one molecule of circular single-stranded DNA (ssDNA) with a length of ca. 2.780 nucleotides (nt). The genome contains six open reading frames (ORFs) that encode six protein products, plus an intergenic region, that does not encode for any protein but is essential for virus replication.

TYLCV and TYLCSV are spread throughout the Mediterranean region with the exception of the territory of ex-Yugoslavia [226,248]. Yield losses caused by this virus depend on the phenological stage of the plant at the time of infection. Notably, early infections can lead to almost 100% crop losses, while late infections or infections of plants with a significant vegetative development prevent the setting of new fruits, while fruits already present ripen although with lower quality [51,52].

TYLCV and TYLCSV cause extremely destructive symptoms, but they infect a restricted number of host species. Symptoms on tomato plants vary depending on the environmental conditions, the development of the plant at the time of infection and the cultivar. When infection occurs during plant development, the symptoms consist of leaf distortions, with side jets and reduced growth, giving the plant a bushy appearance with upright axillary and apical shoots. Typical leaf symptoms include a reduction of the surface with evident yellowing and distortion. Young leaves show a notable reduction of the leaf blade which, in extreme cases, disappears completely, leaving only the main vein. Plant leaflets have their edges visibly yellowed and turned upwards, thus resembling a small cupboard [249] (Figure 12).



Figure 12. Yellowing and dwarfism symptoms caused by Tomato yellow leaf curl disease.

In colder periods, the yellowish colour can acquire purple shades resembling cold damage, as infected plants become more susceptible at low temperatures [2]. TYLCD symptoms may also include the abscission of flowers, failure to fruit set, and the production of unmarketable fruits due to their small size and/or pale colour [250].

In a natural context, TYLCV and TYLCSV are transmitted very efficiently by the garden whitefly (*Bemisia tabaci*, Gennadius), as the minimum acquisition period of the virus by whiteflies is between 15 and 30 min. The transmission is circulative and persistent, with a latency time of 17–21 h and a persistence of 7–20 days, i.e., lasting for the whole life of the insect. Females of the vector transmit TYLCV with up to six times greater efficiency than males [251–253]. In tomato plants, such viruses are neither seed nor mechanically transmitted; therefore, their epidemiological cycle remains closely linked to the insect vector and its hosts. Since the end of the 1980s, whiteflies have become the main animal adversary for horticultural crops in vegetable gardens of Southern Italy, particularly for crops grown in a protected environment, where this insect finds optimal conditions for its development compared to open field. The damages induced by this phytophagous species derive from its direct feeding (sap sucking and production of honeydew which attracts other phytophagous insects), from its ability to transmit over 40 different viruses, and from the relevant spread of different biotypes in the epidemiology of TYLCD. The *Bemisia tabaci* cryptic species complex of whiteflies (*Hemiptera: Aleyrodidae*) includes almost 40 putative species members, among which some are regarded as the worst invasive pests and plant-virus vectors, responsible for the spread of TYLCV and related viruses [254]. Among them, MEAM1 (Middle East-Asia Minor 1, former “B” biotype) and MED (Mediterranean, former “Q” biotype) are the two most destructive *B. tabaci* species [255,256].

The use of serological tests such as ELISA, based on the antigenic properties of the virus coat protein, is not considered sufficiently adequate for the identification and discrimination of the distinct species inducing TYLCD. With its low antigenicity, and due to the difficulties in the virion purification procedures, it is not easy to obtain a sufficiently sensitive and specific antiserum. Molecular techniques such as PCR and real-time PCR are very sensitive and versatile techniques for the diagnosis of geminiviruses [257], while LAMP assay allows a direct detection of the virus in field without the need of DNA extraction [258].

As for all viruses, the control of TYLCV and TYLCSV is of a preventive nature, and in case of overt infection, all measures must be taken to contain the disease within acceptable limits. To do this, especially in protected crops, it is essential to keep the vector under control and eliminate it by monitoring and through chemical and biological control measures. The seed companies have put on the market numerous cultivars and hybrids obtained through genetic improvement, bearing different degrees of resistance, which today greatly help to contain the damage caused by TYLCD. Several breeding strategies have been undertaken to introduce resistance alleles from the germplasm of related wild *Solanum* species, including *Solanum pimpinellifolium*, *S. peruvianum*, *S. chilense*, *S. habrochaites*, and *S. cheesmaniae* [259–261], leading to the introgression of up to six different resistance genes (*Ty-1* to *Ty-6*) [262].

5.4. Tomato Brown Rugose Fruit Virus

Tomato brown rugose fruit virus (ToBRFV) is a recently identified virus causing “the brown rugose” disease on tomato fruits [263]. This virus is assigned to the genus *Tobamovirus*, family *Virgaviridae* [232]. ToBRFV has the typical genome organization of the genus *Tobamovirus*, with a single, non-segmented RNA genome of approximately 6.400 nt containing four open reading frames (ORFs) encoding two replication-related protein complexes of 126 and 183 kDa, respectively, with the latter expressed by the partial suppression of the stop codon (ORF1a and ORF1b), the movement protein (MP) of ca. 30 kDa (ORF2), and a coat protein of about 17.5 kDa (ORF3), expressed via the 3'-coterminal subgenomic RNAs [263]. Following its first identification in Israel and Jordan in 2014/2015, ToBRFV was soon reported in few European, Asian, and Mediterranean countries and few others in the world, and it was shortly assigned as an EPPO A2 quarantine pathogen [172,226,263–265]. In detail, regarding the Mediterranean countries, ToBRFV was reported in Italy, Palestine, Greece, Spain, France, and recently in Egypt [266–270]. Actually, To-

BRFV is considered more dangerous than other tomato tobamoviruses since it infects commercial tomato cultivars carrying the *Tm-2²*-resistance gene active against Tomato mosaic virus (ToMV) [264]. In tomato crop, severe infections may cause yield loss of up to 100% [54].

The main hosts of ToBRFV are tomato and sweet pepper (*Capsicum annuum*) [271,272]. ToBRFV symptoms in tomato plants are extremely variable depending on the cultivar and the environmental conditions. Specifically, it seems that the symptomatic variations are associated to the photoperiod, temperature, and age of the plant at the time of infection. The most common symptoms are leaf deformations, mosaic and blistering, internodal yellowing especially in young leaves, longitudinal stem necrosis, suberification and necrosis of the sepals (Figure 13), necrosis on pedicles, calyces and petioles, fruit abscission, and fruit marbling and deformations. Severe infections may result in plant wilting and yellowing [264,266].



Figure 13. Leaf deformations with mosaic and blistering (A) or yellowing and necrosis (B) caused by Tomato brown rugose fruit virus.

ToBRFV transmission is basically mechanical, but it can also occur through contaminated seeds or fruits [273]. ToBRFV is considered a seed-borne virus, a feature that increases the risk of introduction into other areas where the virus is not yet present [274]. Furthermore, its mechanical transmission increases the risks of its spread through agricultural practices in greenhouses [275]; in fact, the ToBRFV mechanical transmission within crops is frequent, occurring through direct contact with infected plants [263], or through infected sap originating from different surfaces (operator, clothing, pots, working tools and nutrient solutions) [276], propagation materials (grafts, cuttings, seeds), and bumblebees (*Bombus terrestris*) [277].

Several diagnostic techniques are available to determine the presence of ToBRFV, including newly released ELISA-based serological assays, i.e., from Agdia (<https://www.agdia.com/company/news/1698673/tobrfv-elisa-launch>) and Loewe (<https://www.loewe-info.com/categories/elisa-sets-kits-and-controls/plant-viruses/tomato-brown-rugose-fruit-tobamovirus-tobrfv.html>) (accessed on 10 June 2021). For a more specific and reliable diagnosis, more specific molecular tests are used, such as RT-PCR, real time RT-PCR and RT-LAMP [266,278,279].

ToBRFV represents a risk for the entire EPPO region, where tomato and pepper crops are widely cultivated, especially in protected environments that favour the rapid spread

of the virus [273]. There are no curative products, and resistance genes to other tobamoviruses (TMV and ToMV) are not effective against ToBRFV; to date, there are no available cultivars with genetic resistance or tolerance to ToBRFV. However, a resistance trait has been reported recently that is associated with a tolerance locus located on chromosome 11 and with a locus of the *Tm-1* region located on chromosome 2, therefore initiating the resistance strategy against this pathogen [280]. The Bayer company also recently launched grape tomato, pink beef, beefs, and Roma-type tomatoes showing a strong intermediate resistance against ToBRFV. In general, the control of ToBRFV is based exclusively on careful and rigorous prevention activities, with an obligation to use healthy propagation material; carry out checks on imported seeds, especially in case of import from countries where the virus has been reported; and use certified healthy seeds. Frequent monitoring of plants, staff training on the rapid recognition of symptoms caused by this virus and on the disinfection operations suggested during agricultural activities, use of disposable clothing for operators, and sterilization of tools used are additional important measures. In case of suspicious plants, the greenhouse should be placed in isolation, and in case of positive diagnostic tests, all the eradication rules must be implemented, including the destruction of the material and the sterilization of the greenhouse structure, avoiding the cultivation of tomato or pepper for at least one cycle.

5.5. Tomato Mosaic Virus

Tomato mosaic virus (ToMV) is the second economically important tomato infecting virus within the genus *Tobamovirus* (family *Virgaviridae*). ToMV consists of rigid and rod-shaped particles measuring approximately 300×15 nm. Its genome consists of a non-segmented single-stranded positive RNA (ssRNA+) molecule of approximately 6.380 nt [232]. The genome encodes four proteins, among which the 184 and 126 kDa proteins are involved in viral replication. The 184 kDa protein is the only protein required for virus replication, whereas the 126 kDa protein contains a methyltransferase and a helicase domain and increases the efficiency of replication. The ORFs positioned at the 3'-end of the genomic RNA encode the movement protein (MP), involved in the cell-to-cell movement, and the coat protein (CP) [263]. The virions can be found in all plant organs, including the pollen and seeds, probably except from the embryo.

ToMV is spread throughout the Mediterranean region and other areas where tomato is cultivated [281–284]. It can infect several different species, but its main hosts belong to the family *Solanaceae*, mainly tomato and pepper plants, where the yield can undergo reduction between 25–70% [55,56].

On tomatoes, the symptoms may depend on pedo-climatic conditions, age of the plant, virus strain and tomato cultivar. Therefore, leaf green spot mosaic with yellow or white spots and discolouration of the leaf veins can be frequently observed (Figure 14).



Figure 14. Leaf green spot mosaic with yellow spots induced by Tomato mosaic virus.

After ToMV infection, tomato flowers can fall, significantly reducing fruit production. In the greenhouse, during summer, young leaves are deformed and small and may appear lightly spotted, curled, and blistered. During winter, leaves may be so reduced that only the main veins are formed. On both immature and ripe fruits, spot discolorations are noted, associated with necrosis and internal browning of the vascular tissue. Discoloured areas of the fruits can occasionally necrotize or develop depressions, and the fruits appear pitted [2]. In some cases, the spot discoloration caused by ToMV on fruit can be confused with a physiological disorder known as “blotchy ripening,” and sometimes the high temperatures can attenuate or mask the leaf symptoms. Moreover, in tomato cultivars with the *Tm2*- or *Tm2²*-resistance genes, at high temperatures, necrotic fissures can develop on leaves and sometimes on stems and leaf petioles. The production losses are considerable, depending on the infection precocity, and become irrelevant when the infected plant has already produced in 4–5 bunches.

ToMV is easily transmitted through tomato seeds (external contamination), but it can be found in smaller quantities even in the endosperm but never in the embryo [285]; this virus can remain active in the infected seed at least ten years. ToMV is very stable even outside of the plant and can contaminate surfaces and objects, remaining infectious for long periods. Furthermore, it can survive in the soil and other substrates for several years, particularly in leaves and roots residues. In tomato crops, ToMV spreads quickly from an infected plant to a healthy one only by leaf contact, and it can also spread through roots thanks to micro-wounds in the tissues. Moreover, ToMV can be easily transmitted from plant to plant during cultivation operations.

The presence of insect vector has not been demonstrated although aphid transmission has been reported through contaminated insect claws. The main diagnostic methods used to ascertain ToMV infection include biological assays; although laborious, they are indispensable for a clear classification of virus strains and isolates. Serological methods based on antisera obtained from purified virus or coat protein subunits can also be employed; commercial kits effective for the serological diagnosis of ToMV (e.g., ELISA kits, immunostrips) are available. However, only molecular, e.g., multiplex RT-PCR, real-time RT-PCR, or combined serological-molecular methods, such as immunocapture-reverse transcription-polymerase chain reaction-multiplex (IC-RT-PCR-multiplex), are capable of

discriminating ToMV from TMV and other tobamoviruses with very high sensitivity [230,286,287].

Prevention relies on the use of resistant cultivars: genotypes with the *Tm2²* gene are resistant to all the most common ToMV pathotypes, with the exception of the 2² pathotype (not very common in crops). To control the virus, it is necessary to use certified pathogen-free propagation material and to sterilize cutting tools during cultural and manipulation operations since the virus can be spread by contaminated seed and contact [288,289]. Furthermore, large crop rotations are recommended in soils free of vegetation residues and weeds that can be hosts of the virus. Thermotherapy of tomato seeds (70 °C for 24 h) or seed dressing (10% trisodium phosphate or a solution containing 2% hydrochloric acid and 0.3% pectinase) may be applied before sowing allowing the elimination of ToMV from infected seeds [290]. A recent study using the treatment of tomato plants with zinc oxide nanoparticles has been demonstrated to be efficient in boosting immunity against ToMV [291].

5.6. *Parietaria Mottle Virus*

Parietaria mottle virus (PMoV) was identified and characterised for the first time in 1987 in the Turin area (Piedmont region of Italy) on *Parietaria officinalis* L. plants that showed a marked leaf mottling [292]. A variant of the virus, named TI-1 (Tomato-Illarvirus-1), was identified in 1971 on tomato plants in the same geographical area [293]. PMoV is a member of the genus *Illarvirus* within the family *Bromoviridae*. It has a single tripartite positive RNA genome; the RNA1 is monocistronic and encodes the protein 1a, whereas the RNA2 is bicistronic and encodes a RNA-dependent RNA polymerase (RdRp) (protein 2a), and the protein 2b, which is expressed through a subgenomic RNA (RNA4). The RNA3 (2.700 nt) is bicistronic and encodes the movement protein (MP) and the coat protein (CP). The 1a and 2a proteins are involved in genome replication and internal transcription of the subgenomic RNA4 (1.100 nt) [294]. PMoV is constantly detected on tomatoes in some Mediterranean countries, such as Spain, France and Greece [58]. The disease incidence varies between 3 and 30%, but it is possible that up to now the real PMoV incidence has been underestimated, due to the confusion generated by similar symptoms caused by other viruses (TSWV, ToMV and CMV).

The most evident symptom on tomato consists in the alteration of the fruit pigmentation: at the beginning of the disease, the immature fruits show greenish translucent rings with more intense colour shades compared to the background, whereas the ripe and mature fruits show chlorotic rings. Within a short period of time, these rings become brownish, generally in relief, more or less corky and coalescent, causing the deformation of the fruits; some fruits can show the surface almost entirely covered by corky ring formations (Figure 15).



Figure 15. Typical corky rings on tomato fruits induced by *Parietaria* mottle virus.

The leaf symptomatology consists in the formation of irregular necrotic areas that are often coalescent and mostly concentrated in the basal part. In the severest cases, the stem apex necrotises and folds upon itself [2,295,296].

Mechanical transmission of this virus through pollen collected from PMoV-infected tomato plants was reported [297]. In tomato plants, the PMoV transmission was documented by several insects, including thrips and some species used in biological control, starting from infected *P. officinalis* plants at flowering [298]. The virus-contaminated pollen penetrates into the plant tissues through epidermal microlesions, which are formed by the frenetic trophic activity of the insects. On the other hand, virus transmission through seeds was not reported on tomato but occurs in the wild species *P. officinalis* (see below).

Serological and molecular detection methods of the virus are available such as DAS-ELISA, RT-PCR, real time RT-PCR and molecular hybridization [295,296]. A LAMP protocol is currently under validation, and this is expected to allow a direct in-field diagnosis. The PMoV natural host range appears very limited. *P. officinalis* and *Mirabilis jalapa* are considered as the major reservoirs plants of PMoV. In fact, *P. officinalis* plays a key role in PMoV epidemiology since it transmits the virus by seed; therefore, it is essential to eliminate these species when grown close to fields of tomato. Resistant plants could be used in

genetic improvement programs to introduce resistance against PMoV in tomato cultivars [2].

5.7. Pepino Mosaic Virus

Pepino mosaic virus (PepMV) is spread on tomato in the majority of the Mediterranean countries [226]. PepMV is a member of the genus *Potexvirus*, family *Alphaflexiviridae*, and its virions are non-enveloped flexible filaments of 470–580 nm with a diameter of 13 nm. The virus genome is a single-stranded, positive-sense, monopartite polyadenylated RNA of approximately 6.400 nt that encodes five ORFs [232]. The viral RNA molecule presents an untranslated region (UTR) both at the 5' and 3' ends. ORF 1 (164 kDa) encodes the RNA-dependent RNA-polymerase (RdRp), while ORFs 2, 3, and 4 that constitute the Triple gene block (TGB) encode the proteins TGBp1 (26 kDa), TGBp2 (14 kDa), and TGBp3 (9 kDa) involved in the inter-cellular movement. ORF 5 encodes the coat protein (CP) [299]. The replication of the virus genome occurs in the cytoplasm of the host cells. Tomato yield loss can account to about 30–80% [59,60,300]. There are currently five major strains of PepMV: (1) the Peruvian (PE) strain, originally found on pepino plants and on wild *Solanum* spp; (2) the EU-tomato (EU-tom) strain; (3) the US1/Ch1 strain; (4) the Chile-2 (Ch2) strain; and (5) the PES strain (recently isolated from wild tomato populations in Peru) [301]. The PepMV host range is quite limited, considering that the virus mainly infects pepino (*Solanum muricatum* Aiton) and tomato plants. In laboratory, mechanical inoculation experiments proved that PepMV can also infect plants belonging to the family *Solanaceae*, such as potato, eggplant, tobacco, and cucumber. In Italy, it was detected also in basil and in *Petunia × hybrida* Hort. [299] besides tomato. Recently the virus has been found in Spain on amaranth, European black nightshade, mallow, and common sow thistle.

The symptomatic expression of the virus varies due to presence of different strains, to the environmental conditions and seasonal changes [302], and to the physiological state of the plant.

Symptoms generally appear on tomato plants 2–3 weeks after infection and spread within the row. Plants often exhibit dwarfism, and the younger apical leaves may show a widespread chlorotic mosaic, while the lowest show brown or necrotic lesions. If the infection occurs early, a few days after transplantation, the virus can induce symptoms similar to those caused by herbicides and/or by inappropriate hormonal treatments, such as the formation of leaflets with reduced and incised flap (Figure 16); late infections also cause symptoms on fruits, with discoloured spots of marble appearance, often accompanied by necrotic pitting. Other leaf symptoms include scalloping of the leaf margin accompanied by blistering or pale-coloured specks that evolve into bright yellow, angular patches. Additional symptoms include brown streaks that can surround the whole branch, the floral cluster, and the fruits. The fruit cup may show redness with consequent fruit dropping and loss of production. The most typical fruit symptoms are an uneven ripening, a marbled appearance, and occasionally necrotic lesions with cracks (Figure 16). A strong golden pit may be observed on tomato cherry cultivars [2,59,60,300–302].

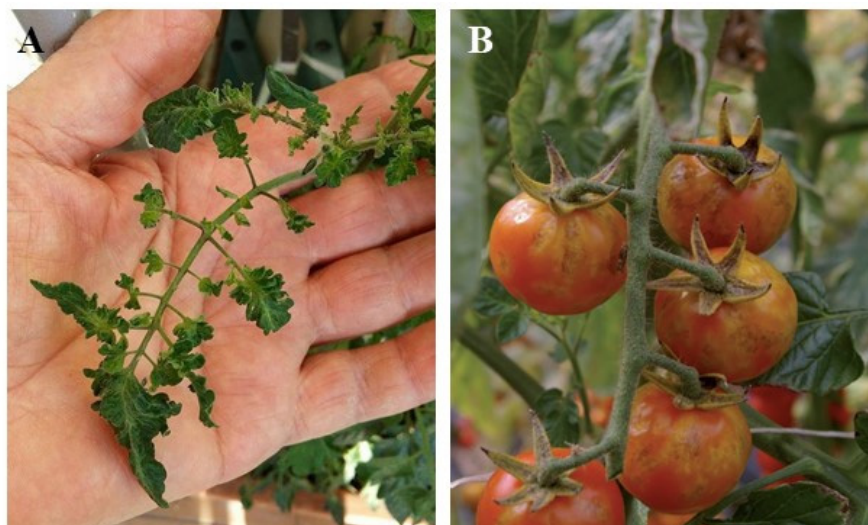


Figure 16. Severe symptoms induced by Pepino mosaic virus on leaves. (A) Leaflets with reduced and incised flap. (B) Fruits with blotchy ripening and marbling.

PepMV is highly contagious and it is mainly transmitted by contact between infected plants or mechanically through contaminated work tools, shoes, clothes and workers' hands, where the virus can remain infectious for approximately 14 days. Furthermore, PepMV can remain vital for about 4 weeks also in the dry plant debris and in tomato roots. The virus incubation period is approximately 10 days, depending on the environmental conditions and the virus load. In addition, some pollinator insects (e.g., *B. terrestris*) can indirectly transmit the virus. Experiments conducted in Italy confirmed a high transmissibility through bumblebees. In Spain, PepMV transmission was demonstrated through the fungus *Oplidium virulentus* [301]. The seed transmission rate is very low, but this way of dispersion should not be underestimated due to the high infectiousness of the virus and its danger in greenhouses. Moreover, this virus can be transmitted by grafting.

Virus detection must be carried out using laboratory analyses since the clearest symptoms occur on fruits and only during certain periods of the year and, moreover, symptoms are not always specific. Good results can be obtained by DAS-ELISA allowing the processing of a large number of samples, at low cost [303]. Molecular diagnosis includes virus-specific detection techniques such RT-PCR [230] and real time RT-PCR [304,305] or other RT-PCR and RT-LAMP techniques which allow differentiating among PepMV genotypes [306,307].

The PepMV is regulated by the European Decision 2004/200/EC which prohibits introduction and transport of contaminated tomato seeds, recommending severe inspections and controls on seeds imported from third countries, as well as the monitoring of infections along the production chain (seeds, nurseries, cultivation sites, markets) by member states. Since its appearance in Europe, PepMV has been included in the A2 EPPO quarantine list [172].

For adequate control, a series of strategies based on integrated control must be implemented, such as using healthy and certified propagation material; preventing the access of vector insects in protected cultivation areas; cleaning of greenhouses, clothing, and cutting tools; destroying plants affected by the virus; placing mats impregnated with disinfectant at the entrance of greenhouses; and allocating workers and means of work to a specific section of the farm to minimize virus spread. Soil solarization is also an effective tool. Dry-heating (72–74 °C for 2–3 days) or disinfection (0.5–1.0% sodium hypochlorite and 10% trisodium phosphate) of tomato seeds may be efficient to eliminate PepMV from contaminated seeds [308,309].

6. Viroid Diseases

6.1. Potato Spindle Tuber Viroid

Among viroids, Potato spindle tuber viroid (PSTVd) presents a major threat to tomato and is listed in the A2 EPPO quarantine pathogen in Annex IAI of the Directive 2000/29/EC of the European Union. It is the type species of the genus *Pospiviroid*, the family *Pospiviroidae* [310]. Originally isolated from potato plants affected by the degeneration disease, it was the first viroid disease to be recognized and investigated by phytopathologists [311]. PSTVd presents a circular, non-coding RNA genome of 359 nt that replicates and spreads systemically in host plants. All functions necessary to establish an infection are mediated by sequence and structural elements within its genome [312].

PSTVd is reported sporadically in European and West Asian Mediterranean countries, while in North Africa, it is reported until now only in Egypt. Yield losses due to PSTVd infection on tomato may reach up to 50–90% depending on the cultivar [62].

Disease development on tomato greatly depends on the temperature; low temperature (around 15 °C) strongly inhibits the disease development, while the pathogen is highly stimulated at high temperatures (25–34 °C) and with dry climates and low humidity (30–40%) [63–65,313]. Moreover, a high light intensity is favourable for disease progression, especially if combined with high temperatures [63,313].

PSTVd initially induces growth reduction with possible apical chlorosis, which may lead to severe stunting, growth arrest (Figure 17), and occasional death of the plants [311,314].



Figure 17. Extreme stunting and growth arrest in tomato plants artificially infected by Potato spindle tuber viroid. (A) Healthy plant. (B) Infected plant.

Downward curling of leaves and leaf chlorosis are frequently observed, and leaves progressively may show vein necrosis. Apical leaves exhibit size reduction, epinasty and rugosity. Tomato fruits of infected plants, if produced, may be smaller compared to healthy counterparts, sometimes showing deformation or discolouration [63,310,315].

Although a wide spread of PSTVd infections has never occurred on tomatoes (it has been eradicated from five EU countries) [316], a high risk of disease spread always persists due to the PSTVd transmission by seeds, aphids (in coinfection with Potato leafroll virus), mechanical transmission by contaminated working tools during cultivation activities, and due to the recent identification of this viroid in ornamental plants, mainly asymptomatic [311,317–320].

Disease management strategies consist in the use of certified planting material, elimination of infected plants or any suspected infected source, control of aphid vectors and

spontaneous plants, hygiene practices and disinfection of cutting tools and agricultural machines between different crops, early diagnosis of the pathogen and symptom monitoring [311,316,321].

7. Effect of Climate Change on Tomato Diseases

Individual and combined weather elements deeply influence the disease occurrence and pathogen infections, each playing an important role. In recent times, climate change has emerged as another major threat for vegetable crops, such as tomato, affecting plant-pathogen interactions and disease epidemiology. Climate change has important effects on this crop, both directly (morphological, physiological and phenotypic changes, plant productivity) and indirectly (temperature increase, water availability for irrigation purposes, drought, soil salinization, soil fertility, change in tomato rhizosphere microbial communities, pest incidences) [322,323]. For this reason, agrometeorological studies and an effective environmental management will be required to overcome this challenge in order to predict new disease outbreaks and protect and/or improve the worldwide food production with human interventions, adaptation, and mitigation strategies. Nonetheless, it is particularly complex to predict the effects of climate change on plant-pathogen interactions. To date, most studies have been carried out under controlled conditions on a small scale and for relatively short periods of time and therefore in situations that may vary greatly from those in the field [324]. The prediction of the plant-pathogen interaction under a projected climate time series can be assessed by modelling the behaviour of the pathogen and/or its epidemics under known conditions. In addition, the available climate models do not include important parameters, such as different agronomic practices, possible adaptations of pathogens/plants, effects on antagonistic population towards a specific pathogen, and the difficulty of applying climatic data series to the field, all of which are necessary in order to construct reliable empirical models [324].

As reported by Sturrock et al. [325], climate change affects the life cycles of pathogens and hosts by changing the distribution and phenology of events (e.g., budbreak, release of pathogen spores, activities of vectors, etc.), thus increasing the difficulties to perform simulation modelling. The major meteorological factors related to climate change that may influence the plant-pathogen interactions are air and soil temperature, CO₂ concentrations, relative humidity, rainfall, wind, and intensity of solar radiation [326], with temperature, relative humidity, and CO₂ concentrations being the most important ones.

In this scenario, the recurrent presence of warmer autumns and mild winter temperature is extremely important for pathogens transmitted by vectors (e.g., plant viruses), such as aphids (worldwide spread throughout all temperate areas), whiteflies (confined to warmer regions), and leafhoppers. In this context, climate change may affect the spread of plant viruses, influencing not only primary infections but also the horizontal transmission to new hosts [327]. The global temperature increase greatly affects the life cycles of both aphids and whiteflies due to shorter developmental times and high number of generations per year [328]. On the other side, it was shown that viral infection can mitigate the heat stress response of tomato, reducing disease symptoms and yield losses [329].

Plant host phenology and physiology may also be affected by climate change, modifying the susceptibility to the pathogen and the pathogen's ability to infect, affecting the attractiveness of the host to vector, the transmission modes, the geographic range of potential vectors, and/or their phenology (overwintering, density, migration, etc.) [330].

The rise in temperatures due to climate change will actually see an increase in crop production by lengthening growing seasons in temperate regions. On the other hand, an increase in frequency of outbreaks, introduction of pathogens to new areas, and plant disease intensity are also foreseen [331]. As for tomato cultivation, the length of the growing seasons mainly concerns open field cultivation, as greenhouse cultivation (in many cases) provides continuous crop cycles. In the latter case, the main threat can be represented by the impact of vectors, which find ideal conditions and cause new infection outbreaks. For this, crop rotation strategies could prevent the development of pests and diseases and

mitigate climate change effects while helping the economic livelihood of farmers by reducing the risks associated to monoculture [332].

Climate change can have considerable effects on the rising of emergent plant pathogens also by altering their temporal and spatial distributions [333], causing substantial physiological alterations in plants [334]. Indeed, in the last decades, there is rising evidence of new records of plant pathogens described. Recent reports have shown increasing disease severity associated with climate change for necrotrophic plant pathogens, such as *A. alternata*, *Cercospora* sp., *Colletotrichum gloeosporioides*, *F. equiseti*, *F. fujikuroi*, and *F. oxysporum* f. sp. *conglutinans* [334–336]. Regarding this review, seven of the tomato plant necrogenic pathogens described, i.e., *Bc*, *Forl*, *Vd*, TSWV, ToMV, PepMV, and PSTVd, have been also reported for their increased pathogenicity or enhanced transmission rates, [19,20,61,63] while up to now, no reports are available for ToBRFV and PMoV.

In the context of climate change, emerging pathogens may appear as exotic (new) or re-emerging (native) pathogens [337]. All the above described pathogens stimulated by climate change can be considered as “re-emerging pathogens” on native hosts since they have been present in the Mediterranean region for decades. These specific pathogens should be monitored with particular attention. On the other hand, ToBRFV and PMoV are emerging exotic viruses in many Mediterranean countries, and for their management and risk analysis, it is crucial to verify if their pathogenicity is exacerbated by climate change. Other pathogens described in this review, although not reported to be stimulated by climate change, are economically very important in the geographical area considered, and their increase is possibly related to other trends, such as globalization of the seed industry and recent phytopathological measures.

Overall, tomato growers need to develop alternative pest management strategies not only to face the emergence of pathogens and pests directly due to climate change but also because of the resulting alteration of their biological control agent populations and predatory organisms [338,339]. High humidity favours the development of fungal and bacterial diseases, such as *A. solani*, *B. cinerea*, *S. lycopersici*, and *P. syringae* pv. *tomato* [14,40,87,340]; high temperatures may stimulate the development of fungal, viral, and viroid diseases, in particular *B. cinerea*, *F. oxysporum* f. sp. *radicis-lycopersici*, TSWV, ToMV, PepMV, and PSTVd [19,26,48,57,63]; and increased CO₂ levels have been shown to exacerbate disease caused by *B. cinerea* [21]. Therefore, the application of various measures aimed to minimize the potential impact of climate change is of utmost importance for a successful disease management in the future. To combat climate change, recently issued directives and policies have recommended tomato growers to reduce the loss of nitrogen synthetic fertilizers and irrigation water. However, these requirements are not easily applied in outdoor cultivation. A pioneering work on open-field hydroponics in horticulture has been initiated by incorporating substrate bags to the field and applying drip irrigation [341]. Other methodologies that might help reduce the water and nutrient losses in open field consist of no-tillage or reduced tillage, mulching, use of biofertilizers and biostimulants, high-density planting, grafting with rootstocks tolerant to nutrient- and water-deficit, use of anti-transpirant compounds and slow-released fertilisers, and application of computational decision models and precision agricultural techniques [342]. Conversely, in protected cultivation, the control of water and nutrient restrains seems to be much more feasible. This may be achieved by the use of automated techniques and through the application of nutrient and water sensors in a soil cultural system or in hydroponics, aeroponics, or aquaponics soilless system [343,344]. Moreover, a selection of new “short-stature” tomato cultivars adapted for the growth in a multi-tiered, narrow-rack, indoor conditions [345] or the creation of small tomatoes suitable for vertical farming using the CRISPR-Cas technology are new inputs that may satisfy the requirements of advanced and sustainable tomato cultivation. Since tomato plants grow mostly outdoors in the Mediterranean region (e.g., open field vs. protected cultivation 5.7/0.5 million tonnes in Italy, 3.3/1.6 million tonnes in Spain) [342], the main impact of climate change on tomato is still to be considered directly in open field. Temperature increases, heat waves, more frequent extreme

temperatures, severe air droughts, and water deficits are the main traits that endanger horticulture production in this area [346]. Although in protected cultivation facilities (greenhouses and screenhouses), water and nutrient supply may be more easily controlled, and the impact of climate change is not as direct as in outdoor cultivation, it is still of high concern. In fact, inner temperature and humidity increase is also altered by external climate change. Adaptation strategies may include cooling, dehumidification, ventilation and use of shading curtains, light-emitting diode (LED) lighting, and use of dye-sensitized solar cells with solar energy collectors [347–349]. Although elevated CO₂ concentration may increase tomato yield, the control of CO₂ in greenhouses and screenhouses is also necessary since it may reduce the content of citric, malic, and oxalic acids and increase the sugar content, having an impact on the final fruit taste [350,351]. In winter production conditions, it is important to improve the use of natural light and to provide additional lighting through a sawtooth roof, anti-reflectance coatings, and LED lamps [348]. Finally, vertical farming may be considered a favourite cultivation facility in the future thanks to the overall control of internal factors and the use of sensors and automation systems. However, in vegetable cropping systems, these strategies are currently rare because of high economic inputs and production costs, limited evidence in scalability, and lack of available precise metrics and standards [352].

Additional measures to contrast climate change encompass the adoption of cultural methodologies, including the change of sowing and harvesting calendar and the selections of species/cultivars with a shorter cultivation period or with traits of resistance/tolerance to drought, high temperature, and salinity. Another possibility is the adaptation of technical measures, which may influence soil erosion and rainwater conservation. Finally, economic measures, through the introduction of various farmer financial incentives and other specific political decisions [353], may help in coping with the adverse effects of climate changes. All of these strategies are expected to help the management of plant diseases, besides the classical preventive and curative control strategies.

8. Future Prospects

Horticulture plays a key role in the economy of countries with a temperate climate. Unfortunately, horticultural crops are affected by several diseases, compromising the economic return of the farmers. In this context, better crop protection strategies require a close synergy between the research community, seed companies, and farmers, trying to fulfil needs of consumers interested in healthy products of high quality.

Here, we have outlined the major and most dangerous pathogens that affect tomato crop in the Mediterranean area and their effects on the cultivation of this important horticultural crop. Considering the tremendous impacts of pesticides on water quality and plants and animals and their linkage with several negative effects on human health, including short-term sickness and several kind of cancers; sustainable agricultural practices, such as crop rotation, precocious detection and prompt eradication of infected plants; and use of resistant varieties, certified propagation material, or chemical prophylaxis against insect vectors, are all strategies useful to contain pathogen infections and reduce the use of pesticides. Nonetheless, innovative strategies of pathogen containment must be adopted at the global level to implement a transition towards sustainable agricultural practices and sustainable living behaviours based on lower pesticide inputs and reduced food loss and food waste. Promising strategies also include the adoption of innovative cultural practices and innovative lighting sources to optimize plant growth. In particular, the technologies based on LEDs are particularly attractive for indoor cultivation of horticultural crops, eliciting not only photomorphogenic and biochemical traits but also physiological responses suitable to counteract pathogen attack [354].

From the biotechnological point of view, one of the biological mechanisms useful to obtain resistance to pathogens relies on RNA-mediated resistance and is specifically based on post-transcriptional gene silencing (PTGS), taking part in the natural and complex pro-

cess universally known as RNA silencing or RNA interference (RNAi) [355]. In this context, new genetic engineering techniques are available to induce plant defence against pathogens, through the so-called Host-induced gene silencing (HIGS), making use of RNAi-based gene constructs to introduce stable genetic resistance in plants by triggering PTGS. Such gene constructs normally include short-inverted sequences homologous to pathogen genes, usually split by a non-coding sequence, such as an intron. As a whole, these so-called hairpin RNA (hpRNA) constructs are under the control of specific promoters and terminators [356,357].

Recently, as an alternative tool to the stable integration of hpRNA-based gene constructs in plants, a new technique named *Spray-induced gene silencing* (SIGS) is being explored. SIGS consists in the topical application of small RNA molecules (sRNAs) to plants. SIGS has great and innovative potential for crop defence against different plant pathogens and pests and is expected to raise less public/political concerns compared to the use of plants genetically modified with hpRNA constructs, as it does not alter the genetic structure of the plant. After the first pioneering work by Tenllado and Díaz-Ruiz [358] reporting the successful application of exogenous dsRNAs against three different viruses, all with a positive, single-stranded RNA genome, several studies have been conducted also on viruses with different genomic structures, as recently reviewed in Dalakouras and coworkers [359].

Recent developments in gene-editing techniques through clustered, regularly interspaced, short palindrome repeats/CRISPR-associated protein (CRISPR/Cas) technologies based on the bacterial immune system are being implemented to obtain plants resistant to fungal, bacterial, and virus diseases [360–365]. The systems based on CRISPR/Cas can be used both to mutate host susceptibility genes involved in a specific interaction with pathogen infection or used to directly target the pathogen genome [366,367]. Encouraging results have been already described, and this system is recently exceeding other genome-editing techniques due to its versatility, velocity, cost-effectiveness, and successfulness. It is predicted that CRISPR/Cas system together with omics techniques will take the major role in the creation of pathogen-resistant plants, yield-increased cultivars, and those resistant to abiotic stresses as well, after overcoming the legislation barriers [368]. Currently, the CRISPR/Cas genome editing has been applied to obtain resistant tomato plants or to decrease pathogen virulence in the case of *F. oxysporum*, *B. cinerea*, *O. lycopersici*, *P. capsici*, *P. syringae* pv. *tomato*, TYLCV, and TYLCSV, either targeting tomato genes (e.g., *SIM1o1*, Solyc08g075770, *SIMAPK3*) or pathogen genes (e.g., *PKS4*, CP, IR, and Rep sequences) [368–375]. In addition, a promising strategy relying on gene editing of the Susceptibility downy mildew resistance 6 (*DMR6*), conferring broad-spectrum resistance to fungi and bacteria, has been recently reported [376]. Nonetheless, the novel availability of tomato transcriptomes obtained following pathogen infection through next-generation sequencing is expected to produce new insights into common molecular response and resistance against pathogens. Therefore, key genes involved in the response to biotic stresses will be targeted through gene-editing techniques to incorporate new sources of resistance, namely those based on plant innate responses [377].

Thanks to advances in sequencing and synthetic biology, new detection frontiers are being opened in diagnostics, relying on the exemplary step that coupled isothermal technologies with CRISPR/Cas enzymes. In this context, it is noteworthy to mention that such techniques have been recently adopted to set up innovative, rapid, and specific next-generation detection methods for tomato plant viruses [378,379].

In addition, further exploitation of new biological control measures (biocontrol agents) [380] through plant priming for resistance, exploitation of nutrient competition, hyper-parasitism, and antibiosis will certainly be boosted in the future as a valid alternative useful to reduce the impact of chemically synthesized pesticides. In particular, a large number of microorganisms have been identified and evaluated for their advancement as biocontrol agents against tomato diseases, as recently reviewed by Karthika et al. [381]. The use of plant growth-promoting rhizobacteria as biological control agents is not only

effective in triggering growth promotional effects but has been shown to reduce the impact of several disease in tomato through various mechanisms, including antibiosis, competition with pathogenic organisms, secretion of compounds that promote plant growth, direct inhibition of the development of the pathogens (siderophores, cell wall lytic enzymes, hydrogen cyanide, as an example), or induction of systemic acquired resistance. However, to maximize the commercial exploitation of plant growth-promoting rhizobacteria and of biological control agents, field and greenhouse trials with tomato plants treated with combinations of inoculants have to be implemented for a fruitful application in industrial agriculture.

There are also recent trends regarding the harnessing of plant microbiomes, allowing to elucidate the plant resistance against pathogens, which will certainly contribute to obtaining resistant tomato plants [382,383].

9. Conclusions

In this review, we have described the most harmful pathogens (fungi, bacteria, phytoplasmas, viruses, and viroids) that affect tomato production in the Mediterranean basin. Due to the profound impact of climate change, the recently revised lists of available pesticides and the market globalization of seed trade, further boost in the spread of these pathogens is expected, additionally aggravating the intrinsic low genetic diversity of the tomato crop, making it extremely susceptible to pathogen attack. Altogether, this imposes the implementation of new breeding strategies, the adoption of rapid diagnostic procedures, and a wide range of sustainable and high-tech agricultural practices to counteract the economic fallouts in this crop.

Supplementary Materials: The following are available online at www.mdpi.com/2073-4395/11/11/2188/s1, Table S1: List of abbreviations and acronyms used in this review.

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