Major Diseases of Tomato, Pepper and Eggplant in Greenhouses

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ABSTRACT

Greenhouse climatic conditions provide an ideal environment for the development of many foliar, stem and soil-borne plant diseases. In the present article, the most important diseases of greenhouse tomato, pepper, and eggplant crops caused by biotic factors are reviewed. Pathogens that cause serious yield reduction leading to severe economic losses have been included. For each disease that develops either in the root or aerial environment, the causal organisms (fungi, bacteria, phytoplasmas, viruses), main symptoms, and disease development are described, as well as control strategies to prevent their widespread outbreak. Since emerging techniques for the environmentally friendly management of plant diseases are at present imperative, an integrated pest management approach that combines cultural, physical, chemical and biological control strategies is suggested. This review is based on combined information derived from available literature and the personal knowledge and expertise of the authors and provides an updated account of the diseases of three very important Solanaceaous crops under greenhouse conditions.

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Invited Review

INTRODUCTION

The cultivation of crops in the greenhouse is the most intensive form of horticultural production (Albajes *et al.* 1999). Investment and labour costs are greater in this sector than in any other; however, the yield, quality and value of crops are correspondingly high (Albajes *et al.* 1999). Insects and diseases are a major challenge to greenhouse production. In such an intensive cropping system several parameters (high humidity and temperature, high plant density, air-exchange restriction) favour the development of a large number of bacterial, fungal, phytoplasma and viral diseases as well as insect vectoring viruses. The resulting microclimate in the greenhouse is favourable to disease epidemics and once inside a greenhouse, pathogens and pests are difficult to eradicate. Successful crop production requires that crop pests and diseases be managed so that their effects on the plants are minimized. Losses may be high if control strategies are not implemented in time. The management of crop diseases aims to prevent the establishment of diseases and minimize their development and spread. Managing disease problems focuses on preventing pathogen populations from becoming too large and uncontrollable. Growers must use all available options and strategies to avoid serious pest and disease problems. Integrated pest management (IPM) is a term used to describe an evolving process where cultural, biological, and chemical controls are included in a holistic approach of pest and disease control.

In the present up-to-date paper, we review the symptoms and conditions that favour the development of the most important diseases of three major greenhouse Solanaceous vegetable crops (tomato, pepper and eggplant) and we outline an IPM-based preventive and therapeutic approach for their successful control.

FUNGAL DISEASES

Late blight

Late blight, the disease that was responsible for the infamous Irish potato famine of the 1840s, is a highly devastating disease affecting both tomato and potato. It can also infect eggplants. The disease is caused by the fungus-like oomycete pathogen *Phytophthora infestans* (Mont.) de Bary. It can infect and destroy the leaves, petioles, stems and fruit of tomato plants and cause 20-70% crop loss (Fry and Goodwin 1997; Knapova and Gisi 2002).

Symptoms
In tomato, leaves and young shoots are infected first. Leaf lesions appear as indefinite, water-soaked spots that can very rapidly enlarge into pale green to brown lesions covering large leaf areas. Fruit lesions appear as dark, olivaceous spots that can cover the entire fruit. The disease can also spread on tomato fruit after harvest. Under wet conditions, the undersides of leaf lesions or the fruit spots can be covered with a velvet white to grey mycelium that consists of the branched conidiophores that emerge through the stomata (Panagopoulos 2000). The pathogen overwinters in volunteer or abandoned tomato plant material in greenhouses or the surrounding area (Vartanian and Endo 1985; Cohen *et al.* 1997). *P. infestans* is mostly active during cool, moist weather; i.e. cool nights and warm days are favourable for late blight development (**Fig. 1A**) (Jones *et al.* 1991; Legard *et al.* 1995; Koike *et al.* 2007).

Disease development
P. infestans can be dispersed by water splash or wind currents for up to several kilometres from the overwintering site to tomato plant tissues via the asexual fruiting bodies, called sporangia (Fry *et al.* 1992). Sporangia are formed at a relative humidity (RH) of 91-100% and temperatures of 3-26°C, with an optimum of 18-22°C. Sporangia can germinate within a few hours after landing on tomato foliage if free moisture is present on the plant tissues. Germination occurs either indirectly via zoospores or directly via a germ tube that penetrates into the foliage, stems, or fruit to initiate infections. The first small lesions are visible after three to four days. The pathogen can sporulate on lesions that are only four to six days old under favourable environmental conditions (leaves wet for more than 10-12 hours at moderate temperatures (15-21°C), an environment quite common under greenhouse conditions). Tomato stems can also be infected, and stem lesions are capable of producing sporangia for a longer time than lesions on the leaves. Disease development (growth and reproduction of the pathogen) is favoured by moderate temperatures (15-25°C) and wet conditions, whereas temperatures above 30°C are not favourable. When the weather conditions and cultural practices (i.e. sprinkler instead of drip irrigation) are ideal, epidemics can be rapid and devastating because of the high reproductive potential of this pathogen. Individual lesions can produce 100,000 to 300,000 sporangia per day. Affected plants during an epidemic appear as though they have been damaged by frost. Thus, the rapid reproduction of the pathogen can defoliate tomatoes and completely destroy healthy fields in a short time (Jones *et al.* 1991; Fry *et al.* 1993; Goodwin 1997; Albajes *et al.* 1999; Rubin *et al.* 2001; Koike *et al.* 2007).

Control
Use of integrated management practices (IPM) is neces-
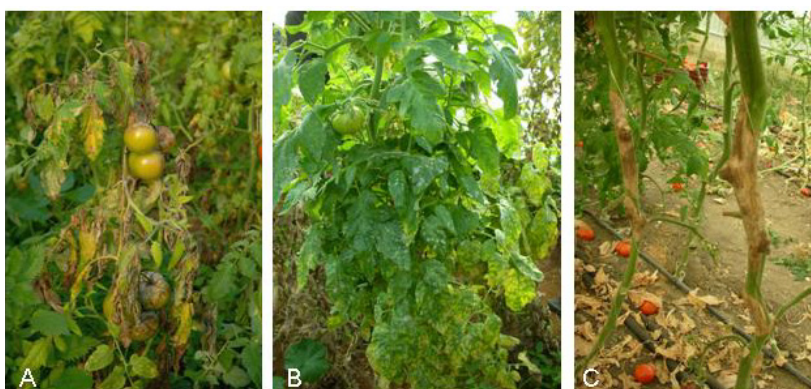
Fig. 1 (**A**) Late blight of tomato caused by the oomycete *Phytophthora infestans*. (**B**) Powdery mildew of tomato caused by the fungus *Leveillula taurica* that grows endophytically (yellow spots on lower leaves) and by *Oidium* spp. that produce epiphytic, conidiophore-bearing mycelium that grows superficially on host surfaces (white spots on upper leaves). (**C**) Gray mold of tomato caused by the fungus *Botrytis cinerea*. (Photograph **B** courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

sary for the successful suppression of late blight. Cultural control measures, such as sanitation strategies that eliminate or exclude infected plant parts from the greenhouse or the surrounding areas, are important in the overall management strategy, while the application of fungicides plays the major role in the control of the disease. Fungicide applications can be adjusted on the basis of forecasting systems that identify favourable weather conditions for disease development. Early in the season, prior to late blight infection, protectant fungicides can provide protection and thus prevent a rapid epidemic. Fungicides with systemic activity penetrate into the plant tissues and are necessary if a crop has been exposed to sporangia within the previous 24 hours. Dithiocarbamates, chlorothalonil, copper fungicides and the systemic phenylamides (metalaxyl etc.) are the most commonly used in greenhouses. Once 5-10% of the foliage has become infected, it is usually not possible to halt the development or progress of the disease. There are some tomato cultivars with strong to moderate resistance to late blight, but the appearance of new pathogen races that break the resistance is very common (Cohen and Grinberger 1987; Jones *et al.* 1991; Albajes *et al.* 1999; Tumwine *et al.* 2002; Koike *et al.* 2007; Chen *et al.* 2008).

Powdery mildew

Powdery mildew can cause 10-90% yield loss in greenhouse tomato, pepper or eggplant crops. The extent of loss depends on the environmental conditions, date of disease onset, and the effectiveness of fungicide control. Hot, dry days with some free water are conducive to disease development (Fletcher *et al.* 1988).

Symptoms
There are two different types of powdery mildew fungi that infect tomato and eggplant: the one is caused by *Leveillula* taurica (Lév.) G. Arnaud (anamorh: *Oidiopsis taurica*), which grows endophytically, and the other is caused by one or more species of *Oidium* (e.g. *O. neolycopesrici*, *O. lycopersici*), which produce epiphytic, conidiophore-bearing mycelium that grows superficially on host surfaces (**Fig. 1B**) (Arredondo *et al.* 1996; Kiss *et al.* 2005). Pepper is only infected by *L. taurica*. The first symptoms of *L. taurica* occur on lower older leaves as bright yellow spots with diffuse margins that progressively enlarge and eventually become necrotic. As infections progress, the entire leaf withers and dies but remains attached to the stem in tomato, whereas in the case of pepper the leaf falls off the plant. The underside of the leaf spot is usually covered by a white powdery growth of the pathogen consisting of conidiophores that emerge through the stomata. Young leaves escape infection until they mature. There are no symptoms on the stems or fruit. However, with extensive loss of foliage, many fruits may be exposed to sunburn. *O. lycopersici* poses a significant threat to glasshouse-grown tomatoes and is of increasing importance in field-grown tomato crops. It mainly causes white, powdery lesions on the adaxial tomato leaf surface. The fungus can also infect petioles, stems and the calyx, but the fruit remain uninfected. Severe infections lead to leaf chlorosis, premature senescence and a marked reduction in fruit size and quality (**Fig. 1B**) (Correll *et al.* 1987; Jones *et al.* 1991; Albajes *et al.* 1999; Whipps and Budge 2000; Pernezny *et al.* 2003; Koike *et al.* 2007).

Disease development
Powdery mildew fungi are obligate pathogens and survive on overwintering tomato, alternate hosts, or possibly as cleistothecia from their respective perfect stages. Asexual spores are airborne and disease development is favoured by mild temperatures below 30°C. Repeated cycles of powdery mildew can lead to severe epidemics that economically damage the crop (Correll *et al.* 1988a, 1988b; Jones *et al.* 1991; Whipps *et al.* 1998; Panagopoulos 2000; Pernezny *et al.* 2003; Koike *et al.* 2007).

Control
Disease monitoring, early detection and prevention of powdery mildew is critical for the successful control of the disease. The greenhouse crops need to be monitored from the beginning of cultivation. The practice of strict hygiene throughout the year is necessary. Once the disease is detected, the application of preventive fungicides, such as sulphur, demethylation inhibitors (DMIs: triadimefon, fenarimol, etc), pyrimidines (ethrimol, bupirimate, etc.), pyrazophos and dinocap, is necessary. If greenhouse crops are severely infected with powdery mildew it is very difficult to control the disease and the crop can be entirely destroyed (Jones *et al.* 1991; Pernezny *et al.* 2003; Koike *et al.* 2007).

Grey mold

Grey mold, caused by the fungus *Botrytis cinerea* Pers.:Fr., has a wide host range, with over 200 hosts. The pathogen can cause damping-off, as well as blight of the flowers, fruits, stems, and foliage of many vegetables and ornamentals. It is a very common disease of Solanaceous crops that can be particularly damaging in greenhouse environments because of the high RH. It is a major cause of post-harvest rot at harvest and in storage (Ferrer and Owen 1959; Coley-Smith *et al.* 1980).

Symptoms
Grey mold can occur on all above-ground plant parts, while stem lesions on seedlings can occur at, or just below, the soil level. Dark brown lesions progressively expand to include the whole leaf, grow along the petiole and finally reach the stem. These lesions may girdle the entire petiole or stem, causing wilting of the plant above the lesion. Entry to the stem may occur through senescent or damaged tissue (**Fig. 1C**). Senescent petals are quite susceptible to grey mold infection and the fungus can easily grow from the petals to sepals and into the developing fruit. Fruits that are attached to diseased foliage may develop rotted areas. Infections on green or ripe fruit typically show soft, decayed, circular rot than can damage the entire fruit. The fungus sporulates profusely on the fruit calyx, or in the center of the fruit lesion where the skin ruptures, and appears as a grey, velvety or fuzzy mold. White, distinct, circular (halo) spots called "ghost spots" may appear on the fruit. These spots occur when a pathogen spore germinates and invades the fruit but then the mycelium dies prior to causing decay. The halo forms around the point of entry. As the fruit ripens, the colour of the halos changes from white to yellow. Although no rot ever occurs with ghost spots, the many halos can cause the fruit to lose market quality (Ferrer and Owen 1959; Coley-Smith *et al.* 1980; Jones *et al.* 1991; Shtienberg *et al.* 1998; Panagopoulos 2000; Pernezny *et al.* 2003; Koike *et al.* 2007).

Disease development
Botrytis cinerea survives as sclerotia or as mycelium in plant debris of the crop or weeds, or in the soil. Conidia from these sources are carried on to the host surface by wind or splashing rain drops. High RH is necessary for prolific spore production. Spores germinate and produce an infection when free moisture (rain, dew, fog, or irrigation) occurs on the plant surface. Optimum temperatures for infection are between 18-24°C, and infection can occur within 5 hours. High temperatures (>28°C) suppress growth and spore production. Dying flowers are a favourable site for infection, but infections can also result from direct contact with moist, infested soil or plant debris. Stem lesions develop either by direct colonization of wounds or through infected leaves (Ferrer and Owen 1959; Coley-Smith *et al.* 1980; Morgan 1984; Jones *et al.* 1991; O'Neill *et al.* 1997; Park *et al.* 1999; Pernezny *et al.* 2003; Koike *et al.* 2007).

Control
There is no known resistance to *B. cinerea* in plant crops. Under greenhouse conditions, effective management

can be achieved by avoiding the conditions that favor grey mold development (high RH and cool temperatures), by adequate ventilation, careful handling to prevent wounding, and removing inoculum sources through adequate plant sanitation. Fungicides might also need to be applied to protect the fruits from grey mold. It is critical to use a diversity of fungicides with different modes of action because the pathogen can easily develop resistance (Fletcher and Scholefield 1976; Leroux *et al.* 2002). Treatment of the crop before the build up of infection might be necessary, especially when cool and humid, or wet conditions prevail (Coley-Smith *et al.* 1980; Jones *et al.* 1991; Utkhede *et al.* 2001).

Early blight

Early blight, caused by the fungus *Alternaria solani* Sorauer (syn. *Alternaria tomatophila*) is an important disease of tomato and eggplant in humid climates or in semi-arid areas where adequate moisture permits disease development. The disease primarily affects leaves and stems, which under favourable weather conditions, if left uncontrolled, can result in considerable defoliation, yield reduction and sunburn damage of the exposed fruits (Jones *et al.* 1991).

Symptoms

During the first stages of the disease, the lesions are small, brown to black and oval or angular in shape, and may be surrounded by chlorotic tissue on mature leaves. As the disease develops, spots enlarge and can be 8-10 mm or more in diameter and contain the characteristic concentric rings, giving a 'target-spot' effect. Stem infections consist of small, brown, sunken lesions that can expand, elongate and girdle the stem, resulting in stem and plant death. Seedlings can also be infected at the soil level when they are less than 3 weeks old, and develop collar rot. Plants with collar rot can become stunted, wilt and die. Fruit infections, at either the green or mature stage, consist of dark brown to black, circular, sunken spots with concentric rings. Lesions may be covered by a velvety mass of black spores. Infected fruit frequently drops, and losses of 30-50% of immature fruits may occur (**Fig. 2A, 2B**) (Panagopoulos 2000). Early blight symptoms on green fruit and stems may resemble infections caused by the *Alternaria* stem canker pathogen, *Alternaria alternata* f. sp. *lycopersici* (Pennypacker 1983; Jones *et al.* 1991; Patterson 1991; Koike *et al.* 2007).

Disease development

Alternaria solani survives between crops on infected greenhouse crop debris and on tomato or eggplant seeds, which also serve as sources of primary inoculum. Additionally, the pathogen survives on volunteer plants and other Solanaceous ho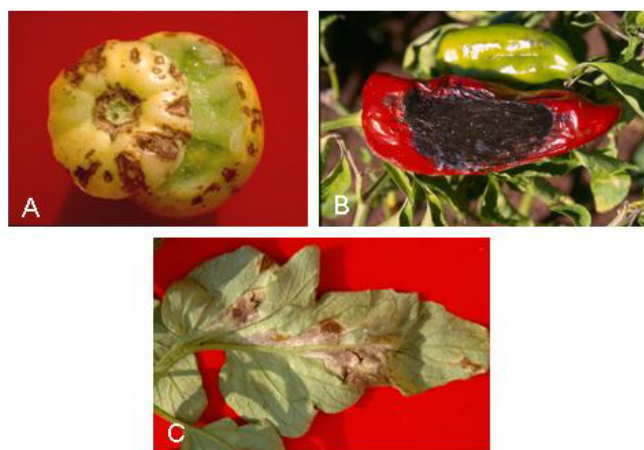sts. The disease is favoured by mild (24-29°C), rainy weather, although in warm climates the disease may also be quite active at higher temperatures. Conidia disseminate via wind and splashing water. The fungus can penetrate the leaf surface directly through the cuticle or enter through wounds and spots begin appearing within 2-3 days. Lesions are more abundant on lower, older, and less vigorous leaves and on early maturing cultivars (Pennypacker *et al.* 1983; Jones *et al.* 1991; Patterson 1991; Koike *et al.* 2007).

Control

Resistant or tolerant cultivars are available. Seeds need to be pathogen-free; in case of infected seeds, treatment with hot water or fungicides can eliminate the pathogen. Regular inspections, eradication of weeds and of volunteer tomato or eggplant plants, removal of symptomatic transplants, and long crop rotations might be necessary for successful disease management. A complete and regular foliar fungicide spray program is usually required to control this disease (Vakalounakis 1991; Brammall 1993; Mills *et al.* 2002) or a combination of minimal spraying and other control practices.

Alternaria stem canker

Alternaria stem canker can be a serious problem in greenhouse tomato crops. The pathogen (*Alternaria alternata* (Fr.:Fr.) Keissl. f. sp. *lycopersici*) can infect all parts of the plant above ground.

Symptoms

Dark brown to black cankers, with concentric rings, form on the stems. These cankers can continue to enlarge and eventually girdle the stem, killing it or the entire plant. A brown, dry rot develops, as well as brown streaks that can extend into the vascular and pith tissue above and below the canker. Infected leaves develop irregularly shaped, dark brown to black areas that are primarily interveinal. Interveinal necrotic leaf lesions are due to a host-specific toxin (AAL-toxin, analogs TA and TB), which is produced by the fungus and moves systemically from the stem canker to the leaves (Gilchrist and Grogan 1976; Witsenboer *et al.* 1992; Koike *et al.* 2007). Fruit symptoms initially develop on green, unripe fruit and appear as dark brown sunken lesions with the characteristic concentric zonation. This green fruit disease is different from black mold disease, which affects only ripe fruit and is caused by a different pathogen, *A. alternata* (Grogan *et al.* 1975; Malathrakis 1983; Jones *et al.* 1991).

Disease development

The pathogen can survive in the soil and infected tomato debris for up to a year. Conidia are easily spread by the wind and require free moisture on the tomato plant to germinate. Rain, overhead irrigation and heavy dew favour the development of the disease. The optimum temperature for disease development is 25ºC. Entry of the fungus into the stem is facilitated by pruning cuts or other wounds; however it can also infect healthy, uninjured plants (Malathrakis 1983; Jones *et al.* 1991; Koike *et al.* 2007).

Control

Resistant cultivars have been successfully bred for the control of Alternaria stem canker. If using susceptible varieties, it is advised not to use irrigation with overhead sprinklers and to rotate crops. Use of registered and effective fungicides can also control this disease (Vakalounakis 1988; Witsenboer *et al.* 1989; Koike *et al.* 2007).

Anthracnose

Anthracnose is caused by several species of the fungus *Colletotrichum*: *C. acutatum* J.H. Simmonds, *C. coccodes* (Wallr.) S.J. Hughes, *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz, *C. dematium* (Pers.) Grove, *C. capsici* (Syd)

Fig. 2 Early blight of tomato (**A**) and pepper (**B**) caused by the fungus *Alternaria* sp. (**C**) Leaf mould of tomato caused by the fungus *Fulvia fulva*. (Photograph **B** courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

Butler & Bisby (Freeman *et al*. 1998). The disease has its greatest impact on the fruit of tomato, pepper and eggplant. It can be a serious disease of processing tomatoes (caused by *C. coccodes*), and tomato processors impose a strict limit on the amount of anthracnose acceptable on the raw product (Batson and Roy 1982; Prusky *et al*. 2000; Pernezny *et al*. 2003).

Symptoms

The disease primarily infects the fruit. Young and green fruits may be infected but disease symptoms are not developed until the fruits begin to ripen. Fruits become increasingly susceptible as they approach maturity. Early symptoms appear on ripe fruit as small, sunken, water-soaked, circular spots that increase in size (12-15 mm in diameter) and contain concentric rings. The lesion centres are usually tan but become black from the fungal structures (microsclerotia and acervulli) that form in the tissues. Under humid conditions, pink-colored spores are released from the fruiting bodies in these lesions. As the fungus spreads within the fruit, a soft decay occurs and secondary organisms can grow, rotting the fruit completely. Infected fruit have a very short storage life. Infection may also occur on vegetative parts, such as stems, leaves, and roots. Leaf infections show small, tan to brown spots that often have a yellow halo in tomato plants, or a dark brown border in pepper plants. Infected roots in tomato exhibit brown lesions and as the root cortex tissue collapses, the black microsclerotia of the pathogen develop abundantly, giving this stage of the disease the name black dot root rot, which is part of the brown root rot disease complex that occurs in greenhouse-tomatoes in Europe (Jones *et al*. 1991; Manandhar *et al*. 1995b; Prusky *et al*. 2000; Pernezny *et al*. 2003; Koike *et al.* 2007).

Disease development

C. coccodes is most frequently associated with the fruit disease and seems to be the only causal agent of black dot root rot. The fungus survives in the soil or plant debris as microsclerotia or acervulli. It can also be seed-borne. In spring, the lower leaves and fruit can be infected by germinating sclerotia and windborne or water-splashed spores. The infections are important sources of conidia for secondary infections throughout the growing season. Optimum conditions for disease development are 20-24°C and wet, humid weather that favours the development of the fungal propagules (Dillard 1989; Manandhar *et al*. 1995a; Byrne *et al*. 1997, 1998; Dillard and Cobb 1998; Prusky *et al*. 2000; Pernezny *et al*. 2003; Koike *et al*. 2007).

Control

Rotation with non-Solanaceous crops at least every other year is recommended to prevent an accumulation of the fungus in the soil. Weed control is important since the anthracnose pathogens have a broad host range. Sprinkler irrigation should be avoided because of the spread of the conidia. Staking of plants and the use of mulch materials can reduce the number of fruit in contact with the soil. Fungicide treatments provide acceptable control if applied on a periodic basis from first fruit cluster to harvest. Especially when the fruit begin to ripen, fungicide applications are very important (Jones *et al*. 1991; Pernezny *et al*. 2003; Koike *et al*. 2007).

Septoria leaf spot

Septoria leaf spot, caused by the fungus *Septoria lycopersici* Speg., occurs worldwide. It is particularly severe in areas where wet, humid weather conditions persist for extended periods. The fungus infects only Solanaceous plants (tomato, potato, eggplant), of which tomato is the most important (Jones *et al*. 1991).

Symptoms

Septoria leaf spot can occur at any stage of plant development. Symptoms may appear on young greenhouse seedlings ready for transplanting or on the lower, older leaves and stems after the first fruit sets. Lesions consist of small, water-soaked circular spots (1.6-3.2 mm in diameter) surrounded by dark brown margins and tan to grey centres, dotted with black pycnidia. Spots can enlarge to about 6.4 mm in diameter and may coalesce. Septoria leaf spot lacks the target-like lesions that are typical of Alternaria blight. Spots may also appear on stems, calyxes, and blossoms, but fruit infection is rare. The disease spreads upwards, from the oldest to the youngest growth. Heavily infected leaves will turn slightly yellow, then brown, wither and drop off. This defoliation may result in fruit sunscald (Jones *et al*. 1991).

Disease development

The fungus overwinters on debris of diseased plants incorporated in the soil and on Solanaceous weeds. Seeds have been shown to carry spores (conida) and produce infected seedlings. Under wet conditions, numerous conidia are produced and exuded from the mature pycnidia. Conidia germinate in the presence of free moisture and penetrate leaf tissue through the stomata. First symptoms appear 5-6 days after inoculation, followed by pycnidia (spore-bearing, fruiting structures) 12-14 days after inoculation. The optimum temperature range for infection, symptom development and sporulation varies from 20-25°C. Spores may be spread by splashing water, wind-blown water, insects, cultivation equipment or the hands and clothing of farmers (Barksdale 1982; Jones *et al*. 1991).

Control

Use of healthy seeds and elimination of the initial sources of inoculum can greatly reduce the extent of disease. Good sanitation practice is also critical. Greenhouse production areas should be free of susceptible Solanaceous weeds and crop debris from the previous season. Registered protective fungicides sprays at regular intervals during the growing season can effectively control Septoria leaf spot. The first sprays are usually needed when early blight control begins and are normally applied on a 7-10 day schedule unless disease pressure is high (Jones *et al*. 1991).

Leaf mold

Fulvia fulva (Cooke) Cif. (syn. *Cladosporium fulvum*) is the causal organism of tomato leaf mold, a serious disease of greenhouse-grown tomatoes especially under high RH. Generally, only the leaves are affected by the fungus, although occasionally the stems, blossoms, petioles and fruit are also attacked (Jones *et al*. 1991; Thomma *et al*. 2005).

Symptoms

The older leaves are first attacked and the initial symptoms are pale green or yellow spots on the upper leaf surface. At the corresponding site on the lower leaf surface, an olive green mold, which consists of the conidiophores of the fungus, develops (**Fig. 2C**). Under severe infection, these spots coalesce and the leaves might curl, wither and drop from the plant. Green and mature fruit can develop a black, leathery rot at the stem end, with as much as one third of the fruit affected (Jones *et al*. 1991; Panagopoulos 2000; Thomma *et al*. 2005).

Disease development

Disease development is optimal between 22-24°C, but it may occur between 4-32°C. Spore germination needs free water and it can not occur if the RH is less than 65%. The pathogen survives as a saprophyte on crop residues, or in the form of conidia or sclerotia. The conidia of *C. fulvum* are generally spread by wind or water splash and can survive for at least one year. The conidia of the fungus can infect successfully if they settle on the abaxial surface of a leaf, germinate, and subsequently enter through open stomata. Contaminated seeds can serve as a source of inoculum (Small 1930; Jones *et al*. 1991; Thomma *et al*. 2005).

Control

Crop residues should be removed and destroyed after harvest. Fungicides effectively control the disease. To reduce leaf mold severity, it is advised to minimize long periods of leaf wetness by avoiding watering of the foliage and by using a good ventilation system. Cultivars that have resistance to *C. fulvum* have been developed, but usually the pathogen is able to overcome this resistance (Jones *et al*. 1991; Stergiopoulos *et al*. 2007).

Grey Leaf Spot

Grey leaf spot can commonly be found in pepper and tomato and is caused by *Stemphylium solani* G.F. Weber and *S. lycopersici* (Enjoji) W. Yamamoto. It has a worldwide distribution and can be a very destructive disease, leading to severe defoliation of the crop (Jones *et al*. 1991; Pernezny *et al*. 2003).

Symptoms

Grey leaf spots are limited to the leaves and only under very favourable conditions can symptoms be observed in the petioles or young parts of the stems. Leaf lesions initially appear as barely visible, small, reddish-brown to black spots. These lesions enlarge to form spots with white centres and red to brown margins in pepper, or a yellow halo in tomato. Eventually, the center of the spot dries out and develops cracks. If numerous lesions develop, the leaves turn yellow, die rapidly, and drop, leading to severe defoliation of the plant. Young plants at the seedling stage are most sensitive to infection (Weber 1930; Hannon and Weber 1955; Rotem *et al*. 1966; Jones *et al*. 1991; Pernezny *et al*. 2003).

Disease development

The pathogens survive on crop residues and in the soil as saprophytes. They can also be seed-borne. In addition, volunteer or other Solanaceous crops, weeds and infected transplants serve as inoculum sources. Wind and splashing water spread conidia to the host. Warm (24-27°C) and humid or wet weather, such as those that occur in greenhouses, are favourable for disease development. These fungi can produce extensive growth during a single night and can develop rapidly within the host (Weber 1930; Hannon and Weber 1955; Rotem *et al*. 1966; Jones *et al*. 1991; Pernezny *et al*. 2003).

Control

Use of pathogen-free seeds and of resistant cultivars is recommended if available, while crop rotation and good sanitation practices can reduce the incidence of disease. Fungicides must be used for disease control when susceptible varieties are grown (Jones *et al*. 1991; Pernezny *et al*. 2003).

Phytophthora root rot

Phytophthora root rot is a widespread and devastating disease of Solanaceous crops, especially under greenhouse conditions.

Symptoms

Phytophthora parasitica Dastur (= *P. nicotianae* var. *parasitica*), *P. cryptogea* (Pethyb.and Laff.) and *P. capsici* Leonian are the main causal organisms of Phytophthora root rot. A root and crown rot extending to a considerable height above the soil level may occur (**Fig. 3A**). The infected area has water-soaked lesions with a dark discoloration, and the pith is usually destroyed. Infected tissues shrink and in humid weather a white mycelium develops. Infected plants wilt and die quickly (**Fig. 3B**). These pathogens can also infect tomato fruit and cause field and post-harvest fruit rots (**Fig. 3C**) (Stevenson 1991; Ristaino 2003).

Fig. 3 Phytophthora root tot of tomato (**A**) caused by the oomycete *Phytophthora parasitica* and pepper (**B**) caused by the oomycete *Phytophthora capsici* (**C**). Tomato fruit rot caused by *Phytophthora* spp. (Photograph **B** courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

Disease development

Phytophthora reproduces by both sexual and asexual means. Oospores, the sexual spores, overwinter in the field and serve as inoculum. The disease is polycyclic. The pathogen produces two mating types. Both mating types are common in fields, and both have been identified on a single plant. Phytophthora produces antheridia and oogonia (male and female gametangia, respectively). Meiosis occurs within the gametangium, and plasmogamy and karyogamy result in the formation of oospores. Oospores germinate either directly by forming a germ tube or indirectly by producing sporangia. Sporangia can germinate directly to produce hyphae or else can differentiate to produce 10-30 zoospores. Zoospore production is typically triggered by flooding. Zoospores are generally short-lived (hours) and quickly differentiate to form adhesive cysts, which in turn germinate to produce hyphae or a secondary zoospore (Tyler 2007). Zoospores are the most important form of inoculum for infection of roots, especially when the soil is flooded. In many species, zoospores swim chemotactically toward compounds released by roots of their host plants (Tyler 2002). The zoospores encyst on the root surface, from where the hyphae penetrate the root directly. Zoospores, as well as sporangia, can also be spread to the upper plant by splashing. The pathogen reproduces asexually by forming sporangia, which are borne on branched sporangiophores, The sporangia are dispersed by wind, rain and irrigation water, releasing the zoospores (Tyler 2007).

Control

Management of Phytophthora blight mainly relies on cultural practices, crop rotation, and selective fungicides. Raised beds, good drainage of the soil bed and subsurface drip irrigation can help manage the disease. Fungicides, such as phenylamides (e.g. metalaxyl, mefenoxam), may help manage root and fruit infections. A complication is that many oomycetes appear to have an extraordinary genetic flexibility which enables them to adapt rapidly, and overcome, chemical control measures and genetic resistance bred into plant hosts (Schmitthenner 1985; Schettini *et al*. 1991; Fry and Goodwin 1997; Chamnanpunt *et al*. 2001).

Verticillium wilt

Verticillium wilt is caused by the soilborne pathogens *V. dahliae* Kleb. and the less frequently encountered *V. alboatrum* Reinke & Berthier. The incidence and severity of the disease vary from year to year and from one location to another, depending on host susceptibility, pathogen virulence, soil type and environmental conditions.

Fig. 4 Verticillium wilt of eggplant (**A, B**), tomato (**C**) and pepper (**D, E**) caused by the soil-borne fungus *Verticillium dahliae*. Symptoms consist of yellowing of foliage (**A, B**), varying degrees of vascular discoloration (**C**), wilt as a result of water stress (**D**) and stunting growth (**E**). (Photographs **C**, **D**, **E** courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

Symptoms

The first symptoms of Verticillium wilt are stunting and a slight yellowing of the lower foliage (**Fig. 4B, 4E**). As the disease progresses, leaves may become more severely chlorotic (**Fig. 4A**). Varying degrees of vascular discoloration may occur (**Fig. 4C**) and plants wilt as a result of water stress (**Fig. 4D**). For a few days, infected plants may recover at night, before permanent wilting sets in and the plants die (Goldberg 2003).

Disease development

Verticillium survives in the soil and crop debris mainly as microsclerotia. Microsclerotia may remain dormant in the soil for more than ten years and are mainly stimulated to germinate by root exudates. The infectious hyphae that emerge from microsclerotia penetrate roots principally in the areas of cell differentiation and in the root hair zone (Schnathorst 1981). Subsequently, the pathogen invades and colonizes the xylem elements, thereby disrupting water transport. Verticillium diseases generally spread through the use of contaminated equipment, the transfer of contaminated soil, irrigation, and the use of infected seed or plant materials, such as rootstocks, bulbs and tubers.

Control

The control of the pathogen is difficult due to the long viability of the resting structures, the broad host range and the inability of fungicides to affect the fungus once it enters the xylem. Therefore, the removal of plants once they are diagnosed with Verticillium wilt disease is currently the most effective means of disease control. Resting structures are desirable targets for Verticillium control because they produce the primary inoculum and are very persistent (Hawke and Lazarovits 1994). Inoculum reduction can be accomplished through soil solarization, chemical soil fumigation or crop rotation. However, for various reasons these strategies are rather inefficient. Soil solarization can only be carried out in geographical areas that have the required climate, and the use of chemicals for fumigation is severely restricted because they are generally detrimental to the environment and/or public health. Finally, crop rotation is rather unappealing as Verticillium resting structures are persistent in the soil, requiring long rotations with mainly monocotyledonous crops to reduce microsclerotia below crop-specific threshold levels (Wilhelm 1955; Evans *et al.* 1967; Fradin and Thomma 2006).

Therefore, the most feasible and economic control is the use of Verticillium-tolerant cultivars. Polygenic resistance to *Verticillium* spp. has been identified in several plant species, including alfalfa, cotton, potato and strawberry (Hunter *et al.* 1968; Simko *et al.* 2004; Bolek *et al.* 2005). Single dominant resistance genes have been identified in cotton, sunflower, potato and tomato (Schaible *et al.* 1951; Lynch *et al.* 1997; Jansky *et al.* 2004). In tomato, the *Ve* locus that provides resistance against Verticillium wilt (Schaible *et al.* 1951) has been used by plant breeders for 60 years and introduced in most cultivated tomatoes. Isolates of *V. dahliae* and *V. albo-atrum* that are contained by the *Ve* locus are designated as race 1, while all other isolates are designated as race 2 (Pegg 1974). Although some studies do not record any Verticillium growth in *Ve*-carrying tomato plants (Williams *et al.* 2002), others note that initial colonization of resistant and susceptible tomato cultivars with race 1 *V. dahliae* is similar (Chen *et al.* 2004). According to the latter study, the fungus enters the xylem and attempts to spread. At this stage, in resistant tomato a rapid coating response prevents fungal proliferation and elimination occurs similarly as in susceptible plants (Gold and Robb 1995; Chen *et al.* 2004). However, whereas in susceptible plants the fungus recovers and starts spreading again, resulting in a cyclical colonization (Heinz *et al.* 1998), in resistant plants the fungus does not substantially overcome the elimination (Gold and Robb 1995; Chen *et al.* 2004). Only recently, the tomato Ve locus has been fully characterized (Kawchuk *et al.* 2001). Positional cloning revealed the presence of two closely linked genes, Ve1 and Ve2, encoding leucine-rich repeat (LRR) proteins that belong to the class of so-called receptor-like proteins (RLPs; Kruijt *et al.* 2005).

Fusarium crown and root rot

This Fusarium disease is caused by the fungus *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *radicis-lycopersici* W.R. Jarvis & Shoemaker, a close relative of the Fusarium wilt pathogen *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *lycopersici* (Sacc.) W.C. Snyder & H.N. Hans. The pathogen damages mostly tomato but it has also been reported in eggplant and pepper and can be particularly severe in greenhouse production systems.

Symptoms and disease development

In plastic greenhouses, a yellowing and chlorosis of the lower leaves appears in infected plants during late winter, when many fruits have already set. In severe infections the whole plant becomes chlorotic and wilts. A dry lesion up to 10 cm long appears on part of, or all around, the collar. There is a brown discoloration of the root system, predominantly at the end of the main root, the base of the stem and the vascular region of the central root. The discoloration does not extend beyond 10-30 cm above the soil line and this feature helps in distinguishing this disease from Fusarium wilt, in which the vascular browning can extend far into the upper stems. A large number of microconidia, which disseminate the pathogen, are produced in the infected stem. The fungus is a soil inhabitant and survives by chlamydospores which develop in the soil (Malathrakis and Goumas 1999).

Control

The infected plants must be carefully removed and destroyed. Effective control of the fungus may be obtained by soil disinfestation, crop rotation, the use of resistant cultivars and by grafting on resistant rootstocks (McGovern 1993).

Fusarium wilt

The most common Fusarium wilt in greenhouses appears on tomato plants. The causal microorganism is *Fusarium oxysporum* Schlechtend.:Fr.f. sp. *lycopersici* Sacc.) W.C. Snyder & H.N. Hans. On the contrary, there are very few convincing cases of a Fusarium vascular wilt in *Capsicum* spp., and other *Solanum* species.

Symptoms

The initial symptom is chlorosis of the lower leaves that often begins on one side of the plant followed by wilting of that foliage. Infected leaves later show curling, browning and drying. As the disease progresses, the entire plant turns chlorotic, wilts and then collapses and dies. Vascular brown discoloration can be seen in infected stems and large leaf petioles. Affected plants and their root systems are stunted. The degree of stunting depends upon the time of root infection (**Fig. 5A**). The overall symptoms are similar to those caused by Verticillium wilt.

Disease development

As a soil inhabitant, *F. oxysporum* f. sp. *lycopersici* can survive for extended periods in the absence of the host, mainly in the form of thick-walled chlamydospores. Indeed, once an area becomes infected with *F. oxysporum*, it usually remains so indefinitely (Agrios 1997). The proximity of roots and the release of root exudates induces the dormant propagules to germinate and initiate infection. After germination, infection hyphae adhere to the host roots (Bishop and Cooper 1983b; Di Pietro *et al.* 2001) and penetrate them directly (Rodriguez-Gálvez and Mendgen 1995). The mycelium then advances intercellularly through the root cortex until it reaches the xylem vessels and enters them through the pits (Bishop and Cooper 1983b). At this point, the fungus switches to a highly peculiar mode of infection, during which it remains exclusively within the xylem vessels, using them as avenues to rapidly colonize the host (Bishop and Cooper 1983a). This is mainly accomplished by the production of microconidia, which are detached and carried upward in the sap stream. The microconidia germinate and the mycelium penetrates the upper wall of the vessels, producing more microconidia in the next vessel.

Control

The management of Fusarium wilt in greenhouses is possible through prevention, using resistant cultivars, dis-ease-free seedlings, new growth media for each crop, good sanitation practices and by maintaining optimum growing conditions.

White mold

This is a common greenhouse disease that damages eggplant, tomato, pepper throughout the world and is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary and *S. minor* Jagger.

Symptoms

White mold is usually first noted on tomato at about the time of flowering. Infection normally begins in the leaf axils or in stem joints where flower petals have fallen and lodged. Water soaked areas develop at these sites. The stems are subsequently invaded and become soft, and eventually large portions of invaded tissue may die. Infected stems are bleached light grey. If infection occurs at the crown or in the lower parts of the main stem, collapse of the canopy and foliage may occur (**Fig. 5B**). Fruit can also be infected and develop a soft, watery rot.

Disease development

The pathogens produce sclerotia on white mycelial mats 7-10 days after infection. *Sclerotinia sclerotiorum* produces sclerotia up to the size of bean seeds, whereas *S. minor* produces smaller sclerotia. Sclerotia fall on to the soil where they can survive for several years. When weather conditions are favourable, they germinate to produce apothecia which release ascospores and cause new infection. High RH and moderate temperatures are required for infection (Pohronenzy 1991; Malathrakis and Goumas 1999).

Control

Control of white mold is based on the application of appropriate fungicides (triazoles, benzimidazoles, dicarboximides, strobilurins) prior to *Sclerotinia* colonization of senescent and dead tissue, or soil disinfection with steam, heat or chemical treatments.

Pythium damping-off

Several *Pythium* spp. (*P. aphanidermatum* (Edson) Fitzp., *P. arrhenomanes* Drechs., *P. debaryanum* Auct. non R. Hesse, *P. myriotylum* Drechs., *P. ultimum* Trow) may attack tomato, eggplant and pepper plants during their early stages of growth, causing seed rot, pre-emergence seedling damping-off, post-emergence seedling damping-off, or stem rot. The attack may cause stand losses and uneven growth.

Symptoms

Pre-emergence damping-off is the most common symptom associated with an attack by *Pythium* spp. Usually, the post-emergence phase of the disease begins as a dark-colored, water-soaked lesion on the root, which extends up the stem to or above the soil line. When a dark-colored and soft lesion develops around a major portion of, or the entire, stem, the seedling falls over, withers and dies (McCarter 1991a).

Disease development

Pythium spp. are spread by sporangia, each of which releases hundreds of zoospores. Zoospores that reach the plant root surface encyst, germinate and colonize the root tissue by producing hyphae. These hyphae release hydrolytic enzymes to destroy the root tissue and absorb nutrients. *Pythium* spp. survive in the soil as saprophytes and most are favored by wet soil conditions and cool temperatures (15-20°C) The irrigation system in greenhouses contributes to the fast development and spread of *Pythium* spp. They form oospores and chlamydospores on decaying plant roots which can survive prolonged adverse conditions and lead to subsequent infections.

Fig. 5 (**A**) Fusarium wilt of tomato caused by the soil-borne fungus *Fusarium oxysporum* f.sp. *lycopersici.* (**B**) White mold of tomato caused by the soil-borne fungus *Sclerotinia sclerotiorum.* (**C**) Corky root rot of tomato caused by the soil-borne fungus *Pyrenochaeta lycopersici*.

Control

Pythium diseases are controlled primarily by cultural and chemical practices. Plants should be placed on raised beds and in well-drained soils. Seed treated with a fungicide mixture for seed-borne and soil-borne pathogens should be used, and plants should be grown under optimal temperature, moisture, and nutritional conditions (McCarter 1991a). Post-planting applications of fungicide may provide control.

Rhizoctonia damping-off

Rhizoctonia spp. infect a large number of plants, including tomato, pepper and eggplant. The pathogen mainly causes damping-off, root rot, and basal stem rot.

Symptoms

Germinating seedlings may be killed before or soon after they emerge from the soil. Before emergence the pathogen may kill the growing tip or produce tan to reddish brown lesions on the germinating seedling. After emergence, the fungus causes brown or nearly black lesions near the soil line. The young stem is constricted at the site of attack, becomes soft, and the plant falls over and dies. A root rot may occur as distinct, dark-colored lesions or brown rot under adverse soil or environmental conditions, or on plants whose roots are damaged. Rhizoctonia stem rot is mainly confined to the collar. Infected plants show pale brown, dry lesions, with circular rings, at the soil level. Growth is stunted and leaves become dull green. Complete wilting soon follows. Mycelial mat develop on the lesions and stems break easily at the infected site (Parmeter 1970; McCarter 1991b).

Disease development

Rhizoctonia spp. can survive indefinitely in soils by colonizing organic material and producing sclerotia. They may be dispersed with contaminated soil or on farm equipment. The fungus invades through wounds, but is capable of directly invading young, succulent tissue. It also produces mycelial mats that enhance the penetration of plant parts. The pathogen causes rapid tissue destruction by strong enzymatic action (Mc Carter 1991b).

Control

The pathogen is controlled primarily by cultural and chemical practices. Seed treated with a fungicide mixture that is active against seed-borne and soil-borne pathogens should be used, and plants should be grown under optimal temperature, moisture, and nutritional conditions.

Corky root rot

The soil-borne fungal disease, corky root, is caused by the fungus *Pyrenochaeta lycopersici* R. Schneider & Gerlach. The pathogen damages mostly tomato, but can also infect eggplant, pepper and other plants. The disease can be serious where the same crops are cultivated repeatedly in the same greenhouse.

Symptoms

First symptoms of the infected plants are poor vigor, stunted growth and wilt. Leaves may develop interveinal chlorosis and later fall off the plant. The most characteristic symptoms appear on larger roots and consist of brown lesions, with a rough corky or wrinkled texture. The developing infection leads to lesions with extensive cracks that run lengthwise along the root, progressively damaging the root system and resulting in disruption of nutrient and water uptake (**Fig. 5C**) (Goodenough and Maw 1973).

Disease development

Pyrenochaeta lycopersici is a soilborne pathogen that survives in the soil as microsclerotia, which are stimulated to germinate by root exudates of the host plant. The mycelia of the germinating microsclerotia attack the root system of the plant, causing rotting of smaller feeder roots, brown lesions on small roots and typical corky lesions on larger roots (Pohronezny and Volin 1991). However, it is not known whether corkiness is a response of the plant to the infection or a feature of the pathogen itself (Blancard 1992). The formation of brown lesions and subsequent loss of fibrous roots at an early stage of growth leads to severe losses in fruit yield; however, infected plants rarely collapse or die (Last and Ebben 1966; Hasna 2007).

Control

In conventional greenhouse crop production systems, soil fumigants such as chloropicrin and methane sodium have been used successfully against corky root disease (Punithalingam and Holliday 1973; Campbell *et al.* 1982; Malathrakis and Kambourakis-Tzagaroulakis 1989). In organic production systems, soil solarization is an effective alternative method for corky root management (Moura and Palminha 1994; Ioannou 2000). Crop rotation and delay of planting until later in the spring, when soils are warmer, can help reduce the yield loss. Grafting on to resistant rootstocks or the use of resistant cultivars can control the disease.

BACTERIAL AND PHYTOPLASMA DISEASES

Bacterial canker

Bacterial canker is one of the most destructive tomato diseases worldwide and is present throughout the tomato and pepper growing regions of the world, where it causes serious losses in both greenhouse and open field tomato crops. The disease is especially severe on transplanted or seeded tomatoes that have been clipped or pruned.

Symptoms

The initial symptoms of canker are the results of systemic wilt of the plant and, to a smaller degree, secondary infections of the parenchymatic cells of the upper portion of the plants (**Fig. 6A**). Young transplants may wilt suddenly and completely. On older plants, leaflets begin to turn brown at the edges, and then die back progressively toward the leaf midrib. Often only one side of a leaflet or plant develops symptoms first, but symptoms eventually spread. Stems may display external discoloured streaks, with stem cankers forming under some conditions. Rarely, cavities may develop within the stems, sometimes splitting open into brown, longitudinal cankers. However, if the infection begins in a wound caused by clipping, the disease can develop in the upper portion of the plant and rapidly move downwards, killing the plant. Internal vascular tissue becomes discoloured turning from light yellow to tan (**Fig. 6B**). As the disease develops, the vascular tissue turns dark brown to red-brown. The central pith tissue of the stem becomes discoloured (turning yellow or brown), separates from the adjacent vascular tissue, and forms hollows in the stem centre. In advanced stages of the disease, the pathogen reaches the plant phloem tissues and causes the plant to grow poorly, wilt and die (**Fig. 6A**). Leaves and stems can develop pale green to creamy white blister-like spots, which become encircled by dark rings of necrotic tissue. As the infection spreads, the leaflets may turn yellow, the veins darken and the spots on the young stems may turn to a tan colour. Fruit symptoms (when they occur) are quite distinctive spots: white and slightly raised, with dark-coloured centres and an opaque white halo. These spots are sometimes termed 'bird's-eye' lesions, giving the fruit a scabby appearance. The white halo turns brown as the spot ages. The vascular areas within the fruit leading to the seed may become yellow (Panagopoulos 2000; Koike *et al.* 2007).

Disease development

Bacterial canker is caused by the bacterium *Clavibacter michiganensis* subsp. *michiganensis* (Smith) Davis *et al.* (syn. *Corynebacterium michiganense*). It is an aerobic, non-

Fig. 6 (**A**) Bacterial canker of tomato caused by the bacterium *Clavibacter michiganensis* subsp. *michiganensis*. (**B**) Bacterial canker discoloration. (Photographs courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

spore-forming, Gram-positive bacterium. Cells may be pleomorphic in culture, but are rod-shaped when isolated from plants. Optimum growth occurs at 24-27°C. Reproduction is characterized by "snapping" division, resulting in V- and Y-shaped arrangements of the cells. Identification of the bacterium can be confirmed by classical and molecular diagnostic methods (Schaad *et al.* 2001; Janse 2005). *C. michiganensis* subsp. *michiganensis* is most important as a pathogen of tomato; however, it can infect other Solanaceous plants such as pepper, eggplant, *Nicotiana glutinosa* and other *Solanum* sp. The bacterium is mentioned in EPPO A2 quarantine lists (Janse 2005).

Primary inocula can come from many sources, including over seasoning in the soil, plant debris, infected volunteer tomato and/or other plants, infected weed hosts, contaminated wood stakes, diseased transplants and infested seeds. Secondary spread can occur by means of splashing water, contaminated equipment, via workers' hands or by pruning staked tomatoes and clipping transplants. In staked tomatoes, the symptoms may first be obvious on the stem where the string has caused an abrasion. In clipped or mowed transplants irrigated by overhead sprinklers, the latent period may be 3-6 weeks, which creates problems in plant certification programs that are based on visual appearance. Infested seed is a particularly important inoculum source because the bacterium can spread and disease can develop when plants are grown under greenhouse conditions. A 1% seed transmission rate is sufficient to result in 100% disease (Jones *et al.* 1992).

Control
The use of healthy seed and transplants is the most effective way to control the disease. Suspect seed should be disinfected by submersion in water at 52°C for 20 minutes, and with copper compounds (Fatmi *et al.* 1991). This method is also effective against *Pseudomonas syringae* pv. *tomato*, and *Pseudomonas corrugata*. Soil solarization for 6

weeks in the summer eliminates the bacterial population in the soil of the greenhouse or the open field, and a 2-3 year crop rotation with a non-host is encouraged. If this it is not possible, soil disinfection by solarization is recommended using transparent polyethylene soil coverage for 1.5-2 months. Under Mediterranean conditions, this effectively controls bacterial canker of tomato in the greenhouse (Antoniou *et al.* 1995). Sterilization of soil, potting mix, and pots or trays should be practised in greenhouse operations. Clippers and pruning tools should be disinfected and new or re-used stakes should be steamed or treated with a 1% chlorine-bleach solution. It is also essential that irrigation water should not pass through infected plantations, and Solanaceous weeds should be eliminated from the greenhouse and surrounding area. The spraying of plants with a Bordeaux mixture, or other copper compounds, after pruning may provide some disease control, while treatment of tomato plants with β-aminobutyric acid (BABA) offers protection against bacterial canker (Jones *et al.* 1992; Anfoka 2000; Panagopoulos 2000; Abbasi 2002; Kabelka 2002).

Bacterial speck

The disease is present in many countries. It has spread extensively and constitutes a very serious problem in greenhouse but also in open field tomato cultivation. It can cause complete loss of production when the infection is in transplants, while losses under mild infection can reach 12% (Jones *et al.* 1992).

Symptoms
The pathogen causes spots on stems (**Fig. 7B, 7D**), petioles, leaves (**Fig. 7A**), stalks, peduncles, sepals (**Fig. 7C**) and fruits. On leaflets, spots are round and dark brown to black (1-3 mm). The halo is lacking in the early stages but develops with time. The lesion extends throughout the entire leaf but is prominent on the abaxial surface. As the disease spreads spots coalesce in large areas of leaves (**Fig. 7A**). On fruits, minute dark lesions or specks might develop. The numerous specks that develop on young green fruit are slightly raised, rarely larger than 1 mm in diameter, and

Fig. 7 Bacterial speck of tomato caused by the bacterium *Pseudomonas syringae* pv. *tomato* on leaves (**A**), stems (**B**, **D**) and sepals (**C**). (Photographs courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

have well-defined margins. However, similar to bacterial spot and bacterial canker, the fruit spots are surrounded by a dark green to yellow halo. In some instances the spots are sunken. The specks are considerably smaller than the spots caused by bacterial spot, do not penetrate the fruit deeply, and can be scraped off with a fingernail. Although bacterial speck seldom reduces yields greatly, it can harm fruit quality (Panagopoulos 2000; Koike *et al*. 2007) (**Fig. 7**).

Disease development

This disease is caused by the bacterium *Pseudomonas syringae* pv. *tomato* (Okabe) Young *et al*. The bacterium is strictly aerobic, Gram-negative, and rod shaped. The bacterium also infects pepper and in artificial inoculations, eggplant. Strains of this pathogen can be host-specific to tomato, while other strains can infect both tomato and pepper. Two distinct races (races 0 and 1) have been documented. Identification of the bacterium can be confirmed by classical and molecular diagnostic methods (Schaad *et al*. 2001; Janse 2006).

The bacterium is seed borne and seedlings are very susceptible. Primary inoculum can also come from soil debris or reservoir plant hosts. Infected seed is a particularly important inoculum source because the pathogen can spread with seed and initiate disease when plants are grown under greenhouse conditions. The bacterium can be disseminated by overhead sprinkler irrigation, splashing water, contaminated tools and implements (clipping of transplants), and workers' hands. Infection is favoured by cool conditions (18-24°C), and high RH. *P. syringae* pv. *tomato* overwinters in seed, in plant debris (up to 30 weeks), in soil (less than 30 days), and on many other plants (Jones *et al*. 1992; Panagopoulos 1995; Koike *et al*. 2007).

Control

Greenhouse tomato crops should be monitored on a routine basis, so that early outbreaks can be controlled. Symptomatic plants should be removed. High-quality seed free of the bacterium should be used. Resistant varieties are not suitable for greenhouses (Roberts *et al*. 1999). Good greenhouse sanitation strategies are highly recommended: clean benches and trays that hold transplants, disinfected clippers, pruning shears, and other equipment that comes into contact with plants. Overhead sprinkler irrigation should be avoided. If the disease problem persists a rotation to a non-host crop should be employed. Destroying weeds around the greenhouse, field or garden will help to reduce survival of the causal bacterium.

Copper products can help control spread of bacterial speck if applications begin when the first symptoms appear or as preventative spray applications (copper plus maneb) to protect transplants. Copper plus mancozeb sprays can also provide some control. Copper-resistant strains of the bacterium have been found in some regions. Recently, a transgenic tomato plant variety Rio Grande with resistance to *Pseudomonas syringae* pv. *tomato* has been developed. Biological control is another strategy for controlling bacterial speck using a non pathogenic epiphytic bacterium that is resistant to copper, or a plant growth-promoting bacterium *Azospirillum brasilense*. Biological control is applied in combination with copper compounds and has given encouraging experimental results (Jones *et al*. 1992; Panagopoulos 2000; Bashan and de-Bashan 2002; Koike *et al*. 2007).

Bacterial spot

Bacterial spot is present worldwide wherever tomatoes and peppers are grown. The disease is most serious in tropical and subtropical regions of high humidity and rainfall. In greenhouses, the disease is much less damaging (Janse 2005).

Symptoms

On leaves, stems and fruits, spots appear that are small (up to 5 cm), circular to irregular in shape, and have a

Fig. 8 (**A**) Bacterial spot of pepper caused by the bacterium *Xanthomonas campestris* pv. *vesicatoria*. (**B**) Bacterial wilt of tomato caused by the bacterium *Ralstonia solanacearum*. (**C**) Tomato big bud or Stolbur caused by a phytoplasma similar to those causing aster yellows. (Photographs courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

slightly greasy feel (**Fig. 8A**). Unlike similar-sized spots caused by the fungus *Septoria lycopersici*, those caused by the bacterial spot pathogen do not develop greyish-brown centres. As the lesions enlarge, they often become surrounded by a yellow halo. If the spots are numerous, they begin to grow together and form significant necrotic areas on the leaves, leading to defoliation in severely infected seedlings. While infected leaves may show some chlorosis, the individual spots are not usually surrounded by yellow halos, although a general yellowing may occur on leaflets with many lesions. Long dark streaks may develop on petioles and stems. In severe cases, bacterial spot can result in stunted growth, a scorched appearance of the foliage, defoliation, and reduced yield. Fruit symptoms are more distinctive than leaf or stem symptoms. Spots on green fruit first appear as black, raised, blisters surrounded by water-soaked areas. As the spots enlarge to 5-8 cm they become grey-brown and scabby with sunken, pitted centres (Jones *et al*. 1992; Panagopoulos 2000; Pernezny 2003; Koike *et al*. 2007).

Disease development

Bacterial spot is caused by the bacterium *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye; genetic group A infects pepper while *Xanthomonas vesicatoria* genetic group B infects tomato. The bacterium is strictly aerobic, Gram-negative, rod-shaped and possesses a single polar flagellum. Xanthomonad pathogens from tomato and pepper hosts are a complex group of organisms. The bacterium is seed-borne and results in the infection of tomato seedlings. Identification of the bacterium can be confirmed by classical and molecular diagnostic methods. The bacterium is mentioned in EPPO A2 quarantine lists (Schaad *et al.* 2001; Pernezny *et al*. 2003; Janse 2005).

The bacterium overwinters on the surface of seeds, in infected debris, in reservoir hosts, and in the soil. Infested seed is an important inoculum source for the survival and dissemination of the pathogen, which is commonly brought into the greenhouse or field via infected transplants. Disease infection is favoured by warm (24-30°C), humid conditions. The bacterium is disseminated within the field by water droplets, clipping of transplants, and aerosols. It penetrates through stomata, or enters through insect puncture or mechanical injury (Jones *et al*. 1992; Panagopoulos 2000; Roberts *et al*. 2004; Koike *et al*. 2007).

Control

Obtaining disease-free transplants is particularly crucial. Seed treatments are recommended to reduce possible trans-

mission of the bacterium. Crop rotations and the eradication of volunteer tomato plants or other weed hosts should be applied to reduce the inoculum. Sprays of a mixed copper product can reduce the spread of disease if applications begin when the first symptoms appear. Copper plus mancozeb sprays provide some disease control (Jones *et al.* 1992; Roberts *et al.* 2004).

Bacterial wilt

Bacterial wilt, or Southern bacterial wilt, is a very serious disease of more than 200 plant species belonging to over 40 families. The disease occurs in greenhouses in cooler climates. Major agronomic hosts include pepper, potato, tobacco, tomato, eggplant, and banana. Reports of complete crop loss in the field are not uncommon (Panagopoulos 2000; Koike *et al.* 2007).

Symptoms
The disease symptoms first appear as a light, partial or total flaccidity (epinasty) of young leaves during the hotter hours of day, followed by recovery at night. Sometimes, only one side or part of the plant may be affected. Under favourable conditions, rapid and complete wilting follows and the plant finally collapses and dies (**Fig. 8B**). The stems of diseased plants, particularly near the base, as well as the roots, present an internal brown discoloration of the vessels. In cross-section, tiny, white, milky drops of viscous fluid ooze from the severed vascular bundles of infected shoots. A simple diagnostic test is to place the lower part of the cut stem into water; a milky, white exudate of bacteria will be seen streaming from the infected stem within 3-5 min. (Jones *et al.* 1992; Panagopoulos 2000; Pernezny *et al.* 2003; Koike *et al.* 2007).

Disease development
Ralstonia solanacearum (Smith) Yabuuchi *et al.*, formerly known as *Pseudomonas solanacearum*, is a Gram-negative, rod-shaped, aerobic, motile bacterium with one to four polar flagella, strains of which differ in host range, geographical distribution, pathogenicity, and physiological properties. Strains are subdivided informally into five races based upon host range, or differentiated into five biovars. Race 1 has a very wide host range of crops and weeds, and strains are predominately biovars 1, 3 and 4. Race 1 strains cause bacterial wilt of pepper and other Solanaceous crops (potato, tobacco, tomato, and eggplant). Race 2 strains cause Moko disease of banana and diseases of Heliconias. Race 3 and biovar 2 strains are the same and affect mainly potato, but occasionally pepper and other Solanaceous crops and weeds. Races 4 and 5 have host ranges limited to mulberry and ginger, respectively. Molecular analysis by RFLP (restriction length fragment polymorphism) and 16S rRNA, grouped all the strains tested into two major divisions that were related to geographical origin. Identification of the bacterium can be confirmed by classical and molecular diagnostic methods. The bacterium is classified in EPPO A2 quarantine lists (Schaad *et al.* 2001; Pernezny *et al.* 2003; Janse 2007).

The bacteria are able to survive in the soil for long periods of time in the absence of host plants. The length of the survival period depends on the strain of bacterium, the soil type, and soil moisture. Survival is longest in moist soil with low to moderate pH, although the disease occurs in almost all soil types or soil pH where host plants normally grow. The bacteria also survive in water for long periods of time. Dissemination occurs by irrigation or rain water, infected seeds of tomato and eggplant, and the soil. *R. solanacearum* enters the plant via the roots, through growth cracks or wounds caused by nematodes, insects or farm equipment. The bacteria colonize the xylem and spread throughout the plant, multiplying to a high population density. Wilt occurs 2-5 days after infection. The bacteria invade the intercellular spaces of the parenchyma cells in the cortex and pith, where the cell walls disintegrate resulting in pockets filled with bacterial cells, polysaccharide and cellular debris. Bacteria are released back into the soil from infected plants roots and decaying plant material. The bacterial wilt pathogen generally does not survive where the mean temperature is <10°C, although survival of some strains of *R. solanacearum* occurs in temperate climates. The disease usually develops when mean temperatures are >20°C, and more severe wilting is seen at temperatures of >30°C and high soil moisture (Jones *et al.* 1992; Panagopoulos 2000; Roberts *et al.* 2004; Koike *et al.* 2007).

Control
The most effective and often only means of control is to avoid planting in infested soil. The disease should be avoided by using pathogen-free seeds grown in sterile soil and transplant trays. A long rotation (5-7 years) with non-host crops, particularly grasses, may reduce the population in infested greenhouse soils. Grafting of susceptible tomato and eggplant scions onto resistant rootstocks has been used and may be an option for pepper also (Jones *et al.* 1992). The greenhouse should be kept free of weeds and volunteer plants. Nematodes in soil should be eradicated since their presence increases bacterial wilt incidence. Soil should not be moved from infected fields on equipment or workers. Workers and equipment should be disinfested. There is no effective chemical control for bacterial wilt although soil fumigation may reduce the population of *R. solanacearum* in soil. Amendments (organic or inorganic) into soil may reduce disease incidence by creating suppressive conditions (Jones *et al.* 1992; Roberts *et al.* 2004; Koike *et al.* 2007).

Tomato pith necrosis

Tomato pith necrosis (also called brown pith necrosis or stem necrosis) has become a problem of glasshouse and open field tomatoes in recent years and is caused by various pathogenic bacteria. In New Zealand it was described in 1973 and first attributed to *Pseudomonas viridiflava*, but later in 1974 to *Pseudomonas cichorii*. In 1978 it was described in England and attributed to *Pseudomonas corrugata* Roberts & Scarlett. In the USA, *Erwinia chrysanthemi* was reported to be the casual agent of pith necrosis, indicating the complexity of the disease. In pepper, the casual agent is *P. corrugata* (Schaad *et al.* 2001; Pernezny *et al.* 2003; Janse 2005).

Symptoms
The disease affects older plants and symptoms usually do not show until fruits begin to develop. The disease usually begins from the site of a wound on shoots and stalks and advances in the pith mainly to the upper part of the plant, although the rest may appear healthy. Occasionally the plant dies when the lower stem is affected. Early symptoms include wilting of young foliage and chlorosis and wilting of older leaves. Affected leaves curl up and turn brown peripherally. Infected stems may have grey to dark brown lesions on the surface. Internally, such stems have pith tissue that contains cavities, is darkly discoloured, and can become hollow or form a chamber. Adventitious roots may grow profusely from these symptomatic stems. Vascular tissue may become dark brown. At an advanced stage, severely infected plants may collapse and die. Overall, tomato pith necrosis symptoms may resemble those of bacterial canker (Jones *et al.* 1992; Panagopoulos 2000; Koike *et al.* 2007).

Disease development
Tomato pith necrosis develops under low night temperatures, high nitrogen levels, and high humidity. Such conditions usually exist in unheated greenhouses. The pathogenic bacteria survive as epiphytes on the surfaces of the plants and under favourable conditions multiply abundantly. They gain entry into the plant mainly through pruning sites, other wounds of the shoot, and even perhaps the roots. The disease is frequently expressed when the first fruit set is near

to mature green. It is uncertain if the disease is seed-borne and more research is warranted on the epidemiology of the disease.

Control

Few control recommendations have been established. Excessively high nitrogen fertilizer rates and overhead sprinkler irrigation should be avoided. Regular sanitation of tools (clippers and pruning shears) with an appropriate disinfectant is highly recommended and plant foliage should not be disturbed when wet. Crop rotation with a non-host crop and eradication of volunteer tomato or weed hosts is also suggested. Preventive sprays with copper compounds after pruning can reduce infection by the pathogen (Jones *et al*. 1992; Panagopoulos 2000).

Bacterial stem rot

Bacterial stem rot occurs in greenhouses as well as in open field tomatoes. The highest incidence of the disease occurs on staked or trellised plants after pruning. The disease is considered to be a relatively minor disease of tomato, but occasionally major losses occur.

Symptoms

The first symptoms of the disease appear as a wilt of the plants at, or after, the time of first fruit harvest. Infected plants develop a black, soil-line lesion that usually results in stem weakening, lower stem breakage, plant stunting, plant wilt (plants remain wilted for many days) and plant death. The soil-line lesion is usually very soft and mushy in texture. The pith usually disintegrates, causing a hollow stem symptom observable in stem cross sections. Vascular discoloration does not extend significantly above or below the hollow stem (Jones *et al*. 1992; Panagopoulos 2000; Koike *et al*. 2007).

Disease development

The causal agents of bacterial stem rot are the bacteria *Erwinia carotovora* pv. c*arotovora* (Jones) Bergey *et al*. and *E. carotovora* subsp. *atroseptica* (van Hall) Dye. *Erwinia chrysanthemi* causes the disease on peppers (Alivizatos 1985). The isolation of the pathogen is rather difficult due to the presence of many saprophytic bacteria in the rotted tissues. It also causes soft rot in many other vegetables before and after harvest. The favourable conditions for growth are high RH and temperatures of 25-30°C. The bacterium is aerobic as well as anaerobic, Gram negative, motile with peritrichous flagella.

The bacterium occurs in greenhouse and field grown tomato. It is possibly spread by insects. The invasion of fruit by fruit worms is usually the source of large amounts of inoculum (Jones *et al*. 1992; Pernezny *et al*. 2003; Janse 2006). Entry is also made through fresh wounds, usually after the removal of shoots and leaves. The bacterium may also be present in senescent leaves and invade stems through the vascular system. In greenhouses, the disease may be ubiquitous in plants under gutters where high moisture conditions are normal (Schaad *et al.* 2001; Pernezny *et al.* 2003; Janse 2006).

Control

Bacterial stem rot can be prevented by appropriate sanitation. Although it is present in all crops, the pathogen does not reach populations that are epidemiologically significant other than in the diseased plant. Therefore, the spread of the bacterium from diseased to healthy plants through cultural practices can be avoided (Jones *et al*. 1992; Panagopoulos 2000; Koike *et al*. 2007).

Tomato big bud and/or Stolbur

Tomato big bud phytoplasma, mainly reported from parts of the world outside the EPPO region, causes a disease similar to stolbur. Since 'stolbur' in Russian is translated as 'big bud' in English, there is some confusion between tomato stolbur and tomato big bud in the literature. Indeed, some authors (Khurana *et al*. 1988) consider the two as simply synonymous. However, there are indications (Clark *et al.* 1989) that tomato big bud phytoplasma is serologically distinct from European aster yellow phytoplasmas. In any case, the vectors of these phytoplasmas in various parts of the world are different, so for the present this question must be considered unresolved. In cultivations such as tomato, pepper, eggplant, lettuce, and potato the disease is uncontrollable.

Symptoms

The symptoms observed on tomato plants are leaf yellowing, a reduction in size, virescence, witches' broom, stunting, as well as flower and fruit abnormalities. The first symptoms of the disease, which appear 6 weeks after infection, include short internodes, blastomania, chlorosis and a reduction in leaf size. Later, growth slows down, flowers deform, petals show greening with connected sepals and pistil elongation. Shoots with a tendency to elongate and thicken often produce extremely deformed flowers. Later the young leaves and sepals develop a purple colour. The fruits are smaller than those of healthy plants, with a lighter red colour and insufficiently or non-developed flesh and harder pulp (**Fig. 8C**) (Jones *et al*. 1992; Panagopoulos 2000).

Disease development

The disease is caused by a phytoplasma (ex mycoplasma, MLO) such as those causing aster yellows. It is reported that the pathogen is transmitted by insects (leafhoppers) *Hyalesthes obsoletus*, *Macrosteless* and *Lygus* sp. (Favali *et al.* 2000; Anfoka *et al.* 2003). Stolbur phytoplasma typically attacks the Solanaceae (45 species). The principal economic hosts are potatoes, tomatoes, *Capsicum* spp. and eggplants. The pathogen survives on weeds (e.g. *Convolvulus arvensis*, *Rumex* sp., *Ranunculus* sp., *Chenopodium album*, *Solanum nigrum*, *Sonchus* sp., *Datura stramonium*) during summer and winter and is transmitted by insects to cultivated hosts. Stolbur phytoplasma is the central and southern European representative of the aster yellows complex on the Solanaceae. Similar, but probably not identical, phytoplasmas occur in other continents. The pathogen is mentioned in EPPO A2 quarantine lists (EPPO/CABI, 1996; Janse 2006).

Control

The symptoms of diseased plants are reduced after applying tetracycline hydrochloride. Control of weeds, leafhoppers and other vectors on a regular basis and phytosanitary measures are usually sufficient (Jones *et al*. 1992; Panagopoulos 2000; Roberts *et al.* 2004; Koike *et al*. 2007).

VIRAL DISEASES

Tobacco mosaic virus (TMV) *and Tomato mosaic virus* (ToMV)

The viruses cause serious economic losses on tomato, pepper and eggplant in both field and greenhouse crops (Panagopoulos 2000). Based on the symptomatology on tomato, the diseases these viruses cause are differentiated as:

Tomato common mosaic

Symptoms appear as mosaic, mottling, malformation, threading, curling, bleaching of the veins and occasionally necrosis of the leaves that sometimes become fern-like and sharply pointed. Mottling and blemishing of the fruits accompanied by reduced yield is common when infection occurs early in the season, leading to stunting and severe chlorosis of the plants. The intensity of symptoms is influenced by environmental and cultural conditions like temperature, light, nutrient availability and water stress (Zitter and Provvidenti 1984). Lately in Greece, TMV-ring and TMV-fruit

Fig. 9 Symptoms of TMV (*Tobacco mosaic virus*) infection in pepper leaves (**A**) and fruit (**B**). Symptoms of CMV (*Cucumber mosaic virus*) infection in pepper fruit (**C**), tomato fruit (**D**) and eggplant leaves (**E**). Symptoms of TYLCV (*Tomato yellow leaf curl virus*) infection in tomato leaves (**F**). (Photographs **A**, **B**, **C**, **D**, **E** courtesy of Prof. E. C. Tjamos, Agricultural University of Athens, Greece).

necrosis (two strains of the virus that are differentiated based on the symptoms they cause on tomato) have been found to cause losses in tomato crops (Panagopoulos 2000).

Symptoms of virus infection in pepper vary with cultivar (Moriones and Luis-Arteaga 1999). They include chlorotic mosaic and distortion of leaves, necrotic local lesions, systemic leaf and stem necrosis and defoliation (Himmel 2003). The pepper strain also causes mosaic and distortion of pepper fruit (**Fig. 9A, 9B**) (Cerkauskas 2004b). The same symptoms are seen on eggplant, although the virus rarely occurs on this species (Panagopoulos 2000).

The causal agents of the disease are various strains of TMV or ToMV (two very closely related viruses) (Zitter 1991). TMV has a wider host range but is more frequently found on tobacco than tomato. The viruses are mechanically transmitted and enter the plants through wounds usually caused by handling of the plants or by insect feeding. Sources of initial inoculum are contaminated tomato seeds, plant debris and infected tobacco products that contaminate workers' hands (Zitter and Provvidenti 1984; Pfleger and Zeyen 1991). Disease diagnosis based only on plant symptoms is not reliable, so it should be based on differential symptom expression on index-plants, serological (Immuno-Sorbent Electron Microscopy, ELISA) or molecular techniques (Panagopoulos 2000).

Tomato single streak

Tomato single streak is caused by a particular strain of TMV, which under certain environmental conditions leads to the development of necrotic spots or brown streaks on the leaves, petioles, stems and fruits of tomato (Panagopoulos 2000). Infected stems become fragile showing brown areas on the bark and in the pith. Spots on the leaves progressively increase in size, leading to leaf death. On the fruits, small slightly sunken brown rings are formed (Zitter and Provvidenti 1984). Disease symptoms may be confused with those caused by bacterial speck of tomato.

Tomato double virus streak

Tomato double virus streak is due to a mixed infection by two different viruses, TMV and PVX (*Potato virus X*),

also a mechanically transmitted virus (Panagopoulos 2000). Infected plants show long brown to black streaks on the petioles, the main leaf veins and the stems. Often, plants are dwarf and leaves wither and curl. On the fruits, irregular brown necrotic spots of various sizes are formed that in many cases coalesce and as the fruit ripens they fall off (Zitter and Provvidenti 1984). Pathogen diagnosis should be based on specific laboratory techniques (index-plants, serology) since disease symptoms are often confused with those caused by other viruses (Panagopoulos 2000).

Tomato internal browning

Tomato internal browning is a defect of the fruit known by various names, such as 'brown wall' or 'internal browning' (Zitter and Provvidenti 1984; Panagopoulos 2000). It manifests itself on the green, unripe fruit mainly around the fruit petiole. The parenchyma cells in this area become brown, corky and eventually collapse and die. Internal vascular browning is easily seen due to the clear flesh of the unripe fruit. When the fruit ripens, infected areas remain green, progressively harden and become chlorotic. This disorder is attributed to late infection of tomato plants by TMV in conjunction with a number of non-parasitic factors, like an excess of nitrogen and phosphorus over potassium, high soil moisture, various conditions of light and temperature or cultivar sensitivity (Zitter and Provvidenti 1984).

Cucumber mosaic virus (CMV)

Symptoms

This virus has an extensive host range, infecting 775 species of cultivated plants or weeds (Panagopoulos 2000), including tomato, pepper and eggplant in both field- and glasshouse-grown crops, which may suffer very serious economic damage due to a loss of yield and downgraded fruit quality. Infected tomatoes show a variety of symptoms, the most striking of which is the bushy appearance of plants, with yellow foliage, dwarfing and shoe string-like leaf blades (Zitter 1991; Cerkauskas 2004a). Often a 'fern leaf' figure is formed on the leaves (Panagopoulos 2000). A lot of flowers become infertile, fruit set is decreased and infected fruit show delayed maturity. Early infection of pepper leads to the development of mosaic and necrosis on the leaves (**Fig. 9E**), while plants become severely stunted with tiny malformed leaves (Murphy 2003). Plants that are infected later in the season show milder symptoms, while blossom drop results in lower yield. In eggplant, the virus causes a yellow mosaic or mosaic, a decrease in plant development and a reduction in yield. Infected fruits are usually smaller, malformed with yellow-white streaks (**Fig. 9C, 9D**) (Panagopoulos 2000).

Disease development

The virus is transmitted by at least 60 species of aphids in a non-persistent manner. In contrast to TMV, it is not seed-borne and does not remain in plant debris in the soil but can be successfully transmitted by infected sap. Initial sources of inoculum are infected weed species or other cultivated host plants (Zitter and Provvidenti 1984). Because the symptoms of the disease may resemble those of other diseases, accurate diagnosis of the virus should be based on laboratory techniques (index-plants, serological or molecular methods) (Panagopoulos 2000).

Tomato spotted wilt virus (TSWV)

Symptoms

The first symptoms of infection of young tomato plants by TSWV include bronzing of the upper leaf surface, sometimes accompanied by small dark spots followed by a down cupping and distortion of the leaflet (Panagopoulos 2000). In time, the leaf blade becomes brown and necrotic, and there is a general stunting, with growing stem tips eventually dieing back (Zitter *et al.* 1989; Zitter 1991). Infected tomato plants have no or poor fruit set; young green fruits

develop mottled spots of a yellow to light green color with a raised center, while on mature tomatoes characteristic orange to red rings appear, resulting in irregular ripening of the fruit. On pepper, virus infection leads to a yellowing and browning of the leaves that eventually become necrotic (Zitter *et al.* 1989). Plants infected at a later stage of growth, manifest symptoms of chlorotic or necrotic flecks or necrotic rings on leaves and stems (Adkins 2003). After fruit set, infection leads to the development of large necrotic streaks on peppers, while younger fruits usually die.

Disease development

The virus is transmitted by several species of thrips in a persistent manner (Zitter *et al.* 1989), with *Thrips tabaci* and *Frankliniella occidentalis* being considered the most important vectors due to their wide distribution and plant-host range (Panagopoulos 2000). The virus can only be acquired by the larvae of the insect feeding on infected hosts; however, the most efficient transmission is carried out by the winged adult thrips feeding on healthy plants. Viruliferous thrips can transmit the virus for a period of 30 days, or for their whole life time. The virus is not transmitted from adult thrips to their progeny through eggs. Virus sources are infected weed species and overwintering adult thrips (Cho *et al.* 1986). TSWV onset and severity is directly connected to conditions that favor the early appearance, high activity and increase of vector populations. Accurate diagnosis of the virus is based on index plants and on serological or molecular methods (Panagopoulos 2000).

Tomato yellow leaf curl virus (TYLCV)

Symptoms

Early infection results in severe stunting of tomato plants, with abnormal and smaller leaflets that are cupped upwards (Panagopoulos 2000). Leaves that develop in later stages are very chlorotic and malformed; their margins are rolled upwards showing intense curling between the veins **Fig. 9F**) (Defra 2004). Infected plants usually exhibit flower drop and yield reduction. When tomato plants are infected early fruit setting stops, while in late infected plants fruits that are already present ripen normally but no new fruits are formed (Zitter 1991). Infected pepper plants may be symptomless or have leaf margins rolled upwards, and interveinal leaf areas become chlorotic with yellow margins (Defra 2004). Eggplant is considered immune to TYLCV infection (Czosnek *et al.* 1993).

Disease development

The virus is vectored by the sweet potato whitefly *Bemisia tabaci* in a persistent manner (Zitter 1991). Although a number of biotypes of the insect have been identified, B and Q biotypes are considered to be the most effective virus vectors and attack a wide range of host plants (Khasdan *et al.* 2005). TYLCV represents a complex of virus species that are present in tropical and sub-tropical areas (Zeidan *et al.* 1998; Rybicki *et al.* 2000). They are all monopartite geminiviruses (except for one bipartite species found in Thailand) belonging to the genus *Begomovirus* (Rochester *et al.* 1994). In southern Europe, two species of TYLCV have been found: one from Israel (TYLCV-IL), which is the most widespread species, and one from Sardinia (TYLC-SV) (Kheyr-Pour *et al.* 1991; Zeidan *et al.* 1998). Since the virus is not sap-transmitted, spread of the disease to new areas and hosts is based on the introduction and movement of the whitefly vector. The virus is acquired by the nymphal or adult stages of the insect after feeding on infected plants; however, effective spread of TYLCV to healthy plants is caused by the adult whiteflies (Defra UK 2004). Various weed hosts, e.g. black nightshade (*Solanum nigrum*), and infected planting material serve as virus reservoirs (Zitter 1991). However, virus spread from those sources requires the presence of the insect vector.

Control of viral diseases of Solanaceous crops

Since there are no efficient chemical treatments to control viral diseases of plants, disease management in practice is focused on measures that reduce or, if possible, eliminate sources of viruses and limit their spread by insect vectors (when they are insect-transmitted).

The most important precautions that should be taken by greenhouse growers of Solanaceous crops are the following:

- Nursery soils should be sterilized before seed planting or nurseries should be located in new areas with no history of a tomato or pepper crop (measures mostly for TMV and ToMV that persist in the soil) (Pfleger and Zeyen 1991; Panagopoulos 2000; Cerkauskas 2004b).
- Only healthy seed should be used in nurseries (Pfleger and Zeyen 1991; Panagopoulos 2000). Alternatively, TMV or ToMV can be eliminated from the seed coat by soaking the seed in a 15% tri-sodium phosphate solution (Na_3PO_3) for 20 min. Seed can also be disinfested by heating at 70°C for 2-4 days in a dry oven, but to avoid damage caution must be taken that the seed is absolutely dry before exposure to this temperature (Zitter 1991).
- Nurseries should be constantly inspected and any plants suspected of viral infection should be immediately removed and destroyed.
- Workers should disinfest their hands by washing them frequently and thoroughly with water and soap before handling plants in the nurseries or in the greenhouse. Tools should be disinfected by immersion in boiling water for 5 min followed by washing in a detergent solution (Zitter 1991; Panagopoulos 2000; Himmel 2003). Commercial bleach is not effective in de-contaminating tools (TMV and ToMV) (Pfleger and Zeyen 1991). Special care should be taken when handling diseased plants to ensure that hands and tools are disinfected before moving to healthy plants. Smoking is not permitted during transplanting or when pruning and tying up plants (this applies to all viruses, but especially to TMV and ToMV).
- Only absolutely healthy plants should be transplanted to the greenhouse (Zitter 1991; Panagopoulos 2000; Himmel 2003).
- Co-cultivation of tomato or pepper with other susceptible hosts in the same greenhouse should be avoided. If susceptible hosts are rotated in the greenhouse, then a free period of at least 4 months should be kept, or alternatively greenhouse soil should be disinfected before each crop (mostly for TMV and ToMV) (Panagopoulos 2000).
- Plant debris should be carefully removed and destroyed, and greenhouses should be kept free of weeds. Crops should be constantly inspected and infected plants should be removed instantly (Zitter 1991; Panagopoulos 2000; Himmel 2003).
- Complete chemical control of aphids (for CMV) and thrips vectors (for TSWV) (Zitter *et al.* 1989), or the use of reflective mulches to deter aphids (Zitter 1991; Himmel 2003; Cerkauskas 2004a) (for CMV). Chemical control of whiteflies (Salati *et al.* 2002) (for TYLCV).
- The use of resistant varieties if available (Zitter 1991; Panagopoulos 2000; Himmel 2003; Cerkauskas 2004a, 2004b). There are commercially available tomato cultivars or hybrids that are resistant to various TMV races or other tomato viruses. Recently, 'premunition' of plants against TMV (inoculation of plants with mild strains of TMV) has been found effective against infection by aggressive TMV strains. However, this is a rather sensitive balance and should be applied only after on-site experimentation. A more advanced method for controlling viral diseases is by genetically engineering resistance via the incorporation of viral genes into the plant genome (transgenic plants). Coat protein-mediated resistance of tomato plants against TMV has been achieved by expressing the virus coat protein gene in tomato plants. However, genetic engineering methods for developing resistance to plant viruses is basically experimental and has found

only very limited practical application to date (Panagopoulos 2000).

- With respect to tomato internal browning, in addition to the above a balanced fertilizer application schedule should be followed to avoid excess nitrogen and phosphate in relation to potassium (Panagopoulos 2000).

CONCLUSIONS AND FUTURE PERSPECTIVES

Greenhouse climatic conditions (warm and humid) provide an ideal environment for the development of many foliar, stem and soil-borne plant diseases. For the majority of aerial pathogenic fungi and bacteria, infection usually occurs when a film or drop of water persists on the plant surface for some hours. Unless temperature, RH and ventilation are well regulated, this surface water can remain in the greenhouse until infection becomes established. Integration of cultural practices, physical, chemical and biological control options is needed to prevent widespread outbreak of greenhouse diseases within the soil or aerial environment. Disease control techniques include the use of disease resistant varieties, disease-free seeds and plants, regulation of air circulation, humidity control, well-drained soil, weed eradication, sanitation, chemical compounds, microbial antagonists, disease-suppressive composts. For instance, to achieve optimum crop yield and quality, integrated disease management is based on the elimination of the pathogen inoculum through high standards of hygiene (sterilizing soil or using soilless media, obtaining disease-free planting material, chlorine bleach rinses of footwear and equipment, weed-free floors, etc.), cultural practices for limiting disease spread, biological and pesticidal control, and, most important, when available, the use of resistant germplasm (van Lenteren 2000).

The increase in world population demands judicious management of plant resources, and advances in plant health management are imperative for humans' sustained ability to benefit from nature. Successful management of plant health requires a greater diversity of research efforts and approaches than ever before. Emerging techniques for the environmentally-friendly management of plant diseases, weeds and pests are now -more than ever- imperative.

Efforts to maintain plant health have evolved through an increased understanding of plant ecology and physiology and the interactions of factors causing adverse effects on plant health. Understanding ecology and the interactions of plant pathogens and their antagonists (biological control agents) has long been recognized as an essential element in designing and implementing effective plant health management programmes. It is crucial to understand the molecular and biochemical mechanisms that underpin these interactions in order to discover new ways of successfully controlling plant pathogens in a sustainable agricultural system. The advent of rapid and cost-affordable gene sequencing facilities has led to full genome sequencing of several plant pathogens, resulting in novel insights of their evolution and pathogenesis. The work on model plants has facilitated and shed light on plant-pathogen interaction mechanisms. Now, appropriate studies need to be undertaken to establish the extent to which model plants are representative of the behavior of crop plants and how to utilize this knowledge in order to develop new resistant plant cultivars (by conventional breeding or transgenic methodologies), the resistance of which cannot easily be overcome by pathogens. Management of viral diseases using gene manipulation has already proved to be quite a successful approach in several cases.

There is an increasing need for research on disease management adapted to organic agriculture systems, which occupy an increasing proportion of the agricultural economy, and the utilization of agricultural waste so as to reduce pollution and benefit plant health. The potential use of organic matter or natural compounds from plants with properties for disease control has already been included in programmes of many research centers throughout the world.

Effective plant health management depends on the availability of accurate data regarding disease causal agents

and mechanisms affecting plant health. Methodology adapted to deriving data ultimately leading to new management systems will need to include traditional methods and modern techniques. Tools for accurately documenting the nature of the problem include such traditional techniques as light and electron microscopy as well as molecular techniques. The toolbox of current molecular techniques has led to the identification of molecular markers and biochemical assays that tremendously improve the speed and accuracy of detection and identification of individual plant pathogens and complexes thereof in laboratory diagnostics. Continuing research will still further improve these detection methodologies.

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