

# Diseases of Passion Flower (*Passiflora* spp.)

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## ABSTRACT

There may be many factors contributing to reduction in longevity and productivity in passion fruit plants, especially diseases of viral, bacterial or fungal etiologies, among which passion fruit woodiness, bacterial spot, root and collar rot, fusarium wilt, anthracnose and scab are the most important. The incidence of woody fruits in young plants totally compromises the productivity and quality of fruits. Fusariosis, root and collar rot have swept out entire crops, leading to irreversible wilt and consequent death of plants. Bacteriosis, anthracnose and scab cause severe losses under favorable environmental conditions and the absence of preventive control. The permanent incidence of diseases in some areas may turn the crop unprofitable, causing its periodical migration to new areas. This review will cover the main passion fruit diseases, their symptoms, etiology, epidemiology and management.

**Keywords:** bacteria, fungi, plant pathogens, phytoplasma, nematodes, virus

**Abbreviations:** **BYMV**, Bean yellow mosaic virus; **CABMV**, Cowpea aphid-borne mosaic virus; **CiLV**, Citrus leprosis virus; **CMV**, Cucumber mosaic virus; **EAPV**, East Asian Passiflora virus; **MarMV**, Maracuja mosaic virus; **PaMV**, Passionfruit mottle virus; **PaVY**, *Passiflora* virus Y; **PaYMV**, Passion fruit yellow mosaic virus; **PGMV**, Purple granadilla mosaic virus; **PGSV**, Passion fruit green spot virus; **PLLMV**, Passion flower little leaf mosaic virus; **PLV**, *Passiflora* latent virus; **PRV**, *Passiflora* ringspot virus; **PWV**, Passionfruit woodiness virus; **ToRSV**, Tomato ringspot virus

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## INTRODUCTION

Passion flower is a tropical plant of the *Passiflora* genus having large genetic variability (~450 species) (Vanderplank 1996). *P. edulis* (purple passion fruit), *P. edulis* f. *flavicarpa* (yellow passion fruit), *P. ligularis* (urucu passion fruit), *P. mollissima* (curuba passion fruit), *P. quadrangularis* (melon passion fruit), *P. alata* (sweet passion fruit), *P. caerulea* (blue flower passion fruit) and *P. laurifolia* (lemon passion fruit) are the most important among the 60 passion fruit species producing edible fruits (Manicom *et al.* 2003). The leaves of some species are used as a substitute for tea, and the leaves and roots of others have medicinal properties.

*P. edulis*, as well as its *P. edulis* f. *flavicarpa* form, is the most important species. It originated in southern Brazil, but was widely distributed in the tropics and subtropics during the 19<sup>th</sup> century. *P. edulis* is grown especially in colder subtropical regions or in tropical lands of high altitude, in countries like South Africa, Kenya, Zimbabwe, India and New Zealand. *P. edulis* f. *flavicarpa* is grown especially in tropical and subtropical regions of Brazil, Ecuador, Venezuela and Colombia. Hybrids between *P. edulis* x *P. edulis* f. *flavicarpa* have been developed in Australia, Hawaii, Israel, South Africa and the USA, and are commercially important in lowland environments inhospitable to *P. edulis*. *P. mollissima* is grown in high altitudes in the Andes, and *P. ligularis* is grown from Mexico to the north of South America. *P. quadrangularis* is the most pervasive species in the tropics, being grown in small scale from Mexico to the South of Brazil. *P. laurifolia* is extensively grown in the Antilles, Guianas, Trinidad and Tobago, Venezuela and Brazil. *P. alata* crops are very common in Peru and distributed throughout Brazil. *P. caerulea* is the most popular ornamental species among Passifloraceae (Manicom *et al.* 2003).

Worldwide production figures for passion fruit are not available. The total world passion fruit production is estimated to be nearly 805,000 tons, with Brazil producing 56% of this total, followed by Ecuador and Colombia, with productions of 250,000 tons and 30,000 tons, respectively (ITI TROPICALS 2007). Latin America supplies the world export market for juice. Other important passion fruit producers are Peru, Australia, New Zealand, South Africa and Hawaii.

The *Passifloraceae* are perennial herbaceous and woody plants that usually have a vine-like growth habit. Diseases can seriously reduce the productivity of all of the species that are important commercially. The typical crop's useful life of 5 years can be reduced to one year due to diseases, whose damage tends to worsen with an increase in the cultivated area in the same region. In some places, a nomadic pattern has been observed for this crop.

## DISEASES CAUSED BY VIRUSES

### Potyvirus diseases

Several passion flower diseases associated with viruses belonging to the genus *Potyvirus* have been described in different parts of the world. The first potyvirus found infecting passion flower was *Passion fruit woodiness virus* (PWV) in

Australia (McKnigh 1953; Taylor and Greber 1973). Potyviruses associated with woodiness diseases were later reported in Nigeria (Martini 1962), Taiwan (Chang 1992) and Japan (Iwai *et al.* 1996), although molecular studies are still needed for a better identification of these viruses. PWV was also identified as the causal agent of passion fruit woodiness in different states of Brazil, based on biological and serological properties (Chagas *et al.* 1981; Kitajima *et al.* 1986; Chagas *et al.* 1992). However, recent molecular studies of several Brazilian isolates of this potyvirus showed that it is a strain of *Cowpea aphid-borne mosaic virus* (CABMV) (Nascimento *et al.* 2004, 2006; Barros *et al.* 2007). Another strain of CABMV, originally designated as *South African Passiflora virus*, was described causing woodiness disease in South Africa (Brand *et al.* 1993). Other potyviruses found infecting *Passiflora* spp. are *Passiflora ringspot virus* (PRV) in Ivory Coast (De Wijs 1974), which also naturally infects *Adenia lobata*, and indigenous Passifloraceae in West Africa (de Wijs 1975; de Wijs and Mobach 1975); a strain of *Bean yellow mosaic virus* (BYMV) in *Passiflora caerulea* in Croatia (Pleše and Wrischer 1984) and Italy (Parrella and Castellano 2002); *Passionfruit mottle virus* in Taiwan (PaMV) (Chang 1992) and Sri-Lanka (Dassanayake and Hicks 1992); a strain of *Soybean mosaic virus* (SMV) in Colombia (Benschler *et al.* 1996); *Passiflora virus Y* (PaVY) in Australia and Indonesia (Parry *et al.* 2004); and *East Asian Passiflora virus* (EAPV) in Japan (Iwai *et al.* 2006a, 2006b). The diseases caused by PWV and CABMV are considered the most important diseases of passion flower crops.

### Symptoms

Symptoms induced by these potyviruses are variable. PWV and CABMV cause severe mosaic, rugosity and distortion on the leaves (Fig. 1A), a reduction of plant development, and woody and deformed fruits (Fig. 1B). Severe mosaic, epinasty, defoliation and premature death of plants are associated with infection with the strain of SMV. Passion flower infected by PRV commonly show leaf mottling and ringspot on the younger leaves. Fruits are symptomless. Some plants may exhibit malformed leaves with severe mosaic. PaMV and PaVY cause mild mottling on leaves, whereas PaMV still induces skin mottling on fruits. Plants infected with EAPV show chlorotic spots on the leaves and dappled or faded fruits.

### Causal agents

Viruses belonging to the genus *Potyvirus*, family *Potyviridae* are filamentous, not enveloped, flexuous, approximately 680-900 nm long and 12-15 nm wide. Virions contain a single molecule of linear, positive sense RNA, with approximately 10,000 nucleotides, which code for a single polyprotein. The genome-derived polyprotein is cleaved into several proteins, some of which form amorphous and pin-wheel inclusion bodies in the cells (Fauquet *et al.* 2005).

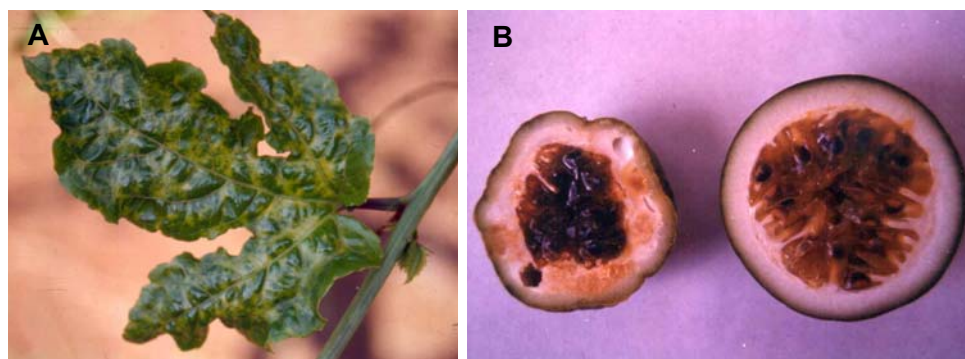


Fig. 1 (A) Leaf mosaic and (B) deformed fruit (left) caused by *Cowpea aphid-borne mosaic virus*.

## Epidemiology

As members of the genus *Potyvirus* they are naturally transmitted by several species of aphids, such as *Myzus persicae*, *Aphis gossypii*, *A. spiraecola*, *Toxoptera citricidus*, in a non-persistent, non-circulative way. Transmission efficiency varies according to the potyvirus and aphid species. These potyviruses can also be transmitted by grafting and experimental mechanical inoculation. Mechanical transmission by knives, scissors and nails during cultural practices of trimming was observed for CABMV in passion flower crops in São Paulo State, Brazil (VA Yuki, unpublished data). None of the potyviruses found in *Passiflora* spp. were transmitted by seeds.

The host range of PWV, CABMV, PRV, PaMV, PaVY, and EAPV is restricted to species in the families Passifloraceae, Fabaceae (Leguminosae), *Nicotiana benthamiana*, *N. clevelandii* and *N. tabacum* (Solanaceae), *Chenopodium album*, *C. amaranticolor*, and *C. quinoa* (Chenopodiaceae), and *Gomphrena globosa* (Amaranthaceae). Any species of *Passiflora*, when susceptible to these potyviruses, develop systemic infection, which may be symptomatic or latent. The reaction of species/varieties of Fabaceae is variable for each potyvirus. Some are unsusceptible; others are susceptible to infection with only local symptoms on inoculated leaves, or local lesion plus systemic infection or only systemic infection. Systemic infection may be symptomatic or latent depending on the interaction species/variety/potyvirus. Species of *Nicotiana*, when susceptible, usually develop systemic infection, whereas the chenopodiaceae show only local lesions on inoculated leaves. SMV and BYMV are viruses that infect mainly species of Fabaceae, and some non-legume species. More detail on the reaction of the different species/varieties to these potyviruses can be found in the following references: Bos (1970, 1972); de Wijs (1974); Chang (1992); Parry *et al.* (2004); Nascimento *et al.* (2006).

## Management

The control of potyvirus diseases in passion flower orchards is difficult. All species explored commercially, such as *P. edulis*, *P. edulis* f. *flavicarpa* and *P. alata* are susceptible to these viruses. Chemical control of vectors is usually inefficient for the control of the diseases caused by potyviruses because of the non-persistent relationship between the virus and the aphid vectors. The virus can be transmitted throughout a proof-feeding of few seconds, before any effective action of the chemical on the aphids. Furthermore, with very few exceptions, in which *Aphis gossypii* was found colonizing *P. edulis* f. *flavicarpa* under greenhouse conditions (Piero *et al.* 2006), aphids usually do not colonize *Passiflora* spp., that is, they are only "visitors" of these plants. In Australia, Simmonds (1959) reported a pioneer work on the efficiency of preimmunization (cross protection) with mild strains for the control of PWV in passion flower. In the 1970's, an accreditation scheme based on mild strain protection was established in New South Wales, Australia, to prevent losses on commercial passion fruit hybrid (*P. edulis*/*P. edulis* f. *flavicarpa*) due to severe strains of the virus (Peasley and Fitzel 1981; Pares *et al.* 1985). In spite of the apparent success of the program, problems associated with the occurrence of a more severe strain of PWV able to overcome mild strain protection and an apparent synergistic effect with *Cucumber mosaic virus* (CMV) infection, which caused tip necrosis disease, were reported (Peasley and Fitzel 1981; Fitzel and Pares 1985; Pares *et al.* 1985). Preimmunization was also studied for the control of the woodiness disease caused by CABMV, formerly attributed to PWV, in Brazil but it was not efficient due to breakdown in protection (Novaes and Rezende 2003). According to these authors breakdown in protection seems to be related to the low concentration and/or irregular distribution of the mild strains of CABMV in the leaves of the plants, which allows the existence of infection sites available for the establishment of the severe strain. In Taiwan, the annual eradication

of affected plantings and replanting with PWV-free seedlings is the usual procedure for the control of the woodiness disease (Chang *et al.* 1992). Studies for the development of transgenic passion flower resistant to CABMV are under way in Brazil (Alfenas *et al.* 2005; Trevisan *et al.* 2006).

In Brazil, at present, several cultural practices have been recommended to minimize the problems associated with CABMV in passion flower orchards. These include the use of virus-free seedlings for new plantings; eradication of old and abandoned orchards before starting new crops; care during trimming operations to eliminate mechanical transmission of the virus; avoid leguminous plants, which may harbor the virus, near the orchard; and rouging of diseased plants by means of systematic inspections during the first five months after transplanting (Gioria *et al.* 2000). These practices may be recommended for any other disease caused by potyvirus in passion flower crops.

## Cucumber mosaic virus (CMV)

This virus was first reported associated with passion fruit woodiness in Australia (Magee 1948; Taylor and Kimble 1964). It is possible that early workers who described CMV in passion flower were, in fact, dealing with mixed infections. The only time CMV was confirmed in passion flower by itself was by Teakle *et al.* (1963) in California; with electron microscopy, they observed CMV virions in *P. caerulea* and *P. alata-caerulea*. Magee (1948) did not have the facilities need to detect dual infections, and Taylor and Kimble (1964) did not test their CMV isolates for the presence of flexuous virus particles. In South Africa, Brand and Wechmar (1993) always found CMV in association with a potyvirus identified as a strain of CABMV in woodiness-affected plants. In Brazil, CMV has been regarded as a minor pathogen for passion flower crops (Kitajima *et al.* 1986), especially because the virus has limited systemic movement in the plant (Gioria *et al.* 2002). Diseased *P. edulis* f. *flavicarpa* exhibit bright yellow mottling on leaves (Fig. 2), starting at random points on the vine and diminishing in intensity towards the tip, which becomes symptomless as it grows. Remission of symptoms is accompanied by the disappearance of CMV, although the mechanism for such a phenomenon is not known. Symptoms on fruits have not been observed. *P. foetida*, however, when infected with CMV displays intermittent yellow mosaic leaves, but the virus does not disappear from the vines (DH Nakano and JAM Rezende, unpublished data). CMV is a type species of the genus *Cucumovirus*. The virus particle is icosahedrally isometric, approximately 30 nm in diameter. It has a single-stranded, positive-sense RNA genome consisting of three unique RNAs, 1, 2 and 3, each of which is encapsidated separately in the same capsid protein. The particles with RNA3 also contain a subgenomic coat protein messenger, RNA4. The presence of all three particles is required for in-



Fig. 2 Bright yellow mottling on leaves of yellow passion fruit caused by *Cucumber mosaic virus*.

fection (Manicom *et al.* 2003). The virus is naturally transmitted by several species of aphids in a non persistent way. It can also be transmitted mechanically to seedlings, although some difficulties on such transmission have been reported for *Passiflora* spp. (Teakle *et al.* 1963; Gioria *et al.* 2002; Manicom *et al.* 2003). No measure for the control of the disease has been recommended for the passion flower crops in Brazil.

### ***Passiflora latent virus (PLV)***

The complete nucleotide sequence of PLV has been recently published and suggests that this virus belongs to the genus *Carlavirus* (Spiegel *et al.* 2007). PLV has filamentous flexuous rods, 600-700 x 12-13 nm, containing a single-stranded RNA. *Passiflora* infection with PLV was first reported for *P. caerulea* and *P. suberosa* in Germany, in the 1960's (Brandes and Wetter 1963). Similar, if not identical, a carlavirus was later found in a sterile hybrid of *P. incarnata* x *P. cincinnata* in Florida, USA (St Hill *et al.* 1992), in germplasm collections in the UK and the Netherlands (Hicks *et al.* 1996), and in *P. edulis*, *P. suberosa* and *P. subpeltata* in Australia (Pares *et al.* 1997). The experimental host range of PLV is narrow and limited to *Passiflora* spp., *Chenopodium murale* and *C. quinoa* (Spiegel *et al.* 2007). In all *Passiflora* species the virus causes an inconspicuous, systemic foliar mosaic. In cooler weather, older leaves are mottled. PLV appears to be a problem primarily on vegetatively propagated ornamental *Passiflora* spp. As a member of the genus *Carlavirus*, PLV should be transmitted by aphids in a non-persistent, non-circulative manner; although experimental confirmation is still necessary. The effect of PLV on production of *Passiflora* spp. is unknown.

### ***Passion fruit yellow mosaic virus (PaYMV)***

Infection of *Passiflora* spp. with PaYMV has only been reported in Brazil and Colombia (Crestani *et al.* 1986; Morales *et al.* 2002). Infected plants exhibit a characteristic bright yellow mosaic, yellow net, and leaf crinckle (Fig. 3). PaYMV belongs to the genus *Tymovirus*. Virus particles are isometric, approximately 30 nm in diameter. The Brazilian isolate was easily transmitted mechanically only to species of *Passiflora* (Crestani *et al.* 1986), whereas a Colombian isolate was also transmitted to three species of *Physalis* (Morales *et al.* 2002). The beetle *Diabrotica speciosa* experimentally transmitted the Brazilian isolate of PaYMV, with low efficiency, but fails to transmit the Colombian isolate of the virus. The virus is apparently not transmitted by seeds. The effect of PaYMV on the yield of *Passiflora* spp. is unknown. Since the report of Crestani *et al.* (1986), the virus has never been found infecting passion flower crops in Brazil. This might be due to the fact that *D. speciosa*, which is a polyphagous beetle and might be the natural vector of PaYMV, is only found occasionally in passion flower plantations.



**Fig. 3** Symptoms of *Passion fruit yellow mosaic virus* on yellow passion flower (left) (photo: EW Kitajima).



**Fig. 4** Vein clearing on yellow passion flower leaf caused by *Passion fruit vein clearing virus*.

### ***Passion fruit vein clearing virus***

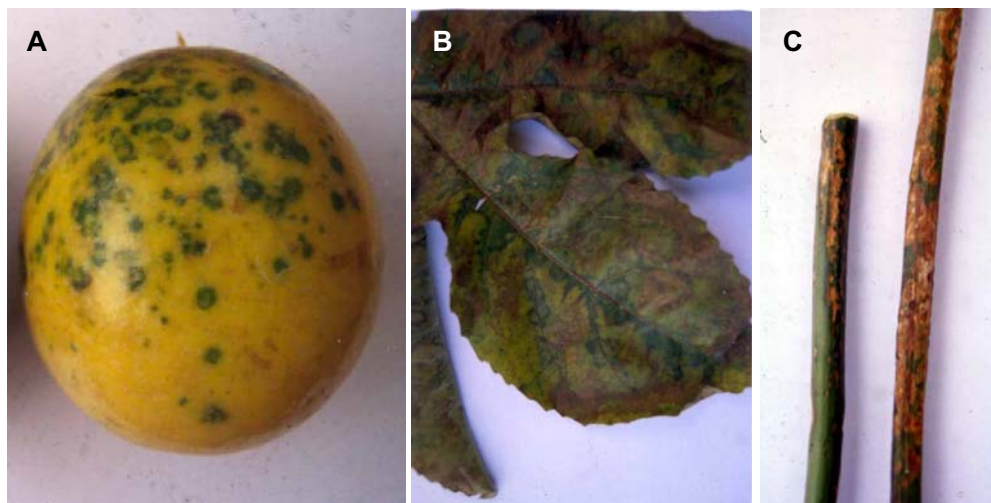
Passion flower vein clearing disease, caused by virus infection, was found in orchards in several Brazilian regions, sometimes causing severe yield loss. In addition to the vein clearing symptom on the leaves (Fig. 4), some plants also show a reduced size of leaves and fruit (Kitajima *et al.* 1986). Baciliform-like particles, typical of Rhabdovirus (Nucleorhabdovirus), approximately 300 nm x 70 nm were found in the perinuclear space of cells from infected plants. The virus was transmitted by grafting to *P. edulis*, *P. edulis* f. *flavicarpa*, and *P. maliformis*, but not by sap inoculation (Chagas *et al.* 1983). Host range and vector are still unknown. So far this virus does not represent a threat to the passion fruit industry in Brazil. Rhabdovirus-like particles (250 nm x 58 nm) were also found in the perinuclear space and the cytoplasm of mesophyll cells of *P. edulis* also infected with PWV (potyvirus) in Australia. The virus was not mechanically transmitted (Pares *et al.* 1983). The available data does not allow the establishment of any relationship between these two rhabdoviruses.

### ***Purple granadilla mosaic virus (PGMV)***

Natural infection of passion flower with PGMV has apparently only been found for *P. edulis* in Brazil (Chagas *et al.* 1984). Infected plants exhibit mild or line pattern mosaic on the leaves (Fig. 5), which turn more evident during cool season and almost disappear during the summer. Fruits from infected plants are smaller, deformed and woody. The virus has isometric particles, approximately 24 nm in diameter,



**Fig. 5** Symptoms of *Purple granadilla mosaic virus* on purple passion flower (photo: EW Kitajima).



**Fig. 6** Green spots on yellow passion fruit (A) and on senescent leaf (B); necrotic lesions on the stems (C) caused by *Passion fruit green spot virus*.

but has not been fully characterized taxonomically. Experimentally PGMV has only been transmitted to *Passiflora* species (Chagas *et al.* 1984). The beetle *D. speciosa* transmitted the virus experimentally (Oliveira *et al.* 1986). No other report of PGMV in passion flower crops has occurred in Brazil.

#### **Passion fruit green spot virus (PGSV)**

This virus was first found in the 1990's, in passion flower crops in the State of São Paulo, Brazil, causing severe damage (Kitajima *et al.* 1997). Later it was found infecting passion flower crops in other regions of the country (Kitajima *et al.* 2003). The name of the disease is derived from the green spots that are 2-5 mm in diameter and develop on mature yellow fruits (Fig. 6A). These spots may be uniformly green with a central necrotic depression. Green areas are present in isolated patches on senescent, chlorotic leaves (Fig. 6B), or along the veins. Necrotic lesions on the stems are frequent (Fig. 6C). When they occur in large numbers, the lesions may coalesce and girdle the stem, which results in subsequent death of the plant and eventually in the destruction of the entire orchard. The virus does not move systemically in the plant, staying restrict to the sites of vector feeding. Virus particles are short bacilliform, membrane bound, (50-70 nm x 100-120 nm) consistently present in the lumen of the endoplasmic reticulum of infected cells, and was tentatively placed in the family Rhabdoviridae (Kitajima *et al.* 1997). The virus is transmitted by *Brevipalpus phoenecis* (Acari: Tenuipalpidae). However, further studies on the genome of PGSV might place it in the new proposed genus *Cilevirus*, whose type member is *Citrus leprosis virus* (CiLV), which is also transmitted by *B. phoenecis*, and has particle morphology and cytopathic effect similar to those of PGSV (Locali-Fabris *et al.* 2006). There are many remaining questions that need to be addressed to better under-

stand the epidemiology of passion fruit green spot disease including identification of the sources of the virus and mites, alternative hosts, etc. The control of the disease has been efficiently achieved by monitoring *Brevipalpus* population and spraying acaricides for the early control of the mites. The following acaricides have been effective: hexythiazox, fenbutatin-oxide, propargite, quinomethionate, and dicofol (Kitajima *et al.* 2003).

#### **Geminivirus diseases**

Infection of passion flower with white fly (*Bemisia tabaci*) transmitted geminivirus have been reported in Puerto Rico (Brown *et al.* 1993) and Brazil (Novaes *et al.* 2003). In Puerto Rico, the geminivirus tentatively designated *Passiflora leaf mottle virus* induced severe curling, distortion, and mottling of leaves and fruits and reduced yields and fruit quality. The virus was experimentally transmitted from infected passion flower to bean and from bean to bean, but not from infected bean to passion flower. The virus was not transmitted by sap inoculation or by seeds of infected plants. The virus was subsequently identified as *Jatropha mosaic virus* and shown to be experimentally transmitted by the B biotype of *B. tabaci* (Brown and Bird 1996). In Brazil, the geminivirus was identified as belonging to the genus *Begomovirus* and tentatively named *Passion flower little leaf mosaic virus* (PLLMV). Infected passion flower exhibited intense yellow mosaic of leaves and drastic reduction on the leaf lamina (Fig. 7A, 7B) and plant development. The number of fruits per plant was small and most of them were deformed. The virus was transmitted by *B. tabaci*, which was found in high population colonizing the plants in two orchards. PLLMV was transmitted mechanically to *Nicotiana benthamiana* (Moreira *et al.* 2006) and by grafting to *P. alata*, *P. quadrangularis*, *P. suberosa*, and *P. serrato-digitata* (Alves and Rezende 2005). Since then the virus has



**Fig. 7** Mosaic and leaf malformation (A); and rugose mosaic (B) of yellow passion fruit plant caused by *Passion fruit little leaf mosaic virus*.

been sporadically detected in isolated plants in passion flower crops in different regions of Brazil.

### **Maracujá mosaic virus (MarMV)**

*P. edulis* infected with MarMV shows symptoms of leaf mosaic and crinkle. Virus particles are rigid rods, approximately 320 nm long and 18 nm in diameter. Transmission occurs without the help of vectors by contact between plants and any agricultural practice that causes mechanical damage. The virus is a member of the genus *Tobamovirus*. Infection of *Passiflora* spp. with different isolates of tobamovirus has been reported in India (Mali and Vyanjane 1980), Peru (Fribourg *et al.* 1987) and in germplasm collections in Florida, USA (St Hill *et al.* 1992). The complete genome sequence of MarMV from Peru was recently obtained and clearly indicated that it is a separate species of the genus *Tobamovirus* (Song *et al.* 2006). Further studies are needed for better characterization of the tobamovirus found infecting *Passiflora* sp. in India and Florida.

### **Tomato ringspot virus (ToRSV)**

Infection of *P. edulis* with ToRSV has only been reported once in Peru, in association with MarMV. The virus has isometric particles, 25-30 nm in diameter, and belongs to the genus *Nepovirus*. It is naturally transmitted by the nematode *Xiphinema americanum*, although a transmission test with *Passiflora* sp. was not done (Koenig and Fribourg 1986).

## **DISEASE CAUSED BY PHYTOPLASMA**

### **Overshooting**

Overshooting of passion flower, caused by Phytoplasma, seems to be an exclusively Brazilian disease. Although there have been reports of this disease in passion flower orchards in Pernambuco State during the 1980s (Kitajima *et al.* 1986), later occurrences of the disease have always involved few affected plants, without apparent damage. The disease is easily identified under field conditions and is characterized by chlorotic small leaves, shortening of internodes, excessive lateral shoots (which's broom) (**Fig. 8**) and abnormal flowers. There may be splitting and fall of fruits during their formation or just a reduction in their size. The phytoplasma which causes overshooting is a prokaryote without a cell wall which invades the phloem of plants. It has been recently classified as a member of the group 16S rIII – B (Ribeiro 2008). It shows fast dissemination by vectors still unknown, although sharpshooters are supposed to be involved, mainly the ones belonging to the *Empoasca* genus, which are often found in this crop. The pathogen may also be spread by grafting. To avoid the introduction of such phytoplasma into new producing areas it is necessary to carry out periodical inspection of plant nurseries and use



**Fig. 8** Witch's broom on yellow passion flower caused by phytoplasma (photo: R Gioria).

healthy seedlings. Plants must be periodically inspected in areas already affected by the disease and diseased plants have to be removed. It is known that phytoplasma-infected plants treated with antibiotics belonging to the tetracycline group show a temporary reduction of symptoms (Bradel *et al.* 2000). There are no studies on the use of this treatment for diseased passion flower.

## **DISEASES CAUSED BY BACTERIA**

Several bacteria are reported as being pathogenic to passion fruit plants in different parts of the world. *Xanthomonas axonopodis* pv. *passiflorae* is responsible for leaf lesions and may cause death of plants; *Pseudomonas syringae* pv. *syringae*, *P. syringae* pv. *passiflorae* and *P. viridiflava* cause leaf spots; *Agrobacterium tumefaciens* causes tumors, mainly in the collar region; *Erwinia carotovora* subsp. *carotovora* causes a soft rot; and *Ralstonia solanacearum* causes a vascular wilt (Bradbury 1986). Among such bacteria, the most important, due to the seriousness of the losses caused, are *X. axonopodis* pv. *passiflorae* and *P. syringae* pv. *passiflorae*.

### **Bacterial spot**

Bacterial spot has been reported in Australia (Bradbury 1986) and Colombia (Castilho and Granada 1995), and is especially important in Brazil, being observed in all producing regions (Pereira 1969; Malavolta 1998). It is the most important bacterial disease of passion flower due to the high susceptibility of economically important cultivars, the high level of damage the disease causes and the difficulty for control. Besides affecting *P. edulis* and *P. edulis* f. *flavicarpa*, the bacteria may also be responsible for natural infection of *P. alata*, *P. amethystina*, *P. serrato-digitata*, *P. cincinnata*, *P. coccinea*, *P. maliformis* and *P. nitida* (Rodrigues Neto *et al.* 1984; Beriam and Malavolta 2001; Junqueira *et al.* 2003). According to Oliveira *et al.* (1994), *P. quadrangularis* is highly susceptible to artificial inoculations.

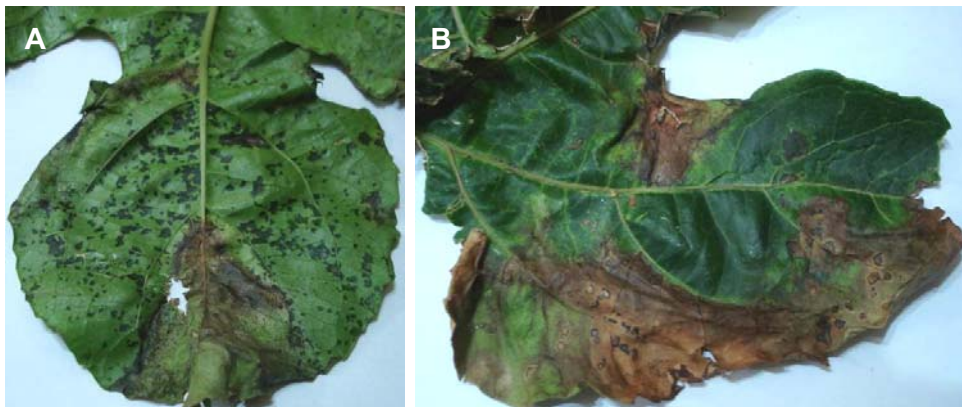
### **Symptoms**

The onset of leaf symptoms include well-defined and generally angular small spots, which are translucent, dark-green, anasarcous (**Fig. 9A**) and encircled by a chlorotic halo. Under favorable conditions, lesions become bigger, turn brown in color and may coalesce, affecting the entire leaf (**Fig. 9B**), causing wilt and fall of leaves. Infection may also spread through leaf veins and reach the vascular system of the vines, causing longitudinal grooves, darkening of vascular systems and portion dry, which reduces fruit production and may even cause plant death (Pereira 1969). Transversal cuts of infected vines exude bacterial pus.

Fruit lesions are dark or brownish green, anasarcous and circular or irregular with well-defined edges. Bacterial exudates, when dry, form a hard crust over the lesions. These spots penetrate the pulp, causing fruits to fall before maturation or making fruits unmarketable (Pereira 1969). The disease may also occur on petals and flowers of *P. alata*, causing slightly round irregular spots of translucent and oily aspect (J. Rodrigues Neto, pers. comm.).

### **Causal agent**

*Xanthomonas axonopodis* pv. *passiflorae* is a rod-shaped, 0.5 x 1.5  $\mu$ m, Gram-negative and aerobic, which does not form spores or capsules and presents a polar flagellum (Bradbury 1986). In culture medium, its colonies are bright yellow, mucous, round and convex, although a strain which does not produce yellow pigments (xanthomonads) has already been observed (Almeida *et al.* 1994). Its optimum temperature for growth is 27°C. The bacterium shows relative stability toward biochemical and physiological tests, and serology (Wendland *et al.* 1996, 1997a; Beriam *et al.*



**Fig. 9** Bacterial spot caused by *Xanthomonas axonopodis* pv. *passiflorae*. (A) Dark green and anarsarcous lesions. (B) Brownish lesions.

1998). However, it presents high genetic and pathogenic variability (Dias and Takatsu 1988), which may make the selection of intra and inter-varietal hybrids more difficult in the short and long run in studies on bacterial disease resistance.

### Epidemiology

The disease is more severe under high temperatures and relative humidity, when the incubation period is shorter, generally lasting 5 to 10 days (Pereira 1969; Piccinin *et al.* 1995).

Local dissemination of *X. axonopodis* pv. *passiflorae* is enhanced by wind-blown rain and irrigation, and by workers handlings wet plants, whereas long-distance dispersal occurs on seedlings and, according to Dias (1990), externally and internally on seeds. Pathogen penetration most frequently occurs via stomata and hydathodes, being favored by plant injuries, followed by colonization of inter-cell spaces in the leaf tissue, as well as vascular tissues. The bacterium survives in the plant diseased tissues and in contaminated crop residues.

### Management

Considering that commercial varieties of *P. edulis* e *P. edulis* f. *flavicarpa* are susceptible to the disease and there are no effective chemical products to control it, most controlling measures are only preventive.

Seeds and seedlings should be from healthy plants and, if possible, should be obtained from disease-free areas. Seed thermal therapy at 50°C for 15 minutes is efficient to eliminate the pathogen without affecting germination (Dias 1990). Some recommended measures to avoid the disease are: new plantings in areas free from the pathogen for at least two years; use of wind breaks; avoid working on wet plants; use adequate amounts of fertilizers, especially nitrogen, which stimulates new shootings and delays maturation, making plants more susceptible to bacterium. The elimination of diseased parts of the plants and the disinfection of pruning tools and hands with bactericide products, such as those using quaternary ammonium and alcohol, may reduce the spread of the pathogen.

Regarding chemical control, applications of copper oxychloride and its mixture with mancozeb at 7 to 15 days intervals decrease the intensity of the disease and favor production (Torres and Ponte 1994; Ruggiero *et al.* 1996). However, under frequent rains and favorable environmental conditions to the pathogen, the use of cupric fungicides or streptomycin sulfate may not be efficient (Romeiro 1995). If there is no fungicide or antibiotic absorption by the plant, the streptomycin sulfate, highly soluble in water, is washed away by the rain. If there is no rain or no sprinkler irrigation, the product shows effective protection (Romeiro 1995).

Among the species that have shown some resistance to the pathogen are *P. suberosa*, *P. setacea*, *P. caerulea*, *P. cinninata*, *P. foetida*, *P. giberti*, *P. mollissima*, *P. maliformis*, *P. laurifolia*, and *P. alata* x *P. macrocarpa* (Rodrigues Neto *et*

*al.* 1984; Oliveira *et al.* 1994; Barbosa, 1995; Wendland *et al.* 1997b). *P. edulis* f. *flavicarpa* transgenic plants showing resistance to bacteriosis are being developed and may be an interesting alternative for growers to control the disease (Castro 2005; Freitas *et al.* 2007).

### Bacterial grease spot

A high incidence of this disease caused by *Pseudomonas syringae* pv. *passiflorae* is observed in underripe fruits, which present small dark green areas, turning into golden to brownish greasy necrotic lesions. Later, a hard crust harboring several kinds of microorganisms covers these lesions. Spots are seldom found on leaves, where the disease causes severe necrotic lesions surrounded by a chlorotic halo. The bacterial grease spot seldom affects vines, where shallow canker lesions may be observed, as well as the death of the tip of the vines (Baigent and Starr 1963). The disease has also been reported in South Africa, New Zealand and Australia (Baigent and Starr 1963; Doepel 1965; Bradbury 1986).

### DISEASES CAUSED BY FUNGI AND FUNGUS-LIKE ORGANISMS

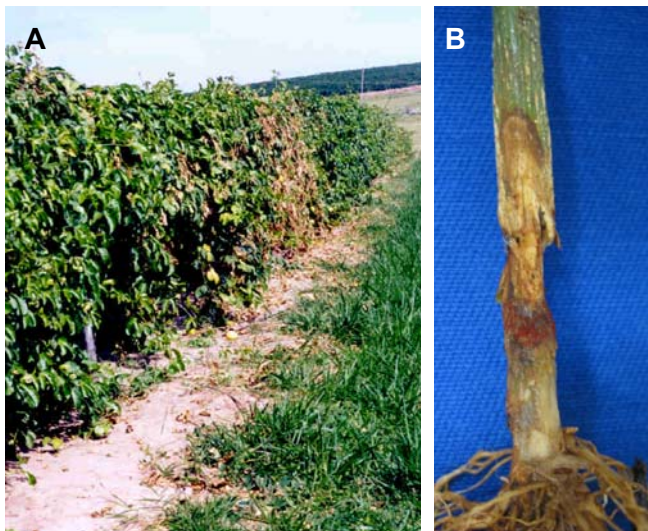
Diseases caused by fungi affect the passion fruit plant from the seedling phase until the adult-plant stage, harming roots, stems, leaves, flowers and fruits. During the postharvest stage, several fungi affecting plants in field conditions are also responsible for great losses during fruit storage, transport and commercialization. Some of the diseases affecting the aboveground part of plants are anthracnose, scab, septoriois and alternaria spot. The most difficult diseases to control are those caused by soil microorganisms, specially fusarium wilt, collar rot and crown rot.

### Collar rot

Collar rot has been identified in Uganda (Emechebe and Mukiibi 1976), Suriname (Power and Verhoeff 1984), Taiwan (Lin and Chang 1985), Venezuela (Cedeño *et al.* 1990), Zimbabwe (Cole *et al.* 1992), China (Li *et al.* 1993), the USA (Ploetz 1991) and Mauritius Islands (Lutchmeah and Musaphur 1993). It is one of the main diseases affecting *P. edulis* f. *flavicarpa* in most Brazilian producing States and is responsible for a decrease in productivity and constant crop migration (Ponte 1993, Fischer *et al.* 2005a). Based on symptoms and the fungi that have been isolated from affected plants, it may also occur in others countries. Collar rot and Fusarium wilt diseases show similar symptoms on passion flower, and are both caused by species of *Fusarium*. The disease has been reported on *P. edulis*, *P. edulis* f. *flavicarpa*, *P. alata*, *P. ligularis*, *P. maliformis* and *P. quadrangularis* (Ssekyaewa *et al.* 1999; Junqueira *et al.* 2005).

### Symptoms

The first aboveground symptom is mild dieback followed by changing of leaf color to pale green, leaf wilt, defoliation



**Fig. 10** (A) Dieback of the canopy and (B) stem canker of passion fruit caused by *Haematonectria haematococca*.

(Fig. 10A) and finally plant death, resulting from the complete necrotic girdling of the plant collar (Fig. 10B) (Cole *et al.* 2002). Necrosis generally reaches 2 to 10 cm above-ground and may migrate to roots. Tumescence and fissures in the affected collar bark show purple lesion borders, where reddish structures slightly bigger than sand grains, which correspond to the pathogen perithecia, may appear under high relative humidity (Emechebe and Mukiibi 1976). The disease generally affects plants one to two years after planting, although it may occur earlier in replanting areas where the pathogen has previously appeared.

#### Causal agent

Collar rot is caused by homothallic strains of *Haematonectria haematococca* (anamorph: *Fusarium solani*). The fungus usually produces single or groups of reddish perithecia after two weeks in culture medium and after seven days on the surface of cankers. They are ~200 µm in diameter, and produce unitunicate asci, 80 µm long, in which eight bicellular ascospores, 14 µm long, are produced (Hanlin 1990). Pathogen growth under *in vitro* conditions was most intense between 25 and 30°C (Ssekyewa *et al.* 1999). Colonies of the pathogen grow 7.5 mm in diameter day<sup>-1</sup> on oatmeal agar, with abundant aerial mycelium and, eventually, numerous sporodochia; they are cream, aqua or blue (Domsch *et al.* 1980; Nelson *et al.* 1983). The anamorph produces micro- and macroconidia on branched and non-branched monophialides. Microconidia are sparse to abundant, usually one-celled, oval to kidney shaped, and have thicker cell walls than those that are produced by *F. oxysporum*. Macroconidia are abundant, cylindrical, thick-walled and stout, with rounded, foot-shaped or notched basal and blunt or rounded apical cells. Chlamydospores are often abundant and form singly or in pairs. Both ascospores and conidia of the fungus are pathogenic (Emechebe and Mukiibi 1976).

#### Epidemiology

The fungus survives for years as chlamydospores in the soil and may be spread by any practice resulting in movement of infested soil. Infected seedlings are also responsible for spreading the pathogen. Artificial inoculation studies indicate that wounding has a profound effect on collar rot development. Lin and Chang (1985), Cedeño *et al.* (1990), Lutcheah and Musaphur (1993) and Fischer *et al.* (2005a) only reproduced the symptoms of the disease when roots or collar of plants were injured before inoculation. Canker development was always greater in injured plants (Ploetz 1991). Emechebe and Mukiibi (1976) increased the percentage of affected plants by 100% by simply hoeing in bet-

ween rows, a practice that presumably injured roots, thereby providing entry points for the pathogen. The disease is known to interact with *Phytophthora* rot, nematodes, ants and termite attacks (Emechebe and Mukiibi 1976; Lin and Chang 1985; Cedeño *et al.* 1990).

Resistance to collar rot increases as plants age. Emechebe and Mukiibi (1976) reported that 76-100% of the 10-week-old plants that were wounded inoculated wilted, whereas only 28-48% of 12-month-old plants succumbed. Lutcheah and Musaphur (1993) observed that the disease is favored by high temperatures and relative humidity.

Despite the fact that *F. solani* is a polyphagous agent affecting a great variety of plants, studies in Taiwan showed that *F. solani* in passion fruit plants is a specialized genus adapted to *Passiflora* (Lin and Chang 1985).

#### Management

Areas previously presenting the disease should be avoided for new plantings and nurseries. Badly-drained soils have to be avoided and careful irrigation has to be conducted in order to avoid the excess of water, water stress as well as injuries to plant collar and roots. Ssekyewa *et al.* (1999) reported that biweekly drenches of copper oxychloride reduced the number of plants developing collar rot. In Brazil, however, under favorable environmental conditions, the use of fungicides has shown to be unsatisfactory. The use of a resistant rootstock is the best way to deal with the problem in contaminated areas. The rootstock *Passiflora caerulea* used in South Africa is resistant to *F. solani*, *F. oxysporum* f. sp. *passiflorae* and *P. nicotianae* (Terblanche *et al.* 1986; Grech and Rijkenberg 1991; Cole *et al.* 1992), while *P. nitida*, *P. laurifolia*, *P. maliformes* and *P. alata* present partial resistance (Delanoë 1991; Ssekyewa *et al.* 1999; Fischer *et al.* 2005a). There are also resistant *P. edulis* f. *flavicarpa* genotypes selected in India, Taiwan and Uganda (Lin and Chang 1985; Ssekyewa *et al.* 1999).

#### Fusarium wilt

The disease was first reported in 1951 in Australia (McKnight 1951), where it became widely spread, affecting purple passion fruit commercial orchards (*P. edulis*). Fusarium wilt has also been reported in Brazil (Carvalho and Carvalho 1968), Panama (Esquivel and Labrador 1977), South Africa (Grech and Rijkenberg 1991) and Venezuela (Bautista and Salas 1995). Incomplete nature of some reports makes it unclear whether collar rot or Fusarium wilt was present. It occurs on *P. edulis*, *P. foetida*, *P. mollissima* and *P. ligularis* (Gardner 1989).

#### Symptoms

When affected by the disease, the glossy green leaves of young passion fruit plants show a pale green color and mild dieback can be observed. Then, drop of lower leaves, general plant wilt and sudden death take place (McKnight 1951). In adult plants, the disease causes the yellowing of young leaves, followed by plant wilt and death. Symptom development may be unilateral or encompass the entire plant. The vascular system becomes darkened at the root, collar, stem and twig areas, condition that may reach an extent of 2 m above the soil line (Kiely and Cox 1961). The disease typically affects the xylem vascular system, leading to the impermeability of vascular walls and preventing the translocation of water to other plant parts. Under high relative humidity conditions, lesions and fissures can be found in the plant collar and stems, which may be confused with rot collar symptoms (Manica 1981).

#### Causal agent

Fusarium wilt is caused by *Fusarium oxysporum* f. sp. *passiflorae*. In culture, colonies are fast growing (4-7 mm diameter on PDA at 24°C), with sparse to abundant aerial myce-



lium, and white, pink, salmon or purple pigmentation (Gerlach and Nirenberg 1982; Nelson *et al.* 1983). When formed, sporodochia are tan to orange, and sclerotia are blue and submerged. Micro- and macroconidia form on branched and unbranched monophialides. Microconidia are one- or two-celled, oval- to kidney-shaped, and are borne in false heads. Macroconidia are four to eight-celled, sickle-shaped, thin-walled and delicate, with foot-shaped basal and attenuated apical cells. Dimensions of the micro- and macroconidia are  $5-16 \times 2.4-3.5 \mu\text{m}$  and  $27-55 \times 3.3-5.5 \mu\text{m}$ , respectively (Gerlach and Nirenberg 1982). Terminal and intercalary chlamydospores are usually globose, and are formed singly ( $7-11 \mu\text{m}$ ) or in pairs on hyphae or conidia. The species has no teleomorph.

### Epidemiology

The pathogen presents resistance spores, known as chlamydospores, which are important long term survival propagules in the soil. After the chlamydospore germination, the fungus can infect the passion fruit plant, triggering the disease. The fungus penetrates into roots and hypocotyl of plants mainly via injuries (Beckman 1987). The pathogen is spread throughout the plant by microconidia produced in the infected vascular system and is passively transported by the transpiration flow (Nelson 1981). As the disease progresses, the fungus may invade tissues adjacent to the xylem, such as the phloem and cortex, causing external cankers or stem fissures (Nelson 1981). Mcknight (1951) observed that from seven to nine days after the inoculation of seedlings, the youngest open leaf showed colorless nervures. This symptom preceded foliar abscission, which was observed about two weeks after inoculation.

Pathogen spread may occur by means of infected seedlings, produced in contaminated soil. There are no reports on dissemination by seeds until now, although several *Fusarium formae speciales* may be spread by seeds. Inside an orchard, the fungus is spread by soil movements (machines, implements, shoes, etc.) and by runoff or irrigation water. The disease intensity is greater in sandy soils and is favored by high temperatures and relative humidity (Kiely and Cox 1961).

### Management

Plantings areas previously affected by the disease should be avoided. It is recommended the use of healthy seedlings and a careful mechanical or chemical control of weeds in order not to injure roots. The disease can be controlled by using resistant rootstocks, such as *P. edulis* f. *flavicarpa*, *P. alata*, *P. quadrangularis* and *P. macrocarpa* (Groszmann and Purss 1958; Manica 1981), or by using resistant hybrids from crosses between purple and yellow passion flowers. Groszmann and Purss (1958) identified a superior wilt-resistant selection of *P. edulis* f. *flavicarpa* that had the added attributes of resistance to nematodes and Phytophthora root; it was still a standard in commercial production 30 years later.

### Phytophthora root and Crown rot

Crown rot has been reported in Australia (Simmonds 1959), New Zealand (Young 1970), Malaysia (Turner 1974), South Africa (Milne *et al.* 1975), India (Ullasa and Sohi 1975), Panama (Esquivel and Labrador 1977), Brazil (Souza Filho *et al.* 1978), Taiwan (Lin and Chang 1985), the USA (Farr *et al.* 1989), Zimbabwe (Cole *et al.* 1992), Colombia (Varón de Agudelo 1993) and Venezuela (Gonzalez *et al.* 2000). It occurs on *P. edulis*, *P. edulis* f. *flavicarpa*, *P. caerulea*, *P. vitifolia* e *P. foetida* (Simmonds 1959; Turner 1974, Grech and Rijkenberg 1991; Cole *et al.* 1992).

### Symptoms

The disease affects adult plants and nursery plants (Fig. 11A). The symptoms observed are mild chlorosis followed by plant wilt, defoliation and death. The symptoms are the result of root and collar rot, which expose the plant cortical tissue (Fig. 11B) (Cole *et al.* 1992; Varón de Agudelo 1993). Plant intumescence and bark fissures can also be found in the collar (Souza Filho *et al.* 1978).

The occurrence of foliar blight and drop of flowers and fruits has also been reported. According to Ullasa and Sohi (1975), injured leaves show a “burned” appearance. There is a change in leaf color from colorless to pale green, with leaves reaching a light copper color. The affected plant shows burned-like black twig tips and flowers which eventually die. Large grayish-green aqueous spots can be observed in fruits, which easily fall down (Inch 1978).

### Causal agent

The following pathogens were identified as the etiologic agents of the crown rot: *Phytophthora cinnamomi*, in Australia and New Zealand (Simmonds 1959; Young 1970); and *P. nicotianae* (syn.: *P. parasitica*), in Zimbabwe, South Africa, Malaysia, Taiwan, Australia, Venezuela and Brazil (Simmonds 1959; van den Boom and Huller 1979; Lin and Chang 1985; Grech and Rijkenberg 1991; Cole *et al.* 1992; Gonzalez *et al.* 2000; Fischer *et al.* 2005b). *P. nicotianae* under high relative humidity conditions can also infect the plant aboveground part (Ullasa and Sohi 1975). They have fungal-like lifestyles but are in the Kingdom *Chromista*, rather than the *Eumycota* (the true fungi). These pathogens produce a variety of propagules including chamydospores, hyphal swellings, oospores, sporangia and zoospores (Erwin and Ribeiro 1996). *Phytophthora* spp. presents hyaline and coenocytic mycelium.

*P. cinnamomi* affects well over 1000 species of plants and produces distinctive corralloid mycelium. Its non-papillate, non-caducous sporangia are elliptical to ovoid, but are rarely formed in culture. Their dimensions range dramatically ( $11-123 \mu\text{m} \times 11-63 \mu\text{m}$ ), depending on the host and reporting authors. Terminal and intercalary chlamydospores,  $31-50 \mu\text{m}$  in diameter, are abundant in culture and usually formed in botryose clusters. Their cell walls are much thinner than those that are produced by other species. Hyphal



Fig. 11 (A) Damping-off and (B) crown rot of passion fruit caused by *Phytophthora nicotianae*.

swellings can be abundant. *P. cinnamomi* is heterothallic. Oogonia are 21-58 µm in diameter, antheridia are amphigynous, and oospores are plerotic. The cardinal temperatures for growth are 5-15, 20-32.5 and 30-36°C (Ploetz *et al.* 2003).

*P. nicotianae* forms non-caducous ellipsoid, ovoid, pyriforme to spherical sporangia with usually a single papillum (Erwin and Ribeiro 1996). They are produced either singly or in sympodia on stalks that range from 100 µm to 595 µm in length, and are 11-60 µm × 20-45 µm, with a length: breadth ratio of 1.1:1.7. The pathogen forms intercalary and terminal chlamydospores that are 13-60 µm in diameter. Most isolates are heterothallic. Antheridia are amphigynous and spherical or oval, and oogonia are smooth, spherical and 15-64 µm in diameter. Oospores are aplerotic. Its cardinal temperatures for growth are 5-7, 27-32 and 37°C (Ploetz *et al.* 2003).

### Epidemiology

The disease appears in specific spots and spreads from one plant to another. High disease incidence is observed in clay and acid soils during rain periods and when temperatures vary between 26 and 30°C. Zoospores produced inside the sporangia and released in the presence of water are attracted by root exudates. Reaching the root surface, the zoospores encyst and germinate, producing hyphae that colonize the intra and inter-cells of the plant roots, destroying the external cortical tissue, reaching the cambium and avoiding sap circulation. Sporangia production always takes place on the soil surface or on the surface of infected organs, as aeration is essential for their formation. Chlamydospores and oospores are resistance spores capable of surviving in soil and plant tissues for several months. Under favorable environmental conditions and in the presence of a host, chlamydospores and oospores can germinate, originating sporangia that may produce a great number of zoospores (Ploetz *et al.* 2003).

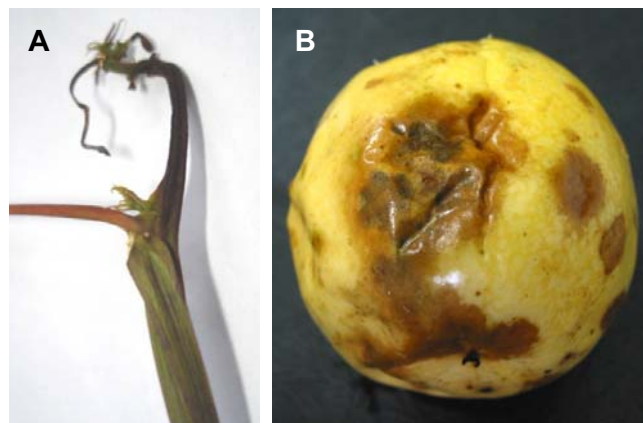
### Management

Besides the prophylactic measures already adopted to control crown rot, the elimination of diseased tissues during the initial stages of the disease and the use of bordeaux mixture are recommended. Applications of fungicides effective against oomycetous organisms directly applied on the plant collar soon after the beginning of the rain season may control the disease (Fischer *et al.* 2005b). Inch (1978) recommended pulverizations with copper oxychloride every seven to ten days to control foliar blight.

*Passiflora caerulea* is more resistant to *P. nicotianae* than *P. edulis* and *P. edulis* f. *flavicarpa* (Terblanche *et al.* 1986; Grech and Rijkenberg 1991; Cole *et al.* 1992). *P. caerulea* was widely used as rootstock for *P. edulis* in an attempt to control the disease in South Africa. However, growers observed that *P. caerulea* is not always resistant, showing high resistance variability to *P. nicotianae*. Moreover, *Meloidogyne* may affect the resistance of *P. caerulea* to *P. nicotianae* (Grech and Rijkenberg 1991). The species *P. suberosa*, *P. foetida* and *P. morifolia* were the most resistant to the disease under greenhouse conditions and were less affected by *H. haematococca* than *P. edulis* f. *flavicarpa*. Their use as rootstock may be a possible controlling measure (Fischer *et al.* 2005a, 2005b).

### Anthracnose

Anthracnose probably occurs wherever this crop is grown and is considered one of the most important passion fruit diseases (Yamashiro 1991; Cedeño *et al.* 1993; Lutchmeah 1993; Wolcan and Larran 2000). The pathogen affects *P. edulis*, *P. edulis* f. *flavicarpa*, *P. alata*, *P. laurifolia*, *P. mollissima*, *P. quadrangularis* and *P. ligularis* (Farr *et al.* 1989; Liberato 2002; Manicom *et al.* 2003). During hot and rainy seasons, in the absence of controlling measures, it causes intense defoliation, twig wilt and fruit rot. In Brazil, An-



**Fig. 12** Symptoms of anthracnose on a passion fruit. (A) Death of the shoots. (B) Rot of fruits.

thrachnose is considered the most important postharvest disease of *P. edulis* f. *flavicarpa*, reducing fruit shelf life (Fischer *et al.* 2007). In planting areas where no control management is adopted and under favorable conditions, up to 80% of plants can die in the second year of the disease (Torres 1983).

### Symptoms

All aerial organs of the plant are attacked (Persley 1993; Goes 1998). Small round light spots that later turn into brown spots, reaching over 1 cm in diameter can be observed on leaves. The centers of the spots become brittle and may break apart. As foliar lesions coalesce, large areas of the leaf die, resulting, eventually, in abscission (Yamashiro 1991). Elongate dark brown spots, up to 4-6 mm in diameter, appear on the twigs and later turn into cankers, exposing the wood. In some cases, lesions can completely surround the twig, making the twig extreme to wilt and die (Fig. 12A).

Affected flowers abort, and immature fruit abscise. Young fruit show oily spots that later become brownish in color. A corklike layer appears on the surface of the spot, which shows a sunken appearance. As fruits mature, round dark spots up to 1 cm in diameter can be observed (Fig. 12B). These spots later turn into soft and sunken rot areas. Lesion may reach large extents on fruits, affecting the pulp and causing the early fruit drop. Lesions on leaves, fruits and twigs often show small black spots called acervuli, which under high relative humidity conditions and average temperatures between 26 and 28°C are covered with an orangish mass formed by conidia soaked in a mucilaginous matrix.

### Causal agent

Anthracnose is caused by *Glomerella cingulata* (anamorph: *Colletotrichum gloeosporioides*). On PDA, colonies are whitish to dark grey with thick to sparse lawns of aerial mycelium (Holliday 1980; Jeffries *et al.* 1990). Conidia are hyaline, one-celled, 7-20 × 2.5-5 µm and either cylindrical with obtuse ends or ellipsoidal with a rounded apex and a narrow, truncate base. They form on light brown conidiophores in irregular acervuli and, upon maturity, appear orange and slimy *en masse*. Acervuli develop in lesions and conidia in acervuli remain viable for long periods, even under adverse climatic conditions. Setae that form in acervuli are brown, 4-8 × 200 µm, and two- to five-celled. The fungus is heterothallic and its teleomorph can be readily induced in culture medium (Wolcan and Larran 2000), but is seldom observed in field conditions, with only two reports in Brazil, on *P. edulis* f. *flavicarpa* and *P. alata* (Yamashiro 1991; Junqueira *et al.* 2005). Perithecia are subspherical, dark brown to black, 90-220 µm in diameter and contain hyaline, unitunicate asci (Cedeño *et al.* 1993; Wolcan and

Larran 2000). Ascospores are unicellular, curved, hyaline and  $14-20 \times 5-6 \mu\text{m}$ .

### Epidemiology

The fungus survives and sporulates in infected tissues and crop residues of passion flower and is most observed in the second planting year. Fungal dissemination in the field is carried out by raindrops, while long-distance dissemination relies on infected seeds, seedlings and cuttings. Long raining periods and average temperatures of  $27^\circ\text{C}$  are the ideal conditions for the occurrence of epidemics. During the winter, even during rainy periods, the incidence of the disease is low in São Paulo State, Brazil (Piza Jr. 1994). Maximum germination of conidia occurred between  $30$  and  $33^\circ\text{C}$  in the dark, and was accelerates between  $22$  and  $25^\circ\text{C}$  in the presence of light (Francisco Neto *et al.* 1994). The incubation time observed in seedlings is six days (Francisco Neto *et al.* 1995).

Host injury increase infection, but is not an obligate requirement (Rocha *et al.* 1996). Quiescent infections occur on immature fruit of *P. alata* and *P. edulis* f. *flavicarpa*, whereby infections stop development after aplerisium formation (Jeffries *et al.* 1990).

Isolates of *C. gloeosporioides* from *P. edulis* f. *flavicarpa* e *P. alata* tested by cross inoculation were more aggressive to their original hosts (Francisco Neto *et al.* 1995). Cross pathogenicity tests of *C. gloeosporioides* of cashew, mango, papaya and passion fruit evidenced that all isolates induced necrotic sunken lesions on fruits, except on passion fruit, which was only susceptible to the passion fruit isolate, suggesting the existence of pathogenic specialization groups (Lima Filho *et al.* 2003).

### Management

Use of pathogen-free seedlings, pruning to eliminate affected areas and improve ventilation and light conditions helps control the disease. Fruit should not be harvest during wet conditions, unduly exposed to the sun, or kept for long in the absence of refrigeration. Pruning should be done when plants are dry, and should be followed with applications of a fungicide. Applications of mixed formulations of protective and curative fungicides are necessary during favorable conditions. Under intense rain periods, fungicides have to be used weekly, while during scattered rain seasons, fungicides have to be used at fifteen-day intervals. Applications can be suspended in dry seasons with no occurrence of dew. Fungicides quoted as efficient against anthracnose are benzimidazole, cupric, dithiocarbamate, chlorothalonil and tebuconazole (Piza Jr. 1994; Phelps 1991).

The fungicides prochloraz and imazalil show the best results for the control of postharvest rots (Benato *et al.* 2002). Studies suggest the use of *Trichoderma* spp. to control the disease in field or postharvest conditions (Rocha and Oliveira 1998). The thermal treatment of *P. edulis* f. *flavicarpa* fruits at  $42.5$  and  $45^\circ\text{C}$  for eight minutes significantly reduces the disease incidence in fruits (Benato *et al.* 2001).

There are few studies on *Passiflora* resistance to anthracnose. *Passiflora nitida* seedlings, when inoculated, are immune to the disease (Oliveira *et al.* 1994). Interspecific hybrids between *P. mollissima* and *P. tripartida*, and *P. mixta* and *P. cumbalensis* have exhibited stable resistance. Studies are needed on the agronomic characteristics of their fruit before they could be used in production (Sanudo-Sotelo and Zuniga-Ravelo 1991).

### Scab

Scab, which is also known as Cladosporium rot, has been reported in Australia (Simmonds 1932), Brazil (Bitancourt 1935), Zimbabwe (Bates 1954) and Venezuela (Rondón *et al.* 1995). The disease can be observed in all Brazilian producing areas and causes significant damages when not controlled (Goes 1998). In nurseries, it can cause death of plants (Torres 1983), while in field conditions it causes the death of twigs and can delay flowering and production, as well as reduce the commercial quality of fruit. The disease affects *P. edulis*, *P. edulis* f. *flavicarpa*, *P. cincinnata*, *P. herbertiana*, *P. nitida*, *P. laurifolia* and *P. amethystina* and is seldom observed in *P. setacea*, *P. giberti* and *P. alata* fruits (Simmonds 1932; Oliveira *et al.* 1994; Junqueira *et al.* 2005). Fruits of *P. subpeltata* show no symptoms (Simmonds 1932).

### Symptoms

The disease mainly affects young tissues of leaves, branches, tendrils, flower buds and fruits. Symptoms on leaves are small round spots, 3-6 mm in diameter. Spots are initially translucent but later become necrotic, showing greenish-grey centers, which correspond to fungal fructification (Fig. 13A). Lesions can perforate leaves or, when they occur on veins, cause them to be deformed; they often cause abscission (Bitancourt 1935). Similar spots may appear on bud sepals or open flowers. High numbers of lesions on flower buds or on peduncles can greatly reduce the number of flower buds (Manicom *et al.* 2003).

Twigs and twig tips initially show lesions similar to the ones on leaves, which later turn into cankers of elongated and sunken aspect that become greenish-grey, where the pathogen fructification takes place. As scar tissue forms, branches become weak and break in the wind (Yamashiro 1991).

On small fruits, symptoms are slightly sunken and dark small circular spots, 5 mm in diameter. On bigger fruits, lesions on fruit skin grow and become corklike, prominent and brownish (Fig. 13B), but do not reach the inner fruit and, consequently, do not affect juice quality (Yamashiro 1991). Several lesions may form on the same fruit, causing it to be deformed and stunted.

### Causal agents

*Cladosporium oxysporum* is responsible in Zimbabwe and Australia and *C. cladosporioides* and *C. herbarum* in Brazil (Bates 1954; Persley 1993; Barreto *et al.* 1996). *C. oxyspo-*

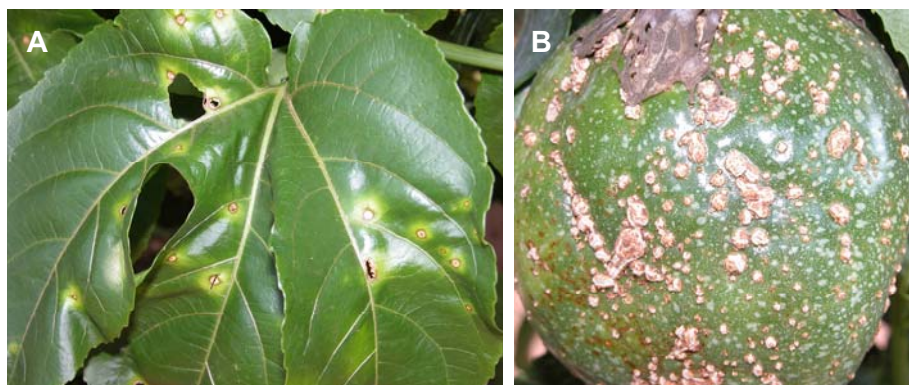


Fig. 13 Scab symptoms on a passion fruit. (A) Leaf (B) Fruit (photos: AM Almeida).

*rum* produces macronematous, straight or slightly flexuous conidiophores that are nodulose, pale or mid pale brown, smooth, and up to 500  $\mu\text{m}$  long and 3-5  $\mu\text{m}$  wide, with terminal and intercalary swellings 6-8  $\mu\text{m}$  in diameter (Ellis 1971). Conidia are cylindrical and rounded at the ends, ellipsoidal, limoniform or subspherical, subhyaline or pale olivaceous brown, smooth, 5-30  $\times$  3-6  $\mu\text{m}$ , and arise simply or in branched chains from terminal swelling, which later become intercalary.

Conidiophores of *C. cladosporioides* and *C. herbarum* are olivaceous brown and bear conidia from the upper to middle portion (Domsch *et al.* 1980). Those of *C. cladosporioides* are 2-6  $\mu\text{m}$  wide and up to 350  $\mu\text{m}$  long, and bare ellipsoid, single-celled, olivaceous brown conidia that are 3-7  $\times$  2-4  $\mu\text{m}$ . Conidiophores of *C. herbarum* are 3-6  $\mu\text{m}$  wide and up to 250  $\mu\text{m}$  long, and bear golden brown conidia, 5.5-13  $\times$  3.8-6.0  $\mu\text{m}$ , that are usually single-celled.

### Epidemiology

Dissemination occurs through infected seedlings, and by wind and sprinkler water. Although conidia are found frequently on seeds, there is no evidence for seed dissemination (Manicom *et al.* 2003).

High relative humidity is needed to promote the infection. Young tissues are more susceptible to the disease than adult tissues (Simmonds 1932). Rocha and Menezes (1997) observed an incubation period of seven days in fruits and of twelve days in leaves. Forty-eight hours after inoculation with *C. cladosporioides*, small necrotic spots appeared on seedlings, which showed burnlike symptoms after two weeks and eventually died in some cases (Barreto *et al.* 1996). The disease severity is higher during springtime, when temperatures are mild. The optimum temperature for the agents varies: *C. oxysporum*, 19.5-24°C; *C. cladosporioides*, 20-28°C; and *C. herbarum*, 28-30°C (Domsch *et al.* 1980; K.G. Pegg, pers. comm.).

### Management

High densities of seedlings have to be avoided in plant nurseries, as well as excessive irrigation. Fungicide applications have to be periodically carried out. The management of adult plants should favor adequate ventilation of plants. Pruning and cleaning of plants have also to be conducted and infected tissues have to be burned, avoiding the transport of infected material. Fungicide applications have to be carried out especially during periods of intense growth and flowering. Fungicides quoted as efficient against the disease are tebuconazole, strobilurin, copper oxychloride, mancozeb, captan and chlorothalonil + copper oxychloride (Piza Jr. 1994; Willingham *et al.* 2002). Menten *et al.* (1993) mentioned the cultivars 'Casca Fina' and 'Australia' of *P. edulis* f. *flavicarpa* as being resistant to the disease. Heredibility of resistance to scab was estimated by Negreiros *et al.* (2004) as 44.7%, whose work involved the selection of at least 10 promising and highly productive progenies.

### Septoria blotch (spot)

Initially described in Peru (Sydow 1939), the septoria blotch has been reported in many countries such as South Africa (Louw 1941), New Zealand (Dingley 1959), Kenya (Ondiekj 1975), Australia (Inch 1978), Venezuela, Trinidad (Punithalingam 1980), Brazil (Yamashiro 1991), Mauritius (Lutchmeah 1993), the USA (Alfieri *et al.* 1994) and Colombia (Trujillo *et al.* 1994). It occurs on *P. edulis*, *P. edilis* f. *flavicarpa*, *P. alata*, *P. quitensis*, *P. macrocarpa*, *P. quadrangularis* (Sydow 1939; Louw 1941; Punithalingam 1980), and weed species such as *P. mollissima* (Trujillo *et al.* 1994). The disease may sporadically cause significant damages, mainly in plant nurseries and crop fields, where the use of chemical control to prevent anthracnose and scab epidemic is not efficient (Yamashiro 1991).



Fig. 14 Septoria blotch symptoms on a passion fruit. (A) Leaf (B) Fruit (photos: AM Almeida).

### Symptoms

Leaves are the most affected organs, showing light brown slightly round necrotic spots of around 4 to 10 mm in diameter (Fig. 14A), normally encircled by a chlorotic halo (Louw 1941). A single lesion per leaf is sufficient to cause abscission, and even leaves without visible symptoms may fall prematurely. According to Yamashiro (1991) and Piza Jr. (1994), when the disease reaches 15 to 20% of leaves in the same plant, partial or even complete leaf abscission is observed. In young twigs, lesions may promote girdling, leading to wilt and twig tips death. Lesions on flowers are similar to those on leaves. The primary infection in the calyx may reach the stalk, causing the early drop of flowers (Louw 1941). The infection may occur at any stage of development in fruits (Inch 1978), which show circular lesions presenting well-defined borders that may reach great extents on fruits, affecting maturation or development. However, damages are only concerned with fruit skin (Fig. 14B). Black punctuations in the center of lesions were also observed and correspond to the pathogen pycnidia. Leaf and fruit abscission, twig wilt and plant death may occur under disease-favoring conditions.

### Causal agents

Three species of *Septoria* are reported as occurring on *Passiflora* species. *Septoria fructigena* has been reported on fruits in Kenya (Natrass 1939) and South Carolina (Farr *et al.* 1989) but, based on its description it is apparently a species of *Phomopsis* (M. Priest, pers. comm.). In 1939, *S. passiflorae* has been described for isolates from diseased passion flower in South Africa (Sydow 1939). Shortly after it was described, Louw (1941) reported a new species in South Africa but, because he was not aware of the earlier report, also used epithet *S. passiflorae*. In 1980, Punithalingam reported that these fungi were distinct, and renamed *S. passifloricola*. Most studies on Septoria blotch mention *S. passiflorae* species, without making a distinction between the two species and without including information on the morphology of the fungus that enabled the identification of the species. However, according to Manicom *et al.* (2003), *S. passifloricola* seems to be more widely spread than *S. passiflora*.

*Septoria passiflorae* produces dark, spherical and subepidermic pycnidia in lesions that are 50-160  $\mu\text{m}$  in diameter. They may erupt and become ostiolate. The short conidiophores bear at their extremities conidia that are one- to four-celled, and filiform at either end or slightly obtuse and round. *S. passiflorae* and *S. passifloricola* can be easily distinguished on the basis of conidial length. *S. passiflorae* has conidia 35-52  $\times$  1.5-2  $\mu\text{m}$  while *S. passifloricola* has conidia 14-22  $\times$  1.5-2 (-2.5)  $\mu\text{m}$  (Cline 2007). The conidia are released in hyaline cirri and are agglutinated by a mucilaginous substance (Holliday 1980).

## Epidemiology

Conidia contained in cirri are spread by water, dew and insects (Inch 1978; Manicom *et al.* 2003). The fungus survives in infected tissues, and mucilage in the cirrus is thought to aid survival. Prolonged rains and mild temperatures favour disease development.

The fungus grows *in vitro* under temperatures varying from 5 to 35°C, showing best development between 20 and 30°C. When artificial inoculations were carried out, no symptoms were observed at 10 or 35°C. The inoculation period ranges from 7 to 15 days, according to the inoculation method adopted (Louw 1941; Trujillo *et al.* 1994).

## Management

Other controlling measures used for other aboveground diseases, such as the use of carbamate and benzimidazole fungicides, are generally enough to avoid damages caused by septorioses in nurseries and field plants. Thiabendazole or thiophanate-methyl + chlorothalonil applied at 15-day intervals showed to be efficient at controlling the disease (Piza Jr. 1994). Although benzimidazoles are effective at controlling septorioses, passion fruit *Septoria* isolates resistant to benomyl have already been verified (Peterson 1977). Thus, it is recommended to use benomyl in a mixture or alternated with fungicides of different modes of action. Bueno *et al.* (2007) assessed the behavior of 47 progenies of *P. edulis* f. *flavicaipa* regarding susceptibility to septoria blotch and found at least three progenies that stood out for their lower incidence and severity of the disease, and which could be further studied aiming at obtaining resistant plants. *P. caerulea* showed resistance to the pathogen (Louw 1941).

## Brown spot

First verified in Australia (Smith 1939), brown spot has been recorded in India (Ram *et al.* 1977), South Africa, Tanzania, Zambia, New Guinea, Fiji (Ellis 1971; Fullerton 1982), Niue Island (Fullerton 1982), Western Samoa (Gerlach *et al.* 1985), Brazil (Ponte 1993), Mauritius (Lutchmeah 1993), Angola (Araújo 1995), Indonesia, Canada, the USA, Hawaii (Manicom *et al.* 2003), and has caused great losses to passion fruit growers in Australia (Smith 1939), New Zealand (Brien 1940) and Uganda (Emechebe and Mukiibi 1975). In Kenya, passion fruit juice production was reduced by 70% between 1966 and 1967 due to the disease (Ondiekj 1975). In Venezuela and Hawaii, the incidence of affected fruits reached 100% in some crops (Aragaki *et al.* 1969). It occurs on *P. edulis*, *P. edulis* f. *flavicaipa*, *P. alata*, *P. cincinata*, *P. quadrangularis*, *P. incarnata*, *P. suberosa*, *P. foetida*, *P. subpeltata* and *P. herbetiana*, and the incidence on yellow passion fruit in areas of high rainfall can be as high as 98% (Rosenberg 1962; Manicom *et al.* 2003; Junqueira *et al.* 2005).

## Symptoms

The two most common brown spot agents produce distinct symptoms. *Alternaria passiflorae* causes reddish brown spots on leaves, with 5 mm in diameter. Under high humidity, spots normally grow larger – reaching more than two centimeters in diameter – become round and zonate. Spores can form a black thin mass covering the middle of the lesion, being more abundant on the abaxial surface (Brien 1940; Holliday 1980). Abscission of affected leaves may occur rapidly, causing intense defoliation. In the twigs, dark brown lesions are more elongated, 2- to 4-cm long, and may cause girdling and death of the terminal portion of these organs (Brien 1940). Slightly circular spots develop on mature fruits or when they are halfway through their growth process. They are reddish brown, sunken and reach 1 to 3 cm in diameter, affecting the pulp and damaging the fruit's commercial value (Fig. 15) (Fullerton 1982).

In contrast, *A. alternata* causes smaller spots, 1-5 mm in



**Fig. 15** Brown spot of passion fruit caused by *Alternaria passiflorae* (photo: M Yamada).

diameter, with chlorotic haloes on leaves and can induce defoliation. The stem lesions it causes rarely kill vines. Spots on fruit have dark green and greasy margins (Manicom *et al.* 2003).

## Causal agent

Nine species of *Alternaria* have been described as pathogens of passion fruit. After *A. passiflorae* and *A. alternata* (Holliday 1980; Goes 1998), the less common species are *A. macrospora*, *A. aliena*, *A. aragakii*, *A. hawaiiensis*, *A. tenuissima*, *A. tropica*, *A. guangxiensis*, *A. bannaensis* and *A. tomato* (Chen and Zhang 1977; Ram *et al.* 1977; Simmonds 1993; Manicom *et al.* 2003).

*A. passiflorae* produces solitary conidia on its host, but chains of up to five in culture (Ellis 1971). They are pale to mid brown, smooth or occasionally minutely verrucose, straight to slightly curved, and obclavate or with the body of the conidium ellipsoidal tapering to the simple or branched beak which is usually the same length or longer than the body. They are 100-250 µm long and 14-29 µm wide at the widest point, have 5-12 transverse and few longitudinal or oblique septa, and are constrict at septa. Conidiophores are produced singly or in groups, straight or flexuous, pale to mid brown, smooth, with several conidial scars, and up to 120 µm long and 6-10 µm wide.

*A. alternata* produce conidia in long chains of ten or more spores. Conidia and conidiophores are medium golden brown. Conidia have short beaks not exceeding one-third the length of the entire spore. They are ovoid, obclavate or obpyriform and 18-63 × 7-18 µm. They can smooth walled or warty, and have three to eight transverse septa and one or two longitudinal septa towards the base. Conidiophores are produced singly or in small groups; and are usually simple, straight or curved, two- to four-celled, up to 50 µm long and 3-6 µm wide. Cardinal temperatures for growth are 2.5-6.5, 25-28 and 31-32°C (Ploetz *et al.* 2003).

## Epidemiology

The conidia are dispersed by wind, water and rain, and, occasionally, by infected seedlings (Manicom *et al.* 2003). The disease is more intense under high humidity and abundant rainfall, along with rising temperatures (Simmonds 1932). Under the conditions found in the Brazilian Cerrado, the disease occurs in fruits during the rainy season and disappears during the dry season (Junquiera *et al.* 2005).

In young plants, after inoculation with *A. macrospora*, the first symptoms are observed four days later while the typical lesions appear ten days later (Ram *et al.* 1977). According to Brien (1940), typical lesions develop in leaves and twigs 22 days after inoculation with *A. passiflorae*, while in fruits they appear 14 days after it. Fullerton (1982) verified that the first symptoms and characteristic lesions display five and nine days after the inoculation of fruits with *A. alternata*, respectively. Infection occurred regardless the presence of injuries.

The pathogen survives in infected leaves, twigs, and

fruits in the plant and on the soil (Brien 1940). Smith (1993) observed that lesions are present in plants throughout the year, in sufficient numbers to ensure the continuity of the inoculum.

### Management

Trimming vines to increase ventilation and penetration by fungicides can reduce disease pressure (Goes 1998). The fungicides recommended are copper compounds, carbamates and strobilurins applied at 7- to 14-day intervals, from the onset of symptoms and at greater intervals when conditions are less favorable (Emechebe and Mukiibi 1975; Willingham *et al.* 2002). Studies also showed that mancozeb + iprodione are effective at controlling the disease under high humidity conditions (Menzel *et al.* 1989). Hutton (1988) verified isolates of *A. alternata* resistant to iprodione when it was used continuously and in isolated way. It is recommended to mix it with mancozeb or copper compounds. The use of more tolerant hybrids to *Alternaria* spp. applied with fungicides allowed better commercial fruit yield than the use of susceptible clones applied with fungicides (Nakasone *et al.* 1975).

### Rust (*Puccinia scleriae*)

Found in Brazil (Albuquerque 1971) and Panama (Esquivel and Labrador 1977), it causes yellow pustules on leaves and twigs which later become brown. High rust severity causes early defoliation and death of branches, reducing the production. Besides *P. edulis*, the disease was verified in *P. glandulosa*, *P. cyanea*, *P. rubra*, *P. serrato-digitata*, *P. suberosa*, *P. tricuspis* and *P. tuberosa* (Viégas 1961; Albuquerque 1971; Liberato 2002). The elimination of weeds of the *Scleria* genera and pulverizations with triadimenol are recommended as controlling measures, once *Scleria* plants are intermediate hosts for the fungus (Gasparotto *et al.* 1993).

### Damping-off (*Rhizoctonia solani* (*Thanatephorus cucumeris*))

The disease causes the damping-off of seedlings, rot of collar and root in adult plants (Inch 1978; Bezerra and Oliveira 1984; Farr *et al.* 1989). High temperatures and humidity, deficient drainage of soil, deep planting, high nitrogen levels and shading favor the disease. Any crop management preventing these conditions contributes to control the disease. In nurseries, it is recommended to disinfect substrates and plant pots. In Western Samoa and Brazil, *T. cucumeris* has been related causing round leaf spots, of light-green color and yellow halo; fungus' hyphae and microsclerotium are observed on foliar lesions (Gerlach 1980; Poltronieri *et al.* 1999).

### Lasiodiplodia rot (*Lasiodiplodia theobromae*)

Fruits display light-brown round spots, which darken and are covered with fungus' mycelium and pycnidia prior to soft rot. The fungus can infect twigs, which causes the darkening of phloem and bark tissues, and final wilt and dry of the twig-end portion after the lesion (Farr *et al.* 1989). The disease has been reported affecting *P. edulis*, *P. edulis* f. *flavicarpa* and *P. quadrangularis* (Roger and Mallamaire 1938; Farr *et al.* 1989; Fischer *et al.* 2007).

### Sclerotinia rot (*Sclerotinia sclerotiorum*)

Plant collar or affected twigs rot, causing plant death or wilt of twigs. Under high humidity conditions, white cottoned mycelium and dark sclerotium (when mature) develop over the lesions (Kagiwata 1990). In Australia, remarkable damages happened in colder months (Blackford 1944).

### Flower rot (*Rhizopus stolonifer*)

Symptoms include dark spots that are water soaked on the interior of the flower bud, especially on the sepals and petals. Later, spots occur over the entire flower bud, and dark grey mycelia and sporangia develop. The flower bud becomes putrid and abscises easily (Manicom *et al.* 2003). Flower rot occurs mainly in the summer, during periods of prolonged rain. Goes (1998) related losses of up to 63% of the flowers of *P. alata* in Brazil. This disease has also been observed causing fruit rot in passion fruit.

### Phomopsis rot (*Phomopsis tersa*)

This disease has been reported in *P. edulis* and *P. edulis* f. *flavicarpa*, affecting leaves, twigs and specially fruits, damaging up to 40% of the production (Sutton 1980; Lutchmeah 1992). Two to three days after harvest, the tissue around the stalk collapses and depresses, turning brown. White mycelium grows over the lesion and covers the stalk. Ten days after harvest, the whole fruit is affected, with display of black fungus fruit bodies on the surface near the stalk. Internally, the rot expands from the stalk to the endocarp and seeds. The fungus enters the fruits mainly through the cut in the stalk, although it can also penetrate the fruit via skin injuries (Lutchmeah 1992).

## DISEASES CAUSED BY NEMATODES

Several nematode species can be found associated with passion fruit plant roots. Attacks by *Meloidogyne* spp. are characterized by the formation of root-knots (Fig. 16) and cysts resulting from the toxic action of certain substances produced and injected by the nematode; the root system also becomes deficient, making the absorption of water and nutrients by the plant more difficult. Consequently, plants show lower growth and foliar yellowing with reduced productivity and longevity. According to Ritzinger *et al.* (2003), some Passifloraceas can present root-knots resulting from infection, but the infection may not progress. Thus, *P. edulis* f. *flavicarpa* and *P. caerulea* show root-knot, but they are not considered hosts, as there is no reproduction of nematodes. On the other hand, *P. alata* and *P. quadrangularis* show root-knots and mass of eggs, and are considered excellent hosts. In South Africa, *M. javanica* is considered a serious pathogen for both yellow and purple passion flower, making plant more susceptible to droughts (Villiers and Milne 1973).

Damages caused by *Rotylenchulus reniformis* are similar to those from *Meloidogyne*, except for the formation of root-knots (Villiers and Milne 1973). An assessment carried out in 1999 in the Brazilian cerrado revealed that *R. reniformis* was present in 36% of the samples collected from declining two-year-old plants, while *Meloidogyne incognita* and *M. arenaria* were present in 47% of symptomatic samples (Sharma and Junqueira 1999). In Fiji, *R. reniformis* was reported to cause foliar chlorosis and found in 84% of



**Fig. 16** Root-knot nematode on yellow passion fruit caused by *Meloidogyne incognita*.

the areas that were surveyed. In Brunei, nematode has been quoted as an agent predisposing plants to crown rot caused by *Phytophthora* spp. (Perregrine and Yuntun 1980). Other nematodes infecting passion flower, though not causing significant damages, are *M. javanica*, *Scutellonema trancatum*, *Helicotylenchus* sp. and *Pratylenchus* sp. The use of healthy seedlings, crop rotation with plants that are not hosts or bad hosts to nematodes, solarization, fallow and nematicides are recommended measures to control nematodes.

## PERSPECTIVES

Passion fruit plants may be severely affected by several diseases caused by fungi, prokaryotes, virus and nematodes. Some of these pathogens spread quickly and whenever susceptible tissues and favorable environment are available they may become a limiting factor for the crop. They reduce plant longevity and the quantitative and qualitative production of fruits.

Despite the economic importance of many diseases mentioned in this review, most published studies are limited to the identification of the causal agent and some epidemiological aspects. With a few exceptions, what is observed is the absence of integrated research programs aimed at the development of methods for the continuous management of the main diseases affecting passion fruit plants. This situation is partly due to the fact that passion fruit production is typical of small growers, and for many of them, it is not considered a primary crop. When most growers from a passion fruit producing region acknowledge the importance of combined actions to minimize the hazardous effects of pathogen dissemination, some of them abandon their orchards worsening the disease problems for the remainder growers. Some stop the production for a while and others move to a different production area, lending a nomadic character to this crop. Due to these reasons there are no strong unions or agro-industrial associations promoting the necessary interaction among those involved in large scale production. These facts are responsible for the small number of studies on passion fruit diseases and make it difficult for researchers to receive financial support for long-term projects on passion flower diseases.

At present, however, adoption of available crop management practices could, by itself, improve this scenario. These include the use of healthy seeds and seedlings, the monitoring and control of pests and weeds, the proper trimming of plants, the conduction of parceled and balanced fertilization, the manual pollination of flowers and adequate irrigation, which will contribute effectively to better plant development. As a result, plants become more resistant/tolerant to diseases, with improved longevity and productivity. In orchards whose production is aimed at the external market, sustainable production practices, such as the conservation of natural resources and substitution of polluting inputs, have to be adopted.

However, such alternatives do not represent a final solution to the problems caused by passion fruit diseases. Some countries, such as Brazil, can count on improved and high quality passion fruit varieties, but plants show low or no resistance and/or tolerance to diseases. Longer research programs, which explore the great genetic variability of *Passiflora* species may bring significant benefits to the identification of sources of resistance that could be transferred to commercial cultivars.

It is possible to predict that vigorous rootstocks, compatible with the main species grown nowadays, will minimize the action of root pathogens in the short run. Thus, it is necessary to reduce production costs of grafted seedlings significantly and to lengthen orchard longevity in order to amortize total costs. As hypocotyl grafting of seedlings has already shown to be very effective, this process may complement conventional grafting on a commercial basis (Nogueira Filho 2003).

In the long run, genetic breeding programs may select species resistant to aboveground pathogens to be used in

interspecific crossings aimed at developing more resistant varieties. Even if there is eventual chromosomal incompatibility among species, the use of biotechnology will be useful to identify, map and clone resistance genes that can be transferred to cultivated species by means of genetic transformation.

The genetic transformation of *P. edulis* f. *flavicarpa* employing pathogen derived genes is already being studied with good perspectives of success for the production of transgenic plants resistant to CABMV (Alfenas *et al.* 2005; Trevisan *et al.* 2006). Genetic transformation with the Atacin A gene and anti-apoptotic baculovirus p35 genes have also been studied to obtain transgenic passion fruit plants resistant to *X. axonopodis* pv. *passiflorae* (Castro 2005; Freitas *et al.* 2007).

Finally, the integration of other research lines has to be stimulated in order to solve questions related to passion fruit sanitary issues, such as the evaluation of new fungicides for the chemical control of some diseases, once such studies comply with the current trends of respecting the ecological balance, human and animal health.

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