OFFERED REVIEW

BACTERIAL DISEASES THAT MAY OR DO EMERGE, WITH (POSSIBLE) ECONOMIC DAMAGE FOR EUROPE AND THE MEDITERRANEAN BASIN: NOTES ON EPIDEMIOLOGY, RISKS, PREVENTION AND MANAGEMENT ON FIRST OCCURRENCE

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SUMMARY

Bacterial diseases are difficult to control (both chemically and biologically), and are restrained primarily by preventive measures. Most important risk factors for the introduction or spread of bacterial diseases in Europe and the Mediterranean basin are imported infected planting material and infected insect vectors. In this review the epidemiology, management and main risks of several emerging bacterial diseases approaching or already present in Europe, their causal organisms and vectors will be highlighted, especially: (a) Citrus huanglongbing (= Citrus greening), caused by the heat-tolerant "Candidatus Liberibacter asiaticus" and heat-sensitive "Candidatus L. africanus". Both liberibacters and the respective psyllid vectors Diaphorina citri and Trioza erytreae are present in the Arabian peninsula, with recent reports of huanglongbing occurring in Iran, Mali, Ethiopia and Somalia. T. erytreae is already present on some Atlantic Ocean islands; (b) leaf scorch and leaf scald diseases of grape and different fruit and ornamental trees, caused by Xylella fastidiosa. For this pathogen, although its presence has not been confirmed in Europe or the Mediterranean basin, local possible vectors such as Cicadella viridis and Philaenus spumarius occur; (c) Citrus canker caused by Xanthomonas citri pv. citri, the most severe form of which, the so-called Asiatic, is already present in Iraq, Iran, Oman, Somalia, United Arab Emirates (UAE), Saudi Arabia, Yemen and Reunion. Outbreaks and/or risk and (possible) emerging character of some other bacterial pathogens not yet present in Europe (i-iv) or already present (v-xvi) are also highlighted: (i) black spot of mango, Xanthomonas citri pv. mangiferindicae, present in UAE and Reunion; (ii) bacterial blight of pomegranate, X. axonopodis pv. punicae, emerging in India; (iii) bacterial blight of guava, Erwinia psidii, emerging in Brazil; (iv) bacterial spot of passion fruit, X. campestris pv. passiflorae, emerging in Brazil; (v) stem rot and leaf spot of maize and center rot of onion, Pantoaea ananatis, an emerging problem for onion in the USA, isolated from seed in South Africa; (vi) almond witches’ broom, “Candidatus Phytoplasma phoenicium” killing thousands of trees in Lebanon and Iran; (vii) potato stolbur, “Candidatus Phytoplasma solani”, spreading from Eastern Europe westwards; (viii) zebra chip disease of potato and yellows of carrot caused by “Candidatus Liberibacter solanacearum (synonym Ca. Liberibacter psylllourous). The potato strains occurs only in North and Central America and New Zealand, but the carrot pathogen has been reported from several Scandinavian countries and Spain with the respective psyllid vectors Bactericera cockerellii and Trioza apicalis; (ix) an apparently 'harmless' “Candidatus Liberibacter europaeus” found in the pear psyllid (Capopsylla pyri) in Italy; (x) bacterial fruit blotch of cucurbits, Acidovorax citrulli, seed-transmitted and the cause of outbreaks in Europe, Turkey and Israel; (xi) a new strain of the potato stem rot bacterium, provisionally named Dickeya solani, emerging in several north-western European countries and Israel; (xii) Stewart’s disease or bacterial wilt of maize, P. stewartii subsp. stewartii, spread by the corn flea beetle Chaetocnema pulicaria, observed in several European countries in which it has not become established due to the absence of vector; (xiii) renewed outbreaks from 2008 of Pseudomonas syringae pv. actinidiae, the agent of bacterial blight, especially on Actinidia chinensis (yellow kiwifruit) but also on A. delicosa in central Italy and, since 2010, in France; (xiv) bleeding canker of horse chestnut, Ps. syringae pv. aesculi, emerging in western Europe; (xv) bacterial canker of stone fruits caused by X. arborecola pv. pruni, with recent outbreaks in Switzerland, Spain (on almond) and in the Netherlands on cherry-laurel (Prunus laurocerasus); (xvi) bacterial leaf spot of poinsettia, X. axonopodis pv. poineetticola observed in greenhouses in several north-western European countries. Ornamental and wild hosts may play an important role in spreading diseases and maintaining the pathogens and their vectors in the environment. These plants should be included in surveys. Rapid and reliable diagnosis remains a key issue, as well as breeding for resistance. All pathogens mentioned are emerging threats, with real risks of introduction and, in some cases, closely approaching or already present in the...
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Mediterranean basin and/or Europe. Introduction of susceptible wild hosts and susceptible cultivars of cultivated hosts must be avoided, as they often lead to introduction of pathogens or to outbreaks due to a ‘jump’ of local pathogen strains to very susceptible cultivars. Introduction of vectors should also be prevented as history teaches that vectors appear first and the pathogens a few years later. Examples of these events are given in the description of the various disease-pathogen combinations. An efficient prevention and control strategy of the diseases mentioned in this review should be based on the so-called pathway protection, i.e. regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-regulated pests, where the following conditions should be enforced and controlled: (i) place of production should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

Key words: quarantine, risk assessment, geographical distribution, host plants, plant pathogens.

INTRODUCTION

Bacterial diseases are often a destructive and/or a major constraint for many crops. Substantial killing of the host and yield losses, often over 50% result from infestations caused by established pathogens such as Erwinia amylovora, Pseudomonas syringae pv. syringae and pv. morsprunorum, Agrobacterium tumefaciens (syn. Rhizobium tumefaciens) Xanthomonas arboricola pv. pruni, Candidatus Phytoplasma mali, Grapevine flavescence dorée phytoplasma, Grapevine bois noir phytoplasma, “Candidatus Phytoplasma pyri” and “Candidatus P. prunorum” (European stone fruit yellows). Quarantine pathogens sometimes spread out of contained loci, presenting a long-term threat to other EU countries (e.g. Xanthomonas arboricola pv. pruni spreading out of France and Italy) and others are an emerging threat outside Europe (Xylella fastidiosa, Candidatus Liberibacter spp. agents of citrus huanglongbing).

Bacterial diseases are difficult to control (both chemically and biologically) and are restrained primarily by preventive measures such as hygiene, use of healthy planting material, good cultural practices and avoidance of risky planting sites. Moreover, they are easily spread by (surface) water, planting material and contaminated agricultural implements/machines and by aspecific or specific insect vectors. Most important risk factors for the introduction of bacterial diseases into Europe are imported infected planting material and naturally spreading infect-
MAIN EMERGING BACTERIAL DISEASES

Citrus huanglongbing caused by “Candidatus Liberibacter” species. General. Huanglongbing or HLB (Chinese for yellow shoot disease) of citrus is caused by a non-culturable, fastidious, phloem-inhabiting, Gram-negative bacterium belonging to the α-Proteobacteria and to the genus “Candidatus Liberibacter”[**] [originally the genus was named Liberobacter (Bové, 2006)]. This disease (Fig. 1A-C) was formerly known as citrus greening yellow dragon disease, mottle leaf disease, ‘likubin’ or vein phloem degeneration. It is one of the most destructive diseases of cultivated citrus for which no effective control is available with the consequence that citrus production declines in all areas where the disease occurs. Lin (1956) determined that this disease is graft-transmissible and described it for the first time in China. The origin of huanglongbing is not clear, probably Asia (India or China) or Africa (Gottwald et al., 2007). For a thorough general review on the disease and its causal organism see Bové (2006) and for a review on its epidemiology see Gottwald (2010).

Three different Liberibacter species causing Huanglongbing have been described based only on 16S rRNA and whole genome sequences information, because these organisms cannot be cultivated in vitro:

a. “Candidatus Liberibacter asiaticus” (Las), originally described as Liberobacter asiaticum, the most aggressive species, is heat-tolerant and stands temperatures above 30°C. The disease caused by this species is found mainly in lowlands where it is transmitted by the heat-tolerant psyllid Diaphorina citri Kuwayama (Fig. 1D,E) [a recent report mentions the black psyllid, Diaphorina communis (Fig. 1D,E) (a recent report mentions the black psyllid Diaphorina citri Kuwayama (Fig. 1D,E) (a recent report mentions the black psyllid Diaphorina communis, as a vector in Bhutan (Donovan et al., 2011)]. Las is widespread in Asia, the Arabian peninsula, Mauritius and Reunion islands and, since 2004, in Brazil, Sao Paulo state (in Brazil some 10% of the infected plants are infected by this Liberibacter species, others are infected by the third species, Ca. L. americanus (Teixeira et al., 2005)). It occurs since 2005 in Florida (USA) (Halbert, 2005).

b. “Candidatus L. africanus” (Laf), described as Liberobacter africanum, less aggressive and heat-sensitive. The disease induced by Laf is suppressed after exposure to temperatures above 30°C, therefore it occurs in the tropics at elevations higher than 700 metres above sea level. Laf is widespread in Africa (Somalia, Ethiopia and Cameroon) and occurs also in Reunion, Mauritius, and Yemen. Its vector is the heat-sensitive psyllid Trioza erytreae Del Guercio.

c. “Candidatus L. americanus” (Lam), closely related to Las but heat-sensitive. It occurs in Brazil and is transmitted by the psyllid D. citri.

A recently described subspecies, “Candidatus Liberibacter africanus” subsp. capensis, was isolated from a symptomless ornamental rutaceous tree (Calodendrum capense) in the Western Cape province (South Africa). In the article describing this subspecies, the genus name Liberobacter was changed for all species into Liberibacter (Garnier et al., 2000). Ca. L. africanus subsp. capensis appears to be widespread in C. capense in South Africa, but is not transmitted to citrus (Phahladira et al., 2012) The complete genome sequence of Las is available (Duan et al., 2009) and, recently, differences between the Asiatic (China) and North American strains of Las were reported (Chen et al., 2010). Liberibacter species are apparently not seed-transmissible (Hartung et al., 2010; Hilf, 2011)

Some biological traits of the psyllid vectors. Both Diaphorina citri and Trioza erytreae feed on the phloem and are experimentally able to transmit both the Asian and African HLB. Fourth and fifth instar nymphs and adults can acquire and transmit the bacteria during their whole life, after a latent period of ca. 10 days, after feeding times varying from 15 to 20 min for D. citri to 24 h for T. erytreae. Multiplication in the vector has not clearly been demonstrated, the bacteria, however, can be found in large amounts in the haemolymph and salivary glands. For T. erytreae there is evidence that transovarial transmission occurs. Other psyllids that thrive on citrus were not found to transmit the bacteria, except for the black psyllid (Diaphorina communis) which is a Las vector in Bhutan (Donovan et al., 2011). Psyllids like to feed on new vegetation flushes the presence of which constitutes a risk for transmission. Although D. citri does not tolerate frost very well, it survived frosty days of up to -5°C in Florida (Bové, 2006; Bralnsky and Rogers, 2007; Gottwald, 2010; Gottwald et al., 2007; Halbert and Manjunath, 2004; Manjunath et al., 2008).


The disease caused by the psyllid D. citri in all countries mentioned. South east Asia: Cambodia, China (including Hong Kong), Indonesia, spreading along southern islands of Japan (Shinohara et al., 2006), Laos, Malaysia, Myanmar, Philippines, Taiwan, east Timor, Thailand, and Vietnam; Indian subcontinent: Bangladesh, Bhutan, India, Nepal, and Pakistan; Western Asia: Iran in Sistan-Baluchistan and Hormozgan provinces (Faghihi et al., 2009; Salehi et al., 2012); Indian Ocean: Comoros Islands, Madagascar, Mauritius, Re-

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* There is a report of successful cultivation, but it was dependent on still unknown growth factors, and cultures did not survive (Sechler et al., 2009).

** Phytoplasmas have been found in plants showing HLB symptoms in some cases (Bové et al., 2008).
union and Sri Lanka; *Arabian peninsula*: Saudi Arabia, Yemen in the south west along the Red Sea; *Africa*: Northern Ethiopia (De Bac et al., 2010); *South America*: Brazil, mainly Sao Paulo state, since 2004; *Caribbean*: Cuba (Martinez et al., 2009), Dominican Republic (Matos et al., 2009), Belize (Manjunath et al., 2010); *North America*: USA (Florida since 2005; Louisiana, 2008; Georgia and South Carolina, 2009; California 2012); Mexico, 2009. (http://www.pestalert.org/oprDetail.cfm?oprID=382; http://www.pestalert.org/oprDetail.cfm?oprID=321&keyword=citrus%20greening); http://www.pestalert.org/oprDetail.cfm?oprID=516, http://www.pestalert.org/oprDetail.cfm?oprID=401)


Laf and Las. Ethiopia, Mauritius, Reunion, Saudi Arabia and Yemen host both vectors (*D. citri* and *T. erytreae*) and both pathogens.

Lam. Brazil. Lam has also been reported (but not confirmed) from one of 97 citrus leaf samples from eight provinces of southern China (Lou et al., 2008).

(ii) Vectors. *D. citri* (EPPO, 2005c). *South-east Asia*: Cambodia, China (including Hong Kong), Indonesia, southern islands of Japan, Macau, Malaysia, Myanmar, Philippines, Taiwan, Thailand, and Vietnam; *Indian sub-continent*: Afghanistan, Bangladesh, Bhutan, India, Nepal, and Pakistan; *Indian Ocean*: Comoros Islands, Madagascar, Mauritius, Reunion and Sri Lanka; *Arabian peninsula*: Saudi Arabia (Wooler et al., 1974), Yemen, Oman; *South America*: Argentina (since 1984 in northeast, since 2006 in north-west, Ramallo et al. (2008)), Brazil (since the 1940’s), Venezuela; *Central America*: Honduras, Belize, Costa Rica; *Caribbean*: Cuba (1999), Haiti (2000), Guadeloupe (1998) (Étienne et al., 1998), Bahamas (1999), Cayman Islands (2000), Virgin Islands and Dominican Republic (2001), Puerto Rico (2002) (Halbert and Núñez, 2004); *North America*: USA Florida (1998), Alabama, Georgia, Mississippi, South Carolina, Louisiana, California (USDA 2010), Texas (2001) (French et al., 2001); Mexico (2009); *Pacific Ocean*: Hawaii, Maui (Conant et al., 2007)

*T. erytreae*. *Africa*: Burundi, Cameroon, Central African Republic, Ethiopia, Kenya, Malawi, Nigeria, Rwanda, Somalia, South Africa, Sudan, Swaziland,

Fig. 1. A. Sweet orange with typical symptoms of huanglongbing or citrus greening (source EPPO, J. Bové). B. sweet orange leaves with more or less typical huanglongbing-induced yellowing. *Sporplasma citri* or zinc deficiency can cause similar symptoms (source EPPO, J. Bové). C. Mandarin fruits with typical greening (courtesy of J. Gottwald). D. Adult *Diaphorina citri* vector of "*Candidatus Liberibacter asiaticus*", the Asian huanglongbing strain. E. Nymph of *D. citri*. Size of the adult 3-4 mm [Source Co-nant et al. (2007)].
Sweet oranges, mandarins and tangelos (species and forms are or can be host of Liberibacters. or similar or orange jasmine (of..., rough lemons, kumquats (Fortunella limettioides) severe symptoms. Lemons, grapefruits, C. limonia, C. paradisi) are generally the most susceptible showing on limes and pummelos (C. inaequalis) and citrus are less severely affected. Symptoms are mild on limes and pummelos (C. grandis). However, in Brazil and the USA all commercial Citrus species have a very similar susceptibility. Other confirmed hosts are Limonia acidissima, Murraya paniculata or orange jasmine (often used as ornamental and for hedges) and the related or similar M. exotica, M. (Bergera) koenigii, Severinia buxifolia and Vepris lanceolata (= V. undulata = Todella lanceolata)

(i) Liberibacter species. Rutaceae: Most Citrus species and forms are or can be host of Liberibacters. Sweet oranges, mandarins and tangelos (C. reticulata x C. paradisi) are generally the most susceptible showing severe symptoms. Lemons, grapefruits, C. limonia, C. limettioides, rough lemons, kumquats (Fortunella spp.) and citrons are less severely affected. Symptoms are mild on limes and pummelos (C. grandis). However, in Brazil and the USA all commercial Citrus species have a very similar susceptibility. Other confirmed hosts are Limonia acidissima, Murraya paniculata or orange jasmine (often used as ornamental and for hedges) and the related or similar M. exotica, M. (Bergera) koenigii, Severinia buxifolia and Vepris lanceolata (= V. undulata = Todella lanceolata)

(ii) Vectors (D. citri and T. erytreae). Rutaceae, all cultivated and wild species listed above and Clausena anisata (= C. inaequalis) and Zanthoxylum capense (= Fagara capensis).

Symptomatology. Symptoms (Fig. 1A-C) may be confused with those of other diseases and disorders. More typically, the first symptoms are one or more yellowing shoots. Leaves are asymmetrical when the two leaf halves are compared and exhibit well-defined yellow areas, called blotchy mottling, that may also occur on fruits. With time, yellow spots may intensify, resembling very much zinc deficiency. Veins may become corky, giving the leaves a thicker appearance. Fruits are often smaller, asymmetrically misshaped (lippedoid) and show persistent green areas, especially at the stylar end. When infected fruits are cut, yellow-brown vascular bundles and necrotic seeds may be observed. The final stages of the diseases are characterized by severe leaf and fruit drop, and the fruit quality impaired. Yield losses may be 30-100% and, within 7-10 years from planting, the groves may become unproductive, decreasing yield losses in 10-12 years, orchards may become unproductive, decreasing yield losses in 10-12 years, orchards may become unproductive, decreasing yield losses in 10-12 years, orchards may become unproductive, decreasing yield losses.

Detection and diagnosis. In the early years detection and diagnosis was mainly based on symptoms, electron microscopy and biological indexing. A monoclonal antiserum was developed but proved too specific. PCR (classical, nested, real-time, qualitative real-time and multiplex) is now the main confirmatory test and is routinely used in many areas, allowing also detection in insects and of latent infections in plants (Benyon et al., 2008a; Li et al., 2006a, 2007; Teixeira et al., 2008). Okuda et al. (2005) have developed a species-specific multiplex TaqMan (real-time) PCR for Las, Laf and Lam (with COX primers/probe for the host plant cytochrome oxidase gene as internal control). For a very sensitive combination of nested PCR and TaqMan (real-time) PCR in a single-tube test see Lin et al. (2010) and a real-time PCR protocol based on prophage genes, that is useful for world-wide detection, also in difficult hosts, see Morgan et al. (2012).

Epidemiology. Main sources: Bové (2006); Gottwald (2010); Gottwald et al., (2007). HLB epidemics develop rather slowly in time (several to more than 12 years), but the disease may spread quite rapidly in a grove (in several years trees may show severe symptoms and within 7-10 years orchards may become unproductive, depending on the age of the trees and the presence and number of vectors). A complicating factor is that many trees may have been already latently infected for a considerable time before symptoms become obvious. The number of latent infected trees may be two- to manifold as compared to symptomatic trees, which hinders early detection of HLB and frustrates eradication campaigns. Spread of vectors and disease has a tree-to-tree pattern although also further spread (a few kilometres) from an infection focus may be possible. Psyllids migrate mostly when host plants are flushing. Long distance dispersal is by infected planting material or by infected psyllids accidentally transported by man on plant material or otherwise. The presence of HLB in a tree may be sectorial, thus the bacteria may not be detectable in the symptomless part. How far psyllids can be carried by high air streams and spread over long distances is still unknown, but its occurrence cannot be excluded. Seed transmission of virulent bacteria has not yet been demonstrated.

Damage and losses. Since resistance against HLB is a dangerous and devastating disease and, as yet, no sources of resistance have been found or created. Yield is decreased, mainly because of reduced growth and fruit drop, and the fruit quality impaired. Yield losses may be 30-100% and, within 7-10 years from planting, the groves may lose productivity completely. It has been calculated that ca. 100 million trees have been killed in south-east Asia, India, Arabian peninsula, and South Africa, leading to decline of the citrus industry in these areas. In southwestern Saudi Arabia, sweet orange and mandarin have practically disappeared already during the 1970s. Since 2004, ca. 1 million trees have been destroyed in Brazil due to HLB infection. In Florida HLB was detected for the first time in 2005 and by 2009 it has spread to most citrus-growing areas, so that eradication efforts were given up (Bové, 2006; Gottwald, 2010) and the select agent status for all three Liberibacter species by USDA APHIS abandoned.
Main risk factors. HLB and its vectors have not yet been reported from the European mainland or the Mediterranean basin, where there are no local psyllid vectors known. Long distance spread of Liberibacter spp. and vectors is possible and will mainly be with planting material. In Kenya, infected breeding material from abroad was suspected to be the source of initial infection (Magomere et al., 2009). Note that HLB is approaching the Mediterranean basin mainly from three sides: (i) Saudi Arabia, where both Laf and Las are present and could move further north (e.g. also with people travelling to and from Mecca), possibly also for Las and its vector should global warming continue. Note that Las and its vector could also establish in subtropical Florida; (ii) Africa, where Laf and its vector are already present in Ethiopia and Somalia and Las is established in north Ethiopia; (iii) Iran, where Las has been reported in 2009. Note that D. citri was present in Florida since 1998 and HLB followed in 2004 (also see Bové, 2006), and that the Laf vector is already present in the Atlantic, i.e. the Canary, Madeira, Porto Santos and St. Helena islands. Murraya paniculata (orange jasmine), a popular landscape plant, played an important role in spread of HLB and D. citri in Florida (and probably also to other states), also via garden centres (Manjunath et al., 2008). Murraya species of Asian origin have been marketed (also in bonsai form) in Europe for quite some years. Infections may go unnoticed for several years after they became established due to latency or overlooking of the early symptoms. HLB is suspected to have been present several years before its official detection in Florida in 2005 (Gottwald et al., 2007; Gottwald, 2010).

Prevention and management following first introduction. Once established, HLB bacteria and vectors are very difficult to control. Main strategies are geographical isolation and certification/indexing programs for budwood sources and nursery production in insect-proof greenhouses, chemical and/or biological vector control and intensive surveying (by visual inspection and laboratory testing) with removal and destruction of infected trees (both visually and latently infected trees) wherever possible. Healthy budwood can be obtained by shoot-tip grafting, or alternatively by heat treatment, e.g. water-saturated hot air at 49°C for 50 min and fumigation of budwood against HLB vectors is possible (EPPO, 1988). The visual inspections in the groves should be very intensive and performed from moving inspection platforms. Chemical sprays, for vector control, to be effective, should also be performed frequently. Antibiotic treatment has been abandoned completely as it was not effective and is not human and environmental friendly. Biological control was effective in Reunion Island, with hymenopterous ectoparasites Tamariixia dryi for Laf and T. radiates for Las, probably because hyperparasites of the parasite were absent. At present the only successful prevention/control methods appears to be the one followed in São Paulo state in Brazil, following the introduction of huanglongbing in 2004. The control program that was immediately installed in 2004 by responsible organisations, became very successful after eight years, when disease incidence on more than 200,000 ha was reduced to less then 1%. This was because: (i) at the beginning of the program the disease incidence was still low (less then 8%) and (ii) it could be executed on large, easy to handle farms (>500 ha). The basic principles of the so-called Three-Pronged System (TPS) are: (i) intensive vector control, (ii) reduction of inoculum by tracing, identifying and removing infected trees, and (iii) production of healthy nursery stock in covered, insect proof nurseries and their use for re-planting (Bové, 2012; Belasque et al., 2010).

Genetic resistance development has started, but is still in its infancy (Grosser et al., 2008). Still, breeding for resistance will remain one of the most important ways to control this disease and those described in the following paragraphs.

HLB bacteria and their vectors should be prevented from entering the Mediterranean basin by strict inspection and laboratory testing of imported breeding material from risk areas. In the European Union the importation of citrus planting material from third (non-EU) countries is already prohibited (Annex III A of Directive 2000/29/EC and EPPO (EPPO, 1988) and the introduction of plants for planting and cut citrus branches from countries where HLB or its vectors occur should also be prohibited. However, when such material is imported, it should be fumigated and quarantined for at least two years. Since Liberibacter spp., D. citri and T. erytreae are quarantine organisms included in the EPPO A1 list and EC Annex II/1a list, the following additional measures can be enforced [see also Bassanezi et al., 2008; Bové, 2006; EPPO, 1988; Gottwald, 2010; USDA, 2010. For (pro-active) measures advised by the Australian government, see http://www.agnet.org/library/eb/607/):

- Murraya spp. and ornamental citrus spp. already present in garden centres or in plantations should be surveyed, including vector sampling. In suspect areas this could also been done in (young) citrus orchards.
- Importations from risk areas of these plants strictly inspected and laboratory-tested.
- Proactive training of diagnosticians, surveyors, nursery managers and producers.
- Production in citrus nurseries should be in secure, insect-proof screenhouses.
- Once an introduction has occurred, infected trees should be destroyed and vectors controlled by chemical insecticides.
- When executing severe eradication programs, governments should consider compensation or facilitate insurance to secure full cooperation also from small growers and private tree-owners.
– Further training of all parties involved and development of a public information program.

**Leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by *Xylella fastidiosa***.

*General.* The bacterium *Xylella fastidiosa* is a xylem-inhabiting, vector-transmitted, very slow-growing, Gram-negative bacterium. It was cultured and described for the first time in 1987 in the USA as the cause of Pierce's disease (PD) of grapevine (disease observed already in 1884) and as the cause of phony peach disease (PPD) in peach, *Prunus persica* (disease observed in 1890 in the USA). In 1993 *X. fastidiosa* was identified as the cause of citrus variegated chlorosis (CVC) or citrus X disease in Brazil. The bacterium also causes a number of so-called leaf scorch diseases to *Prunus* spp. (including almond leaf scorch or ALS in *P. amygdalus* and plum leaf scald or PLS in *P. domestica*), *Acer* spp., *Carya illinoinsensis* (pecan), *Coffea arabica* (CLC, in Brazil isolated in 1995 and also pathogenic to citrus), *Hedera helix*, *Morus rubra*, *Nerium oleander* (OLS), *Platanus occidentalis* (Fig. 2A), *Quercus* spp. and *Ulmus americana*. *X. fastidiosa* is also the agent of alfalfa dwarf and of wilting of *Vinca major*. Many wild plants such as grasses, sedges and trees may carry the pathogen, often without showing symptoms. None of these diseases is apparently seed-borne. Seed transmission, however, has been reported in sweet orange, *C. sinensis* (Li *et al.*, 2003). They occur mainly in tropical/subtropical areas, although leaf scorch diseases are present also in much colder climate, e.g. oak leaf scorch in eastern North America up to Canada.

Several pathogenic varieties of the bacterium have been described, that are often host-specific (e.g., the PD strain will not cause disease if introduced to peach or plum). The following subspecies have been described:

(i) *Xylella fastidiosa* subsp. *fastidiosa* (erroneously named *X. f. subsp. piercei*), PD and LSA, strains from cultivated grape, alfalfa, almond (two), and maple; (ii) *X. fastidiosa* subsp. *multiplex*, PPD and PLS, strains from peach, elm, plum, pigeon grape, sycamore, almond and recently pecan (Melanson *et al.*, 2012); (iii) *X. fastidiosa* subsp. *pauca*, CVC, strains from citrus and probably those from coffee (CLC); (iv) *X. fastidiosa* subsp. *sandy*, strains from *Nerium oleander* (OLS); (v) *X. fastidiosa* subsp. *tasbke*, strains from the ornamental tree *Chitalpa tasbentensis*.

*X. fastidiosa* isolates are genetically very similar, but studies on their biological traits have indicated differences in virulence and symptomatology. As mentioned, taxonomic analyses have identified several subspecies, and phylogenetic analyses of housekeeping genes have shown broad host-based genetic differences. However, results are still inconclusive for genetic differentiation of isolates within subspecies. In a recent study, sequences of nine non-housekeeping genes were used to study 54 *X. fastidiosa* isolates infecting different host plants. Strains could again be divided into the known *X. fastidiosa* subspecies, but also showed new within-subspecies differentiation, including geographic differentiation, and some host-based isolate variation and specificity (Parker *et al.*, 2012).

*Xylella fastidiosa* is a quarantine organism on the EPPO A1 list and EC Annex II/1a list. For further and extensive data on biology, hosts, geographical distribution and epidemiology see recent reviews (Purcell, 1997; Chatterjee *et al.*, 2008; Janse, 2010; Janse and Obradovic, 2010) and the extensive *X. fastidiosa* website, edited and maintained by A. Purcell and R. Almeida: http://www.cnr.berkeley.edu/xylella/. For diagnostic methods, see EPPO 2005a and Janse *et al.* (2012).

**Hosts.** A full host list can be found on http://
www.cnr.berkeley.edu/xylella/. Some hosts relevant for the Mediterranean basin are: Nerium oleander, Platanus occidentalis (sycamore), Quercus spp. (oak), Ulmus americana (elm tree), Ambrosia artemisiifolia ( Ragweed), Morus alba (white mulberry), Prunus angustifolia (Chickasaw plum) and Rubus spp. In Taiwan a pear leaf scorch was described in 1990 on Pyrus pyrifolia (Japanese pear), cv. Hengshan and P. serotina (Asian pear), which was found to be caused by a bacterium very similar to _X. fastidiosa_, but differing from North and South American strains in serological properties and housekeeping gene sequences (Leu and Su, 1993; Chen et al., 2006). Hosts such as Asian pear were recently introduced in central Europe (Romania) and Japanese pears, or nashi, were planted as a novelty crop in southern Europe especially in the 1980’s. It is not known whether the latter two species have ever been subjected to surveys for _X. fastidiosa_.

**Symptoms and transmission.** For symptoms on different hosts, see http://www.cnr.berkeley.edu/xylella/. In general, early symptoms are a slight chlorosis or bronzing along leaf margin or tip that intensifies and that may become water-soaked before browning and drying. These symptoms are first found on a few branches, then they extend to most of the whole canopy (so-called leaf scorch or scald symptoms). A narrow chlorotic band that becomes especially clear in autumn delineates the affected area. A premature defoliation may take place with new malformed leaves are formed. Fruits can be abnormally shaped and stems may show internal and external discolorations, dieback and abnormal growth, leading to eventual death of the host. Vectors are mainly sharpshooters and froghoppers or spittlebugs (Cicadellidae) that lack a latent period, and have no transovarial transmission of the bacterium. The pathogen shows persistence in the vector adults, and may transmit the pathogen to the hosts through feeding. In North America main vectors (for PD unless indicated are _Cuerna costalis_ (PPD), _Draculacephala minerva_ (green sharpshooter) important also for ALS transmission in California; _Graphocephala atropunctata_ (blue-green sharpshooter), the most important before the arrival of _Homalodisca vitripennis_ (formerly _H. coagulata_) the glassy-winged sharpshooter, _G. versuta_ (PPD); _Hordnia circellata_, very efficient; _H. insolita_ (PPD), _Oncometopia nigricans_, _O. orbis_ (PPD), _Xyphon_ (formerly _Carmenacephala_) _fulgida_ (red-headed sharpshooter). CVC vectors in Brazil are _Acrogonia terminalis_ that lays eggs externally on the leaves, _Dilobopterus costalimai_ and _Oncometopia fascialis_. Local possible vectors for Europe are Cicadella viridis and _Philaenus spumarius_ (meadow spittlebug) (Fig. 2B). Because of the presence of these potential vectors the establishment of the pathogen in Europe is not strictly connected to introduction of foreign recognized vectors.

**Risks.** _X. fastidiosa_ is an emerging threat in the south-west USA, mainly due to recent establishment of the glassy-winged sharpshooter (GWSS, _H. vitripennis_), providing much more efficient transmission than local vectors, and leading to very serious outbreaks of PD in grapevine, ALS and OLS. GWSS probably first entered California as eggs deposited in plant tissues. In Central and South America _X. fastidiosa_ has become very noxious due to the rapid expansion (most likely via distribution of infected planting material) of CVC in _Citrus_, leading to more than a third of all trees in the area having symptoms of CVC, and _CLC_ in coffee. As to Europe, there are only a few unconfirmed reports of the presence of _X. fastidiosa_ in the grapevine in Kosovo (Berisha et al., 1998) and in almond in Turkey (Güldür et al., 2005). Since _X. fastidiosa_ has more than 150 hosts and many of them, including _Vitis_, are and are imported (often as planting material), risk of introduction (especially in latent form) must not be underestimated. Absence of the diseases caused by _X. fastidiosa_ will mainly be due to the absence of suitable vectors. However, introduction of the pathogen and infected vectors with plant material cannot be excluded. Moreover, also local Cicadellidae (see above) could become potential vectors. Therefore, _X. fastidiosa_ has the A1 quarantine status in the EPPO region and _H. vitripennis_ that has a very large host range and feeds on almond, peach and plum was recently placed on the EPPO alert list. As in the more southern parts of the USA, European _Vitis_ varieties are very susceptible to _X. fastidiosa_. This is really a risk if a vector that could survive the winters of southern Europe were introduced, the pathogen would become established in wild hosts (wild and domestic plums and wild cherry are symptomless reservoirs in the USA) and cause spring infections that are most likely to persist the months. The same risk holds true for _Citrus_ (sweet oranges, mandarins, and tangerines) and other hosts, such as almond, plum and peach that are widely grown in southern Europe, especially in the warmer Mediterranean basin, where a disease-favourable combination of warm nights, regular rainfall/high humidity and long growing season, is present. The conclusion is that _X. fastidiosa_ is a real threat for Europe, not only for _Vitis_ and _Citrus_ but also for stone fruits (almond, peach and plum) and oleander (GWSS likes to feed on oleander), that is difficult to prevent from entering and difficult to control once established, deserving more attention than up till now. Resistance in European grapes is scarce or even absent. Vector control proved not to be very effective in the USA. Cultural practices to keep plants in optimum condition are of importance, but not sufficient, and the use of avirulent strains for cross-protection is still in its infancy.

**Citrus canker, caused by Xanthomonas citri pv. citri (X. axonopodis pv. citri).** _General._ Asian citrus canker, a spot disease of _Citrus_ spp. characterized by corky le-
Geographical distribution. Xcc originates from and is widespread in Asia, including Georgia, Iran, Iraq, Oman, Saudi Arabia, UAE and Yemen. Australia (eradicated), Argentina, Belau, Brazil, Caroline Islands, Cocos Islands, Comoros, Congo Democratic Republic, Ivory Coast, Fiji, Gabon, Madagascar, Mauritius, Mozambique (eradicated), Netherlands Antilles, New Zealand (eradicated), Micronesia, Palau, Papua New Guinea, Paraguay, Reunion, Seychelles, South Africa (eradicated), Uruguay, USA (CABI/EPPO, 2006). There are recent reports from Somalia (Balestra et al., 2008) and Mali (Traoré et al., 2009) and Ethiopia (A* strains, Derso et al., 2009).

Hosts. Cultivated hosts are Aegle marmelos (golden apple), Casimiroa edulis (white sapote), Citrus aurantifolia (lime), C. aurantium (sour orange), C. blyttii (mauritius bitter orange), C. junos (yuzu), C. limetta (sweet lemon tree), C. limon (lemon), C. madurensis (calamondin), C. maxima (pummelo), C. medica (citron), C. natsudaidai (natsudaidai), C. reshni (Cleopatra mandarin), C. reticulata (mandarin), C. reticulata x Poncirus trifoliata (citrumelo), C. sinensis (navel orange), C. sunki (sour mandarin), C. tankan (tankan mandarin), Citrus unshiu (satssuma), Citrus x paradisi (grapefruit), Eremocitrus glauca (Australian desert lime), Limonia acidissima (elephant apple), Poncirus trifoliata (trifoliata orange or Japanese bitter orange). Minor hosts are Fortunella japonica (round kumquat) and F. margarita (oval kumquat). Wild hosts are Ageratum conyzoides (billy goat weed), Severinia buxi folia (box orange or boxthorn) and Swinglea glutinosa (= Aegle decandra, Limonia glutinosa).

Symptoms and transmission. For pictures of symptoms and of the citrus leaf miner, P. citrella, see http://www.plantmanagementnetwork.org/pub/php/review/citruscanker/). Small spots, first visible on the upper leaf blade, appear on the leaves, shoots, twigs and fruits, to become raised pustules or blister-like eruptions (Fig. 3A). With time, the lesions increase size (up to 10 mm) and turn brown and necrotic with a depressed centre, and are sometimes surrounded by a yellow halo. On the fruits, the lesions can be mistaken for scale insects (e.g. the California red scale, Aonidiella auranti). The bacterium is a wound parasite and, as mentioned, the citrus leaf miner (P. citrella) contributes to disease spread and severity. Citrus canker is especially epidemic and damaging on seedlings and young trees, especially after storms (hurricanes) under warm weather conditions, but because of dependence on these weather conditions epidemics are sporadic. Full-grown trees show much less disease and damage (Goto, 1992). The bacterium can survive in a latent form in and on diseased shoots and discoloured bark tissue of the trunk, and may reoccur suddenly after several years (sometimes even as long as 10 years).

Risks and damage. Heavy losses were reported in epidemics, due to premature fruit drop and fruits with
spots that cannot be marketed or start rotting, thus must be destroyed. Furthermore, quarantine measures such as burning of trees and destruction of fruits may add to these losses (Goto, 1992). In severe cases, almost 100% of the fruits and leaves of young, susceptible trees may be infected and the plant growth is delayed for a number of years. The direct Government costs in the USA for the eradication activities from 1995 to 2006 were calculated to be more than $1.3 billion. From 2006 to 2009, together with costs for the control of Citrus huanglongbing the amount was $90 million. The citrus acreage in Florida decreased since 1996 by ca. 33% (Lowe, 2010). In January 2006, the USDA determined that canker had become so widespread in Florida that eradication was no longer feasible. Use of healthy planting material and use of other measures, including weather forecasting, in an integrated way have been applied in the control of Citrus canker with some success.

Resistance has been found especially in *C. mitis* (calamondin) and *Fortunella* (kumquat). *C. reticulata* (mandarin) is tolerant.

Fig. 3. A. Wart-like excrescences induced by *Xanthomonas citri* pv. *citri* on sweet orange (*Citrus sinensis*). B-E. Symptoms of *Xanthomonas citri* subsp. *mangiferaeindicae* infection on mango (*Mangifera indica*). Necrotic leaf spots (B). Twig canker (C). Fruit spots with gummy exudates (D). Close up of the spots (E) [Gagnevin and Opruvost (2001). Courtesy O. Pruvost]. F. Necrotic spots on pomegranate (*Punica granatum*), caused by *Xanthomonas axonopodis* pv. *punicae* (source Dr. R. Kumar, http://www.nhm.nic.in/Vasanta_Pome.ppt#294,22,Slide 223). G. Necrotic lesions along the main vein of leaves of guava (*Psidium guajava*), caused by *Erwinia psidii* (Source: Prof. M.F.S. Papa, Sao Paulo University Brazil and APSnet Image Resources). H. Watersoaked leaf spots on passion fruit (*Passiflora* spec.), caused by *Xanthomonas campestris* pv. *passiflorae* (Courtesy: Dr. S.O.M. El Tassa, Departamento de Fitosanidade, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil).
to Europe and Xcc has been intercepted on this material (author's personal experience). However, the risk of dispersal by infected fruit was evaluated to be very small or absent if fruits are disinfected before shipment (Gottwald et al., 2009). Without disinfection, survival chances in symptomatic fruits are apparently also small (Shiotani et al., 2009), but not impossible, as shown by the identification of Xcc on imported fruits by the Dutch Plant Protection Service (author's personal experience). Since Xcc is endemic and spreading in countries surrounding the Mediterranean basin, some of which have also huanglongbing problems, and the citrus leaf miner is widespread in the area, the conclusion is that this pathogen is a real and emerging threat.

BACTERIAL DISEASES WITH AN EMERGING CHARACTER, BUT NOT YET PRESENT IN EUROPE OR THE MEDITERRANEAN BASIN

Black spot of mango - *Xanthomonas citri* pv. *mangiferaeindicae*. A leaf spot and canker disease of mango (*Mangifera indica*) called bacterial black spot, was described in South Africa by Doedoe (1915) who named the causal agent *Bacillus mangiferae* [later also named *Xanthomonas mangiferaeindicae*, and *X. campestris* (*axonopodis*) pv. *mangiferaeindicae*]. Ah-You et al. (2007, 2009) showed that this bacterium is much related to a pathogen from cashew (*Anacardium occidentale*) and named the two bacteria *X. citri* subsp. *mangiferaeindicae* and *X. citri* pv. *anacardii*, respectively. Both mango and cashew belong to the family Anacardiaceae. Symptoms of black spot start as small water-soaked spots that become later raised and necrotic, sometimes surrounded by a narrow yellow halo. On the fruits the water-soaked spots become star-shaped and crack and often show exuding gum. Severe infection under influence of rainstorms may lead to premature leaf and fruit drop, twig cankers and twig death (Fig. 3 B-E). Other (rare) hosts are amarella (*Spondias dulcis*, syn. *S. cytherea*) and Brazilian pepper (*Schinus terebinthifolius*), both belonging to the Anacardiaceae. When other diseases and pests are controlled, black spot is a limiting disease to mango production, because it is very difficult to contain. In most susceptible cultivars up to 100% fruit loss may occur. Many commercial cultivars are very susceptible. In 1996 and 1997, severe black spot epidemics were observed in many mango-growing areas of South Africa, causing almost 100% fruit loss on the most susceptible cultivars and ca. $1 million economic loss. There is production of mango in Europe and the Mediterranean basin (e.g. Spain, Italy, Israel and Portugal). Black spot occurs in Australia, Comoro Islands, many areas in southern and eastern Africa and Asia, Mauritius, New Caledonia, Reunion, Taiwan, and the United Arab Emirates (Gagnevin and Pruvost, 2001). Discrimination of strains from mango and some related hosts and from different geographic origin (Asia, Africa and Brazil) was possible using RFLP (Gagnevin et al., 1997). Long distance dissemination of the pathogen is thought to be by infected planting material. Epiphytic/endophytic populations of the pathogen occur (Pruvost et al., 2009). Seed transmission has not been demonstrated.

Bacterial blight of pomegranate - *Xanthomonas axonopodis* pv. *punicae*. A bacterial disease on pomegranate (*Punica granatum*, family Lythraceae) was observed for the first time in 1952 in New Delhi, India and described by Hingorani and Sing (1959) as being induced by *Xanthomonas punicae* (later classified as *X. campestris* pv. *punicae* and *X. axonopodis* pv. *punicae*). First symptoms are water-soaked spots on leaves and fruits (Fig. 3F). On the leaves, spots become necrotic and, when they coalesce, severe leaf drop may occur. Fruits may crack and drop. Black necrotic spots occur on branches, that become weak and may split. Pomegranate is produced mainly by India (50%), Iran (35%), some of the former Soviet Union states and in the Mediterranean basin, especially in Spain (2.5%), Morocco, Egypt and Turkey. Bacterial blight has developed into a very serious disease in India, where it causes very heavy losses (up to 100%) in many pomegranate-growing areas. (Kumar et al., 2006). Dispersal at a site is by rain splash, insects and tools, whereas over long distances is by infected plant material. Like *X. citri* pv. *citri* and pv. *mangiferaeindicae*, stormy weather conditions are important for epidemic outbreaks. *X.a.* pv. *punicae* overwinters in infected leaves of neem (*Azadirachta indica*, family Meliaceae) planted along pomegranate fields (Dhasandar et al., 2004; Sharma et al., 2008; Kumar et al., 2009) since these former two pathogens have already spread from the more eastern parts of Asia, *X. a. pv. punicae* can be seen also as a potential threat that may soon show its presence closer to the Mediterranean basin.

Bacterial blight of guava - *Erwinia psidii*. A vascular disease of guava (*Psidium guajava*) was reported as bacterial blight from Brazil and the causal agent denoted *Erwinia psidii* (Neto et al., 1987), was held responsible for the outbreaks in the main production areas of southeastern and central regions of the country (Tokeshi et al., 1980). Symptoms are local and systemic. Leaves show large necrotic lesions along the main veins (Fig. 3G) and at the margins (scorching), or small water-soaked spots, sometimes with a chlorotic halo, that later become necrotic and coalesce causing leaf drop. When bacteria reach the xylem they spread into branches, trunk and roots. In severe cases, trees are defoliated and die. Symptoms on the fruits are not common. This disease could be of importance to guava-growing countries such as Egypt that produces yearly some 230,000 tons of guava fruits, being the fifth producer in the world after India, Pak-
istan, Brazil and Mexico. In Brazil, pathogen dispersal often occurs with contaminated planting material (Marques et al., 2007; Teixeira et al., 2008b).

**Bacterial spot of passion fruit - Xanthomonas campestris pv. passiflorae.** A destructive disease of cultivated passion fruit (Passiflora edulis and P. edulis var. flavicarpa), was described by Pereira in 1969 in Brazil (El Tassa, 2002). This disease is characterized by the presence of water-soaked, greasy lesions of irregular shape on the leaves, surrounded by chlorotic areas that result in extensive necrosis when they coalesce (Fig. 3H). Fruits display greasy spots that make them unsuitable for consumption and industrial processing. This disease has an emerging character in Brazil, where ca. 25,000 ha are given over to passion fruit and has become a major problem to production. Hosts are *Passiflora alata, P. amethystina, P. coccinea, P. edulis, P. edulis var. flavicarpa, P. maliformis, P. nitida* and *P. serrato-digitata* (Neto et al., 1984; Torres Filho and Ponte, 1994; Gonçalves and Rosato, 2000; Lopes et al., 2006).

**BACTERIAL DISEASES/PATHOGENS WITH AN EMERGING CHARACTER, ALREADY OCCURRING IN EUROPE OR THE MEDITERRANEAN BASIN**

**Almond witches’ broom - Candidatus Phytoplasma phoenicium.** This devastating disease of almond (*Prunus amygdalus*), shows typical witches’ broom symptoms, i.e. small yellow leaves on proliferating shoots that wither and die in later stages (Fig. 4A-C). It

![Fig. 4. Symptoms of witches’ broom in almond. on flowering twigs (A), mature branches (B) and tree (C) caused by “Candidatus Phytoplasma phoenicium” (courtesy E. Choueiri). D. Malformations of tomato (*Lycopersicon esculentum*), caused by potato stolbur, “Candidatus phytoplasma solani” (Source EPPO).](image-url)
was reported from Lebanon by Choueiri et al. (2001), and was classified and named by Verdin et al. (2003). *Candidatus* Phytoplasma phoenicium belongs to the pigeon pea witches’ broom group (16SrIX). In a few years time, more than 100,000 trees were killed in different areas in Lebanon. This phytoplasma was also found on peach and nectarine (Abou-Jawdah et al., 2009) and was recently also reported from Iran on almond (Zirak et al., 2009) and on GF-677 (*Prunus amygdalus* x *Prunus persica*) (Salehi et al., 2011). Vectors are suspected to be leafhopper which have not yet been identified (Abou-Jawdah et al., 2011). This pathogen is not included in quarantine lists, but certainly deserves attention as an emerging threat for almond. Stem cutting culture with thermotherapy was successfully used for regeneration of phytoplasma-free plantlets of almond (Chalak et al., 2005). Recently Davis et al. (2010) have reported a phytoplasma closely related to Ca. P. phoenicium as the cause of witches’ broom affecting *Juniperus occidentalis* (western juniper) in Oregon (USA).

**Potato stolbur - *Candidatus* Phytoplasma solani.**

*Ca.* Phytoplasma solani is a non-culturable, insect-transmitted bacterium that induces bushy growth and malformations in solanaceous (Fig. 4D) and other hosts (EPPO/CABI 1996) and is on the EPPO A2 quarantine list. It belongs to the so-called Aster yellows or stolbur (16Sr-XII-A) phytoplasma group, the latter having a very wide host range (‘Bois noir’ of grapevine is caused by a phytoplasma of the same group). Severe outbreaks occur under dry weather conditions when vector populations develop explosively on wild hosts. In some years, substantial damage was reported from south-east Europe and Russia. Spreading in potato is slow. Vectors are mainly leafhoppers of the family Cicadellidae, the most important of which are the polyfagous *Hyalesthes obsoletus*, the true bug *Lygus pratensis* (family Miridae) and some other cicads, such as *Macrostelea quadrripunctatus*. Potato stolbur has been reported from Austria, Bulgaria, Czech Republic, Germany, France, Greece, Hungary, Italy (Berger et al., 2009), Israel, Poland, Romania, Russia, Serbia, Switzerland, Turkey and Ukraine. Main hosts for the bacterium are potato, tomato, eggplant, pepper and weeds such as black nightshade, bindweeds (*Convolvulus arvensis*, *Calystegia sepium*), stinging nettle, *Cardaria* or *Lepidium* and *Lavandula*. Larvae of cicadellids feed on the roots from which they can acquire and transmit the bacterium. Potato stolbur might spread more, when the climate warms up. *H. obsoletus* recently spread in Germany from the Moselle valley and by the end of 2009 stolbur phytoplasma was detected in association with bindweed in Rheinland-Pfalz (EPPO Reporting Service 2010/155). In eastern Europe (Bulgaria, Romania, Serbia), *Ca.* phytoplasma solani has been causing for many years a disease transmitted by *Reptalus panzeri* (Jovic et al., 2009) called ‘corn redening’, responsible for 10-90% crop losses. Diseased plants show a red discoloration of stems and main leaf vein and abnormal ears. In Romania the cultivation of potato cv. Lady Rosetta was stopped in disease-sensitive areas, severe losses occurred from 2006-2008, in 2008 circa 75 ha were infected, of which 45 ha suffered total crop loss. Also from Russia, the region Severe damages were reported in 2003 from Russia in an area around Krasnodar (www.costphytoplasma.eu) as well as in the Czech Republic (Navrátil et al., 2009). *Ca.* P. solani does not persist much in potatoes during storage, so that only few diseased plants develop from infected tubers. Late infections do not influence the yield. The disease should not be a problem in a well-tended crop, were healthy, certified planting material is used and where a careful weed and vector control takes place.

**Zebra chip disease of potato or psyllid yellows of solanaceae and carrot - *Candidatus* Liberibacter solanacearum (synonym *Ca.* Liberibacter psyllolourus).**

This non-culturable bacterium is closely related to earlier mentioned Liberibacter species occurring in *Citrus*, and has been placed on the EPPO alert list (http://www.eppo.org/QUARANTINE/Alert_List/bacteria/Liberibacter_psyllolourus.htm). *Ca.* Liberibacter solanacearum (*CaLS*) was first described in New Zealand in 2008 (Lief tink et al., 2011) as *Ca.* L. psyllolourus, and was later found also the USA (Hansen et al., 2008) where it has an emerging character. In the USA it has been reported from Texas in 2010 (French-Monar et al., 2010) and in 2011 from Idaho, Washington state and Oregon (Crosslin et al., 2012). It also occurs in Guatemala, Honduras, Canada and, in tomato, in Mexico (Munyaneza et al., 2010c). The tomato/potato psyllid *Bactericera cockerelli* is a vector that feeds mainly on solanaceous hosts, but it occurs on many other hosts and causes so-called psyllid yellows. It is present in North and Central America and, recently (2000), it appeared in New Zealand. The bacterium can be spread with potato seed, tomato plantlets and fruits. Seed transmission has not been reported. Apparently, *CaLS* is quite versatile and adapted to diverse climatic regions such as desert, steppe, Mediterranean, marine coast, humid continental and humid subtropical. Its hosts are *Capsicum annuum*, *C. frutescens*, *Lycopersicon esculentum*, *Physalis peruviana*, *Solanum betaceum* and *S. tuberosum* (Fig. 5A). The disease is called Zebra chip because characteristic brown stripes develop when potato tubers are cooked (Fig. 5B). The complete genome sequence of *CaLS* has been determined (Lin et al., 2011). More recent records of *CaLS* in carrot (*Daucus carota*) are from: (i) Finland where the bacterium is transmitted by the psyllid *Trioza api culis* and causes, together with its vector, substantial crop losses. Infected plants show typical leaf curling, yellow and purple discoloration of the leaves, stunting of roots and shoots, and proliferation of secondary roots (Munyaneza et al., 2010a, 2010b); (ii)
Canary Islands (Tenerife, since 2009) where the bacterium is apparently transmitted by the psyllid *Bactericera trigonica* (Alfaro-Fernández et al., 2012a). Subsequently it has also been reported from mainland Spain, where it infects celery (*Apium graveolens*) in several regions, also in mixed infections with *Ca. P. solani* and, strangely enough, with the bacterium *Spiroplasma citri* (Alfaro-Fernández et al., 2012b); (iii) Norway (since 2011) where *CaLS* causes extensive damage to commercial fields with an incidence of 10-100% (Munyaneza et al., 2012a); (iv) Sweden, since 2011 (Munyaneza et al., 2012b). Until now, *CaLS* has not been reported from potato, probably because the vector *B. cockerelli* does not occur in Europe.

Other phytoplasmas that could become a threat will not be treated in this review, apart from their mention in Table 1. For a recent overview, see COST Action FA0807 “Integrated Management of Phytoplasma Epidemics in Different Crop Systems.”

**Harmless (?) “Candidatus Liberibacter europaeus” found in *Cacopsylla pyri*.** An apparent harmless Liberibacter species, named “*Candidatus L. europaeus*” was reported from Italy to occur in a high percentage of the psyllid *Cacopsylla pyri* that thrives on pear trees (*Pyrus communis*). The bacterium could be experimentally transmitted by the psyllid to pear but, apparently, ut remains until now non-pathogenic (Raddadi et al., 2011).

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**Table 1. Emerging phytoplasmas reported at the Meeting on “Emerging phytoplasma diseases of stone fruits and other crops and their possible impact on EU countries”, held in 2011 in Istanbul, Turkey.**

<table>
<thead>
<tr>
<th>Name</th>
<th>16S rRNA Group</th>
<th>Latin name</th>
<th>Country where reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almond witches’ broom</td>
<td>16S rIX-B</td>
<td>“Ca. P. phoenicium”</td>
<td>Lebanon</td>
</tr>
<tr>
<td>Cassava frog skin</td>
<td>16S rIII-L</td>
<td>“Ca. P. asteris”</td>
<td>Colombia</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16S rI-B</td>
<td>“Ca. P. fraxini”</td>
<td>Italy, South Africa</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16S rVII-A</td>
<td>“Ca. P. ziziphi”</td>
<td>Chile</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16S rXII</td>
<td>“Ca. P. aurantifolia”</td>
<td>China, Korea, Italy</td>
</tr>
<tr>
<td>Jujube witches’ broom</td>
<td>16S rV-B</td>
<td>“Ca. P. trifolii”</td>
<td>USA</td>
</tr>
<tr>
<td>Lime witches’ broom</td>
<td>16S rII-B</td>
<td>“Ca. P. americanum”</td>
<td>USA</td>
</tr>
</tbody>
</table>

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**Bacterial fruit blotch of Cucurbitaceae - *Acidovorax citrulli* (syn. *A. avenae* subsp. *citrulli*).** Symptoms of bacterial fruit blotch are water-soaked leaf spots on seedlings that may coalesce in later stages and kill the plantlets. Mature plants show reddish brown streaks along main veins and, most characteristically, dark green spots on the fruit surface (watermelon, Fig. 5C) or inside the fruit (melon and pumpkin) which are accompanied by rind depressions and cracks from which bacteria may ooze. Hosts are watermelon (*Citrullus lanatus*) and melon (*Cucumis melo*), but also *Cucumis sativus* (cucumber), *Cucurbita pepo* (squash), and *C. moschata*, which show symptoms only on the leaves, and wild cucurbits, e.g. *Citrullus lanatus* var. *citroides*. Deng et al. (2010) reported *A. citrulli* outbreaks on *Piper betle* (betelvine, Piperaceae) in Taiwan. Strain diversity, i.e. mild strains on hosts other than watermelon and more aggressive strains from watermelon, has been observed in the USA (Walcott et al., 2004). Recent outbreaks of bacterial fruit blotch were reported in Europe from Greece (2005), Hungary (2007, apparently with watermelon transplants from Turkey), Israel (2000 and 2003, melon and watermelon), Turkey (1995, Marmara region and 2005 Mediterranean region) and Italy (2009, melon) [see also: http://www.eppo.org/QUARANTINE/Alert_List/bacteria/Acidovorax_citrulli.htm; Holeva (2009); Hopkins et al. (2001); Hopkins and Thomson (2002)]. The bacterium is mainly spread with seed and
planting material and the disease it elicits may result in extensive damage and losses (40-100% in USA and Brazil). Control measures to be implemented are: (i) use of healthy tested seed (seed treatment has not been effective up to now); (ii) seed test by a check on 10,000 seedlings/lot in a greenhouse (so-called sweatbox method); (iii) inspection of plants during the growing season and destruction of infected plant material. In recent years also PCR screening tests have been developed (Bahar et al., 2008; Jing et al., 2011; Woudt et al., 2012). Positive results of all these methods still need to be verified by isolation of the pathogen. Perhaps RT-PCR in combination with malditof (matrix assisted laser desorption ionization time-of-flight mass spectrometry) could be used to exclude false positives (Wang et al., 2012).

A new strain of a potato stem rot bacterium belonging to the genus Dickeya, provisionally named ‘D. solani’. Bacterial stem rot of potato, caused by different ‘cold tolerant’ biovars of Erwinia chrysanthemi, now named Dickeya dianthicola and D. chrysanthemi, has been reported from many European countries since the 1970s, and is regarded as a quality disease (Janse and Ruissen, 1988; Toth et al., 2011). A taxonomic revision placed Erwinia chrysanthemi biovars into six species of the newly created genus Dickeya, namely D. chrysanthemi biovar chrysanthemi and biovar parthenii, D. dadanti, D. dianthicola, D. dieffenbachiae, D. paradisiaca and D. zeae (Samson et al., 2005). Among them there are ‘warm tolerant’ species like D. dadanti and D. zeae that were found not only in potato in hot climates, but also in many ornamental plants, often grown in greenhouse in temperate regions (Janse and Ruissen, 1988; Janse and Scheepens, 1990; Samson et al., 1990). Recently a new ‘warm tolerant’ variant of Dickeya was observed, first in the Netherlands since 2000 (Czajkowski, 2009a, 2009b, 2011; J. Van Vaerenbergh, personal communication), then in Poland, Belgium, Finland, France, Israel and most recently also in the UK (2010), Denmark and Sweden (2011). This variant is virulent under warm climatic conditions and is closely related, but not similar to D. dadanti, it has been provisionally denoted D. solani (Czajkowski, 2011). A recent taxonomic study indicates that it is not certain whether this variant is indeed a new species (Van Vaerenbergh et al., 2012). Warm tolerant Dickeya spp. have been isolated from surface waters and D. solani, that shows a strong clonal character, in the Netherlands also from hyacinth and iris in the bulb production area where no potatoes are grown (Toth et al., 2011; Van Vaerenbergh et al., 2012). A theory is that a warm tolerant species (D. dadanti or zeae) escaped from greenhouse wastewater disposals to surface water and mutated to a form that clonally spread in hyacinth, iris and potato. As with the other Dickeya and Pectobacterium species in potato, effective control is possible and regulated via certification schemes. It should be based on the use of tested, healthy seed and careful cultural practices (careful and dry harvesting, proper storage and ventilation, avoiding of desprouting and cutting seed, hygiene on the farm, etc).

### Stem rot and leaf spot of maize and ‘center rot’ of onion - Pantoea ananatis

Pantoea ananatis can cause a number of diseases on different hosts, e.g. stem rot and leaf spot of maize, internal fruit rot of melon and pink disease of pineapple. It also infects onion (Allium cepa) inducing ‘center rot’, outbreaks of which have been reported since 1997 in the USA, and was isolated from onion seed in South Africa (Goszczynska, 2006). Seed transmission was confirmed in the USA (Walcott et al., 2002). The thrips Franklinella fusca can transmit the bacterium which apparently survives as a saprophyte on many weed and crop hosts (Gitaitis et al., 2003).

### Stewart’s disease or bacterial wilt - Pantoea stewartii subsp. stewartii

This vascular disease, characterized by white stripes on the host leaves, is widespread in North America. The main host is maize, especially sugar maize, but also the so-called ‘dent’, ‘flint’, ‘flour’ and popcorn types. The bacterium is mainly spread by Chaetocnema pulicaria, the corn flea beetle. P. subsp. stewartii and bacterial wilt have been reported from, but are not established in Austria, Greece, Italy, Poland, Romania and European Russia. Main source of introduction is contaminated seed from North America, but pathogen and disease disappear apparently some years after introduction, due to absence of the vector beetle in Europe. Local vectors do not transmit the bacterium as far as is known. In Italy, substantial damage was reported in the years 1940-1950, and some reoccurrence, but not very damaging in 1983-1984 (Mazzucchi, 1984; www.eppo.org/QUARANTINE/bacteria/Pantoea_stewartii/ERWIST_ds.pdf). Further introductions could occur in the future, but will remain relatively harmless as long as the vector is not introduced or local flea beetles, as potential vectors, do not acquire and transmit the pathogen.

### New outbreaks of bacterial (blight) canker of kiwifruit - Pseudomonas syringae pv. actinidiae

Bacterial canker of kiwifruit was first observed in Japan in 1984 (Takikawa et al., 1989). It was also reported from China where, as we know now, it occurred already as early as 1984/1985 (Liang et al., 2000). Subsequently it was found in Korea and Italy (1992). Hosts are green kiwi (Actinidia deliciosa), yellow kiwi (Actinidia chinensis), A. arguta, and A. kolomikta. Since 2008 bacterial canker outbreaks have repeatedly been observed in Italy (Emilia-Romagna, Lazio, Piemonte and Veneto) on green kiwi, causing a mild leaf spot and some canker formation on branches and trunk. There was also a re-
port from Iran in 1994 and a recent occurrence of the so-called Asian or mild strain in Australia (EPPO Reporting Service 2011/130). In spring and autumn of 2008 and winter 2008/9, however, severe outbreaks occurred on yellow kiwi characterized by wilting, necrosis, severe leaf spot and canker formation (Fig. 6A-C), especially the cvs Hort 16A and Jin Tao cultivated in central Italy (Latina province). Recently, severe outbreaks have also occurred on green kiwi cv. Hayward (Ferrante and Scortichini 2010) and the disease has spread to Calabria, Campania, and Friuli-Venezia Giulia regions in 2011 (EPPO Reporting Service 2011/131). *P. s. pv. actinidiae* was recently observed in yellow and green kiwi in France [severe form found in the Rhône-Alp area in 2010 (EPPO Reporting Service 2012/002)], Spain (Abelleira et al., 2011; Balestra et al., 2011), Portugal (Balestra et al., 2010), Switzerland (2011), Chile (2011) (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm) and Turkey, where it is present since 2009 in the Black Sea area (Bastas and Karakaya, 2012). Interestingly, the severe form is also known to occur since 2010 in New Zealand and has spread on the North and South Island (Everett et al., 2011; Young, 2012). In this country another mild strain causing only leaf spots has also been observed, which may be not *P. s. pv. actinidiae* (Vanneste et al., 2010). The Asian mild strain was recently reported to be present in Australia (EPPO Reporting Service 2011/130). A PCR test for identification and epidemiological studies was developed by Vanneste et al. (2010). Epidemics of bacterial blight of kiwi occur usually after frost damage. In Japan and Korea bacterial blight is the most limiting

Fig. 6. A-C. Symptoms induced by an aggressive strain of *Psudomonas syringae pv. actinidiae* on yellow kiwifruit (*Actinidia chinensis*) in Italy. Wilting and necrosis (A), necrotic leaf spots (B), canker with a reddish ooze (C) (Courtesy M. Scortichini). D-F: Symptoms induced by *Psudomonas syringae pv. aesculi* on Horse chestnut (*Aesculus hippocastanum*). Severe wilting and yellowing (D), red ooze from a canker (E), extensive cankers on a branch (F) (Courtesy Aesculaap, NL). G: Leaf spots with a yellow halo and ‘shotholes’ on cherry laurel (*Prunus laurocerasus*) caused by *Xanthomonas arboricola pv. pruni* (Source Naktuinbouw, NL).
factor to kiwi crops, although mild strains appear to occur there. From Italy in recent years more than 2 million euro damage was reported. In a recent taxonomic study, with extensive sequence analysis of the diverse strains, Mazzaglia et al. (2012) came to the conclusion that severe strains from Europe and New Zealand indeed differ from the mild strains that occur in Japan, Korea and were also present in Italy in the past, and that are similar to strains from China, the country where the bacterium probably originates. They also showed that New Zealand and European strains differ to a small extent and both are more close to the Chinese strains, implicating a possible origin of these strains in China, probably imported with planting material from that country. Due to the emerging character of the new severe strains of \( P. \text{syringae pv. actinidiae} \), EPPO has placed it on its alert list (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm).

An emerging disease in forestry: Bleeding canker of horse chestnut - \( \text{Pseudomonas syringae pv. aesculi} \).
Since 2002 an emerging bacterial disease, named bleeding canker of horse chestnut (\( \text{Aesculus} \) spp., especially \( A. \text{hippocastanum} \)) was observed first in the Netherlands, then in Belgium, France, Germany and the UK. It is thought that the disease had longer been present in the countries concerned, with an incidence of 40-60%.

The bacterium shows a clonal character and adaptation to horse chestnut (Green et al., 2010, 2012). Symptoms are cracks with reddish-brown exudate on trunk and branches, and extensive necrotic cankers in phloem, where death of cambium leads to dieback (Fig. 6D-F). Insect transmission cannot be excluded and flower infection has been observed. Trees aged 10 to 30 years can be killed in a few years time (van Beuningen et al., 2009). It was determined that \( P. \text{pv. aesculi} \) does not survive exposure to 39°C for 24 h and initial experiments treating young trees for 48 h at this temperature were promising (Keijzer et al., 2012).

See also: Bultreys et al. (2008); Janse et al. (2006); Schmidt et al. (2008); Webber et al. (2008).

Spreading of bacterial canker and leaf spots of stone fruits - \( \text{Xanthomonas arboricola pv. pruni} \).
Recent outbreaks of bacterial canker induced by the quarantine pathogen \( \text{Xanthomonas arboricola pv. pruni} \) on peach, nectarine (\( P. \text{persica var. nectarina} \)), apricot, cherry and/or plum have been reported from Slovenia (1994), France (1995), Spain (1999) and Iran (2005). The disease is established in Austria, Bulgaria, Italy, Moldova, Montenegro, Russian Federation and Ukraine. On peach, small cankers on twigs and leaf spots are formed; on plum and apricot, holdover cankers on trunk and larger branches are the main problem. On cherry, fruit infection is most damaging (Roselló et al., 2012; Stefani, 2010). \( X. \text{a. pv. pruni} \) would be a risk for north-west Europe should climate change towards higher temperatures continue. The bacterium was identified from plum planting material originating from Asia in 1994 and spread to peach, with an outbreak in 1994 and further ones in later years (Seljak et al., 2001). In France, a severe outbreak took place in 2000 on peach and nectarine (EPPO reporting service 2006/235). \( X. \text{a. pv. pruni} \) was reported from Hungary in propagating material of plum (2004), then in an apricot orchard (Nemeth, 2007). In Italy severe outbreaks occurred in the 1990s on Japanese plum (\( P. \text{salicina} \)) and nectarine, after introduction of the very susceptible cv. Calita. A first report of \( X. \text{a. pv. pruni} \) infection to \( P. \text{laurocerasus} \) in a nursery in Toscany was also from Italy in 2005.
In Switzerland, the first record was in an apricot orchard in 2005 and in two Japanese plum orchards in 2009, near Martigny (Pothier et al., 2009). In the Netherlands, a first outbreak in \( P. \text{laurocerasus} \) occurred in 2008, in the west of the country, where plants showed shot-hole symptoms in the leaves (Fig. 6G) (EPPO Reporting Service 2009/178). Infections were again identified in different nurseries in 2009-2011 (Bergsma-Vlami et al., 2012). In Spain the bacterium was found on peach in 1999, further outbreaks in almond in 2006 and 2009 in Valencia and Aragon (Palacio-Bielsa et al., 2010, Roselló et al., 2012). Temperatures of 15-28°C, heavy rain and wind in springtime stimulate epidemics. The economic impact of \( X. \text{a. pv. pruni} \) consists of reduced quality and marketability of fruits and reduced productivity of the trees, as well as higher production costs.

Bacterial leaf spot of poinsettia - \( \text{Xanthomonas axonopodis pv. poinsettiiicola} \).
This bacterium was originally found in poinsettia (\( \text{Euphorbia pulcherrima} \)) in India (Patel et al., 1951), then reported from Florida (USA) (Chase, 1985). Other Euphorbiaceae are susceptible, such as \( E. \text{beterophylla} \) (wild poinsettia, mainly occurring in North America, but now widespread in Italy), \( E. \text{milii} \) (crown-of-thorns), \( \text{Codiaeum variegatum} \) (croton) and \( \text{Manihot esculenta} \) (cassava) (Chase, 1985; CABI, 2011). \( E. \text{pulcherrima} \) originates from Mexico and is a very popular Christmas pot plant in European countries, on which \( X. \text{a. pv. poinsettiiicola} \) causes brown to black leaf spots, sometimes surrounded by a yellow halo. Over time the spots may coalesce and the leaves turn completely yellow and drop, severely impairing the commercial value of the plants (Wohanka, 2004). The disease occurs in Cocos Islands (territory of Australia), the Philippines (Quimio, 1974), Taiwan (Lee et al.,
CONCLUDING REMARKS

A number of specific and general prevention and control measures for emerging diseases have been listed under Liberibacter spp. The following further measures should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

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comb. nov. and delineation of four novel species, *Dickeya davidii* sp. nov., *Dickeya daeicola* sp. nov., *Dickeya dieffenbachiae* sp. nov. and *Dickeya zeae* sp. nov. *International Journal of Systematic and Evolutionary Microbiology* **55**: 1415-1427.


Mediterranean basin and/or Europe. Introduction of susceptible wild hosts and susceptible cultivars of cultivated hosts must be avoided, as they often lead to introduction of pathogens or to outbreaks due to a ‘jump’ of local pathogen strains to very susceptible cultivars. Introduction of vectors should also be prevented as history teaches that vectors appear first and the pathogens a few years later. Examples of these events are given in the description of the various disease-pathogen combinations. An efficient prevention and control strategy of the diseases mentioned in this review should be based on the so-called pathway protection, i.e. regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-regulated pests, where the following conditions should be enforced and controlled: (i) place of production should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

Key words: quarantine, risk assessment, geographical distribution, host plants, plant pathogens.

INTRODUCTION

Bacterial diseases are often a destructive and/or a major constraint for many crops. Substantial killing of the host and yield losses, often over 50% result from infestations caused by established pathogens such as Erwinia amylovora, Pseudomonas syringae pv. syringae and pv. morpurorum, Agrobacterium tumefaciens (syn. Rhizobium tumefaciens) Xanthomonas arboricola pv. pruni, Candidatus Phytoplasma mali, Grapevine flavescence dorée phytoplasma, Grapevine bois noir phytoplasma, "Candidatus Phytoplasma pyri" and "Candidatus P. prunorum" (European stone fruit yellows). Quarantine pathogens sometimes spread out of contained loci, presenting a long-term threat to other EU countries (e.g. Xanthomonas arboricola pv. pruni spreading out of France and Italy) and others are an emerging threat outside Europe (Xylella fastidiosa, Candidatus Liberibacter spp. agents of citrus huanglongbing).

Bacterial diseases are difficult to control (both chemically and biologically) and are restrained primarily by preventive measures such as hygiene, use of healthy planting material, good cultural practices and avoidance of risky planting sites. Moreover, they are easily spread by (surface) water, planting material and contaminated agricultural implements/machines and by aspecific or specific insect vectors. Most important risk factors for the introduction of bacterial diseases into Europe are imported infected planting material and naturally spreading infected insect vectors. Therefore, early detection and correct identification/diagnosis are of utmost importance. In recent reviews, current classic and molecular methods for detection and identification of bacterial pathogens of fruit trees and nuts, including Xylella fastidiosa, have been described (Janse, 2010; Janse and Obradovic, 2010; Janse et al., 2012). In this contribution the epidemiology and main risks in the framework of prevention and management in case of first occurrence of some emerging bacterial diseases approaching the Mediterranean basin and Europe, their causal organisms and vectors, will be highlighted, with special reference to: (i) Citrus huanglongbing (HLB, formerly called Citrus greening), caused by the heat tolerant "Candidatus Liberibacter asiaticus" and the heat sensitive "Candidatus L. africanus". Both liberibacters and the respective psyllid vectors Diaphorina citri and Trioza erytreae are present in the Arabian peninsula, with recent reports of huanglongbing occurring in Iran, Mali, Ethiopia and Somalia and T. erytreae already present on some Atlantic Ocean islands (for references see under Geographical distribution of the specific pathogens later in the text). Furthermore, in less detail: (ii) leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by Xylella fastidiosa. For this pathogen, although its presence in Europe and the Mediterranean basin has not been confirmed, local possible vectors such as Cicaella viridis and Philaenus spumarius occur; (iii) Citrus canker, caused by Xanthomonas citri pv. citri (syn. X. axonopodis pv. citri, X. citri subsp. citri), the most severe form of which, the so-called Asiatic, is already present in Iraq, Iran, Oman, Somalia, UAE, Saudi Arabia, Yemen and Reunion (for references see under Geographical distribution of the specific pathogens later in the text).

Outbreaks and the possible emerging character of some other bacterial pathogens will be dealt with (e.g. Xanthomonas citri pv. mangiferaeindicae approaching the Mediterranean basin like X. c. pv. citri and the devastating "Candidatus Phytoplasma phoenicium" that occurs on almond in Lebanon). Furthermore, some diseases already present in the area which show an emerging character, such as the bacterial canker of kiwifruit (Actinidia spp.) caused by Pseudomonas syringae pv. actinidiae in Italy and France in the last three years, will also be addressed.

Since initial management and risk avoiding measures following an introduction are more or less the same for most of the above-mentioned pathogens, they will be detailed for HLB. It will be argued that the diseases addressed in this presentation are emerging threats, with real risks of introduction and economic damage and in some cases closely approaching the Mediterranean basin. The aim of this contribution is to create awareness of the risks of these diseases, thus enabling prevention, early detection and proper actions once introduction has occurred.
MAIN EMERGING BACTERIAL DISEASES

Citrus huanglongbing caused by “Candidatus Liberibacter” species. General. Huanglongbing or HLB (Chinese for yellow shoot disease) of citrus is caused by a non-culturable, fastidious, phloem-inhabiting, Gram-negative bacterium belonging to the α-Proteobacteria and to the genus “Candidatus Liberibacter” [originally the genus was named Liberobacter (Bové, 2006)]. This disease (Fig. 1A-C) was formerly known as citrus greening yellow dragon disease, mottle leaf disease, ‘likubin’ or vein phloem degeneration. It is one of the most destructive diseases of cultivated citrus for which no effective control is available with the consequence that citrus production declines in all areas where the disease occurs. Lin (1956) determined that this disease is graft-transmissible and described it for the first time in China. The origin of huanglongbing is not clear, probably Asia (India or China) or Africa (Gottwald et al., 2007). For a thorough general review on the disease and its causal organism see Bové (2006) and for a review on its epidemiology see Gottwald (2010).

Three different Liberibacter species causing Huanglongbing have been described based only on 16S rRNA and whole genome sequences information, because these organisms cannot be cultivated in vitro:

a. “Candidatus Liberibacter asiaticus” (Las), originally described as Liberobacter asiaticum, the most aggressive species, is heat-tolerant and stands temperatures above 30°C. The disease caused by this species is found mainly in lowlands where it is transmitted by the heat-tolerant psyllid Diaphorina citri Kuwayama (Fig. 1D,E) [a recent report mentions the black psyllid, Diaphorina communi s, as a vector in Bhutan (Donovan et al., 2011)]. Las is widespread in Asia, the Arabian peninsula, Mauritius and Reunion islands and, since 2004, in Brazil, Sao Paulo state [in Brazil some 10% of the infected plants are infected by this Liberibacter species, others are infected by the third species, Ca. L. americanus (Teixeira et al., 2005)]. It occurs since 2005 in Florida (USA) (Halbert, 2005).

b. “Candidatus L. africanus” (Laf), described as Liberobacter africanun, less aggressive and heat-sensitive. The disease induced by Laf is suppressed after exposure to temperatures above 30°C, therefore it occurs in the tropics at elevations higher than 700 metres above sea level. Laf is widespread in Africa (Somalia, Ethiopia and Cameroon) and occurs also in Reunion, Mauritius, and Yemen. Its vector is the heat-sensitive psyllid Trioza erytreae Del Guercio. c. “Candidatus L. americanus” (Lam), closely related to Las but heat-sensitive. It occurs in Brazil and is transmitted by the psyllid D. citri.

A recently described subspecies, “Candidatus Liberibacter africanus” subsp. capensis, was isolated from a symptomless ornamental rutaceous tree (Calodendrum capense) in the Western Cape province (South Africa). In the article describing this subspecies, the genus name Liberobacter was changed for all species into Liberibacter (Garnier et al., 2000). Ca. L. africanus subsp. capensis appears to be widespread in C. capense in South Africa, but is not transmitted to citrus (Phahladira et al., 2012) The complete genome sequence of Las is available (Duan et al., 2009) and, recently, differences between the Asiatic (China) and North American strains of Las were reported (Chen et al., 2010). Liberibacter species are apparently not seed-transmissible (Hartung et al., 2010; Hilf, 2011).

Some biological traits of the psyllid vectors. Both Diaphorina citri and Trioza erytreae feed on the phloem and are experimentally able to transmit both the Asian and African HLB. Fourth and fifth instar nymphs and adults can acquire and transmit the bacteria during their whole life, after a latent period of ca. 10 days, after feeding times varying from 15 to 20 min for D. citri to 24 h for T. erytreae. Multiplication in the vector has not clearly been demonstrated, the bacteria, however, can be found in large amounts in the haemolymph and saliva glands. For T. erytreae there is evidence that transovarian transmission occurs. Other psyllids that thrive on citrus were not found to transmit the bacteria, except for the black psyllid (Diaphorina communis) which is a Las vector in Bhutan (Donovan et al., 2011). Psyllids like to feed on new vegetation flushes the presence of which constitutes a risk for transmission. Although D. citri does not tolerate frost very well, it survived frosty days of up to -5°C in Florida (Bové, 2006; Bransky and Rogers, 2007; Gottwald, 2010; Gottwald et al., 2007; Halbert and Manjunath, 2004; Manjunath et al., 2008).


(i) Bacteria. Las (transmitted by D. citri in all countries mentioned). South east Asia: Cambodia, China (including Hong Kong), Indonesia, spreading along southern islands of Japan (Shinohara et al., 2006), Laos, Malaysia, Myanmar, Philippines, Taiwan, east Timor, Thailand, and Vietnam; Indian subcontinent: Bangladesh, Bhutan, India, Nepal, and Pakistan; Western Asia: Iran in Sistan-Baluchistan and Hormozgan provinces (Faghihi et al., 2009; Salehi et al., 2012); Indian Ocean: Comoros Islands, Madagascar, Mauritius, Re-
union and Sri Lanka; Arabian peninsula: Saudi Arabia, Yemen in the south west along the Red Sea; Africa: Northern Ethiopia (De Bac et al., 2010); South America: Brazil, mainly Sao Paulo state, since 2004; Caribbean: Cuba (Martinez et al., 2009), Dominican Republic (Matos et al., 2009), Belize (Manjunath et al., 2010); North America: USA [Florida since 2005; Louisiana, 2008; Georgia and South Carolina, 2009; California 2012]; Mexico, 2009. (http://www.pestalert.org/oprDetail.cfm?oprID=382; http://www.pestalert.org/oprDetail.cfm?oprID=321&keyword=citrus%20greening); http://www.pestalert.org/oprDetail.cfm?oprID=516, http://www.pestalert.org/oprDetail.cfm?oprID=401)


Laf and Las. Ethiopia, Mauritius, Reunion, Saudi Arabia and Yemen host both vectors (D. citri and T. erytreae) and both pathogens.

Lam. Brazil. Lam has also been reported (but not confirmed) from one of 97 citrus leaf samples from eight provinces of southern China (Lou et al., 2008).


![Fig. 1](image-url). A. Sweet orange with typical symptoms of huanglongbing or citrus greening (source EPPO, J. Bové). B. sweet orange leaves with more or less typical huanglongbing-induced yellowing. *Spiroplasma citri* or zinc deficiency can cause similar symptoms (source EPPO, J. Bové). C. Mandarin fruits with typical greening (courtesy of J. Gottwald). D. Adult *Diaphorina citri* vector of “*Candidatus Liberibacter asiaticus*”, the Asian huanglongbing strain. E. Nymph of *D. citri*. Size of the adult 3-4 mm [Source Conant et al. (2007)].

**Natural hosts.** Main sources: Anonymous (2007); CABI/EPPO (1998a, 1988b); EPPO (2005c, 2005d); Beattie et al. (2008); Bové (2006); Gottwald et al. (2007).

(i) Liberibacter species. Rutaceae: Most Citrus species and forms are or can be host of Liberibacters. Sweet oranges, mandarins and tangelos (C. reticulata x C. paradisi) are generally the most susceptible showing severe symptoms. Lemons, grapefruits, C. limonia, C. limettioides, rough lemons, kumquats (Fortunella spp.) and citrons are less severely affected. Symptoms are mild on limes and pummelos (C. grandis). However, in Brazil and the USA all commercial Citrus species have a very similar susceptibility. Other confirmed hosts are *Limonia acidissima, Murraya paniculata* or orange jasmine (often used as ornamental and for hedges) and the related or similar *M. exotica*, *M. (Bergera) koenigii*, *Severinia buxifolia* and *Vepris lanceolata* (= *V. undulata* = *Toddalia lanceolata*).

(ii) Vectors (*D. citri* and *T. erytreae*). Rutaceae, all cultivated and wild species listed above and *Clausena anisata* (= *C. inaequalis*) and *Zanthoxylum capense* (= *Fagara capensis*).

**Symptomatology.** Symptoms (Fig. 1A-C) may be confused with those of other diseases and disorders. More typically, the first symptoms are one or more yellowing shoots. Leaves are asymmetrical when the two leaf-halves are compared and exhibit well-defined yellow areas, called blotchy mottling, that may also occur on fruits. With time, yellow spots may intensify, resembling very much zinc deficiency. Veins may become corky, giving the leaves a thicker appearance. Fruits are often smaller, asymmetrically misshaped (lopsided) and show persistent green areas, especially at the stylar end. When infected fruits are cut, yellow-brown vascular bundles and necrotic seeds may be observed. The final stages of the diseases are characterized by severe leaf and fruit drop, twig and stem die-back and death of the trees (especially when infected by Las). For photos of symptoms and psyllids, see [http://anrcatalog.ucdavis.edu/ pdf/8205.pdf](http://anrcatalog.ucdavis.edu/pdf/8205.pdf); [http://entnemdept.ufl.edu/creatures/citrus/acpsyllid.htm](http://entnemdept.ufl.edu/creatures/citrus/acpsyllid.htm); [http://www.forestryimages.org/browse/subthumb.cfm?sub=4695&start=8](http://www.forestryimages.org/browse/subthumb.cfm?sub=4695&start=8); [http://www.eppo.org](http://www.eppo.org).

**Detection and diagnosis.** In the early years detection and diagnosis was mainly based on symptoms, electron microscopy and biological indexing. A monoclonal antibody was developed but proved too specific. PCR (classical, nested, real-time, qualitative real-time and multiplex) is now the main confirmatory test and is routinely used in many areas, allowing also detection in insects and of latent infections in plants (Benyon et al., 2008a; Li et al., 2006a, 2007; Teixeira et al., 2008). Okuda et al. (2005) have developed a species-specific multiplex TaqMan (real-time) PCR for Las, Laf and Lam (with COX primers/probe for the host plant cytochrome oxidase gene as internal control). For a very sensitive combination of nested PCR and TaqMan (real-time) PCR in a single-tube test see Lin et al. (2010) and a real-time PCR protocol based on prophage genes, that is useful for world-wide detection, also in difficult hosts, see Morgan et al. (2012).

**Epidemiology.** Main sources: Bové (2006); Gottwald (2010); Gottwald et al., (2007). HLB epidemics develop rather slowly in time (several to more than 12 years), but the disease may spread quite rapidly in a grove (in several years trees may show severe symptoms and within 7-10 years orchards may become unproductive, depending on the age of the trees and the presence and number of vectors). A complicating factor is that many trees may have been already latently infected for a considerable time before symptoms become obvious. The number of latent infected trees may be two- to manifold as compared to symptomatic trees, which hinders early detection of HLB and frustrates eradication campaigns. Spread of vectors and disease has a tree-to-tree pattern although also further spread (a few kilometres) from an infection focus may be possible. Psyllids migrate mostly when host plants are flushing. Long distance dispersal is by infected planting material or by infected psyllids accidentally transported by man on plant material or otherwise. The presence of HLB in a tree may be sectorial, thus the bacteria may not be detectable in the symptomless part. How far psyllids can be carried by high air streams and spread over long distances is still unknown, but its occurrence cannot be excluded. Seed transmission of virulent bacteria has not yet been demonstrated.

**Damage and losses.** Since resistance against HLB is a dangerous and devastating disease and, as yet, no sources of resistance have been found or created. Yield is decreased, mainly because of reduced growth and fruit drop, and the fruit quality impaired. Yield losses may be 30-100% and, within 7-10 years from planting, the groves may lose productivity completely. It has been calculated that ca. 100 million trees have been killed in south-east Asia, India, Arabian peninsula, and South Africa, leading to decline of the citrus industry in these areas. In southwestern Saudi Arabia, sweet orange and mandarin have practically disappeared already during the 1970s. Since 2004, ca. 1 million trees have been destroyed in Brazil due to HLB infection. In Florida HLB was detected for the first time in 2005 and by 2009 it has spread to most citrus-growing areas, so that eradication efforts were given up (Bové, 2006; Gottwald, 2010) and the select agent status for all three Liberibacter species by USDA APHIS abandoned.
Main risk factors. HLB and its vectors have not yet been reported from the European mainland or the Mediterranean basin, where there are no local psyllid vectors known. Long distance spread of Liberibacter spp. and vectors is possible and will mainly be with planting material. In Kenya, infected breeding material from abroad was suspected to be the source of initial infection (Magomere et al., 2009). Note that HLB is approaching the Mediterranean basin mainly from three sides: (i) Saudi Arabia, where both Laf and Las are present and could move further north (e.g. also with people travelling to and from Mecca), possibly also for Las and its vector should global warming continue. Note that Las and its vector could also establish in subtropical Florida; (ii) Africa, where Laf and its vector are already present in Ethiopia and Somalia and Las is established in north Ethiopia; (iii) Iran, where Las has been reported in 2009. Note that D. citri was present in Florida since 1998 and HLB followed in 2004 (also see Bové, 2006), and that the Laf vector is already present in the Atlantic, i.e. the Canary, Madeira, Porto Santos and St. Helena islands. Murraya paniculata (orange jasmine), a popular landscape plant, played an important role in spread of HLB and D. citri in Florida (and probably also to other states), also via garden centres (Manjunath et al., 2008). Murraya species of Asian origin have been marketed (also in bonsai form) in Europe for quite some years. Infections may go unnoticed for several years after they became established due to latency or overlooking of the early symptoms. HLB is suspected to have been present several years before its official detection in Florida in 2005 (Gottwald et al., 2007; Gottwald, 2010).

Prevention and management following first introduction. Once established, HLB bacteria and vectors are very difficult to control. Main strategies are geographical isolation and certification/indexing programs for budwood sources and nursery production in insect-proof greenhouses, chemical and/or biological vector control and intensive surveying (by visual inspection and laboratory testing) with removal and destruction of infected trees (both visually and latently infected trees) wherever possible. Healthy budwood can be obtained by shoot-tip grafting, or alternatively by heat treatment, e.g. water-saturated hot air at 49°C for 50 min and fumigation of budwood against HLB vectors is possible (EPPO, 1988). The visual inspections in the groves should be very intensive and performed from moving inspection platforms. Chemical sprays, for vector control, to be effective, should also be performed frequently. Antibiotic treatment has been abandoned completely as it was not effective and is not human and environmentally friendly. Biological control was effective in Reunion Island, with hymenopterous ectoparasites Tamarixia dryi for Laf and T. radiates for Las, probably because hyperparasites of the parasite were absent. At present the only successful prevention/control methods appears to be the one followed in São Paulo state in Brazil, following the introduction of huanglongbing in 2004. The control program that was immediately installed in 2004 by responsible organisations, became very successful after eight years, when disease incidence on more than 200,000 ha was reduced to less than 1%. This was because: (i) at the beginning of the program the disease incidence was still low (less than 8%) and (ii) it could be executed on large, easy to handle farms (>500 ha). The basic principles of the so-called Three-Pronged System (TPS) are: (i) intensive vector control, (ii) reduction of inoculum by tracing, identifying and removing infected trees, and (iii) production of healthy nursery stock in covered, insect proof nurseries and their use for re-planting (Bové, 2012; Belasque et al., 2010).

Genetic resistance development has started, but is still in its infancy (Grosser et al., 2008). Still, breeding for resistance will remain one of the most important ways to control this disease and those described in the following paragraphs.

HLB bacteria and their vectors should be prevented from entering the Mediterranean basin by strict inspection and laboratory testing of imported breeding material from risk areas. In the European Union the importation of citrus planting material from third (non-EU) countries is already prohibited (Annex III A of Directive 2000/29/EC) and EPPO (EPPO, 1988) and the introduction of plants for planting and cut citrus branches from countries where HLB or its vectors occur should also be prohibited. However, when such material is imported, it should be fumigated and quarantined for at least two years. Since Liberibacter spp., D. citri and T. erythraeae are quarantine organisms included in the EPPO A1 list and EC Annex II/Ia list, the following additional measures can be enforced [see also Bassanezi et al., 2008; Bové, 2006; EPPO, 1988; Gottwald, 2010; USDA, 2010. For (pro-active) measures advised by the Australian government, see http://www.agnet.org/library/eb/607/]:

- Murraya spp. and ornamental citrus spp. already present in garden centres or in plantations should be surveyed, including vector sampling. In suspect areas this could also been done in (young) citrus orchards.
- Importations from risk areas of these plants strictly inspected and laboratory-tested.
- Proactive training of diagnosticians, surveyors, nursery managers and producers.
- Production in citrus nurseries should be in secure, insect-proof screenhouses.
- Once an introduction has occurred, infected trees should be destroyed and vectors controlled by chemical insecticides.
- When executing severe eradication programs, governments should consider compensation or facilitate insurance to secure full cooperation also from small growers and private tree-owners.
Further training of all parties involved and development of a public information program.

Leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by *Xylella fastidiosa*. General. The bacterium *Xylella fastidiosa* is a xylem-inhabiting, vector-transmitted, very slow growing, Gram-negative bacterium. It was cultured and described for the first time in 1987 in the USA as the cause of Pierce’s disease (PD) of grapevine (disease observed already in 1884) and as the cause of phony peach disease (PPD) in peach, *Prunus persica* (disease observed in 1890 in the USA). In 1993 *X. fastidiosa* was identified as the cause of citrus variegated chlorosis (CVC) or citrus X disease in Brazil. The bacterium also causes a number of so-called leaf scorch diseases to *Prunus* spp. (including almond leaf scorch or ALS in *P. amygdalus* and plum leaf scald or PLS in *P. domestica*), *Acer* spp., *Carya illinoinsensis* (pecan), *Coffea arabica* (CLC, in Brazil isolated in 1995 and also pathogenic to citrus), *Hedera helix*, *Morus rubra*, *Nerium oleander* (OLS), *Platanus occidentalis* (Fig. 2A), *Quercus* spp. and *Ulmus americana*. *X. fastidiosa* is also the agent of alfalfa dwarf and of wilting of *Vinca major*. Many wild plants such as grasses, sedges and trees may carry the pathogen, often without showing symptoms. None of these diseases is apparently seed-borne. Seed transmission, however, has been reported in sweet orange, *C. sinensis* (Li et al., 2003). They occur mainly in tropical/subtropical areas, although leaf scorch diseases are present also in much colder climate, e.g. oak leaf scorch in eastern North America up to Canada.

Several pathogenic varieties of the bacterium have been described, that are often host-specific (e.g., the PD strain will not cause disease if introduced to peach or plum). The following subspecies have been described: (i) *Xylella fastidiosa* subsp. *fastidiosa* (erroneously named *X. f.* subsp. *piercei*), PD and LSA, strains from cultivated grape, alfalfa, almond (two), and maple; (ii) *X. fastidiosa* subsp. *multiplex*, PPD and PLS, strains from peach, elm, plum, pigeon grape, sycamore, almond and recently pecan (Melanson et al., 2012); (iii) *X. fastidiosa* subsp. *pauca*, CVC, strains from citrus and probably those from coffee (CLC); (iv) *X. fastidiosa* subsp. *sandy*, strains from *Nerium oleander* (OLS); (v) *X. fastidiosa* subsp. *tasbke*, strains from the ornamental tree *Chitalpa tasbentensis*.

*X. fastidiosa* isolates are genetically very similar, but studies on their biological traits have indicated differences in virulence and symptomatology. As mentioned, taxonomic analyses have identified several subspecies, and phylogenetic analyses of housekeeping genes have shown broad host-based genetic differences. However, results are still inconclusive for genetic differentiation of isolates within subspecies. In a recent study, sequences of nine non-housekeeping genes were used to study 54 *X. fastidiosa* isolates infecting different host plants. Strains could again be divided into the known *X. fastidiosa* subspecies, but also showed new within-subspecies differentiation, including geographic differentiation, and some host-based isolate variation and specificity (Parker et al., 2012).

*Xylella fastidiosa* is a quarantine organism on the EPPO A1 list and EC Annex II/Ia list. For further and extensive data on biology, hosts, geographical distribution and epidemiology see recent reviews (Purcell, 1997; Chatterjee et al., 2008; Janse, 2010; Janse and Obradovic, 2010) and the extensive *X. fastidiosa* website, edited and maintained by A. Purcell and R. Almeida: http://www.cnr.berkeley.edu/xylella/. For diagnostic methods, see EPPO 2005a and Janse et al. (2012).

Hosts. A full host list can be found on http://

![Fig. 2. A. Sycamore (Platanus spp.) leaf with scorch symptoms caused by the bacterium Xylella fastidiosa. (Courtesy A.H. Purcell). B. Philaenus spumarius, a potential vector of X. fastidiosa with a widespread occurrence in Europe (Source EPPO).](attachment:image)
www.cnr.berkeley.edu/xylella/. Some hosts relevant for the Mediterranean basin are: *Nerium oleander*, *Platanus occidentalis* (sycamore), *Quercus* spp. (oak), *Ulmus americana* (elm tree), *Ambrosia artemisiifolia* (ragweed), *Morus alba* (white mulberry), *Prunus angustifolia* (Chickasaw plum) and *Rubus* spp. In Taiwan a pear leaf scorch was described in 1990 on *Pyrus pyrifolia* (Japanese pear), cv. Hengshan and *P. serotina* (Asian pear), which was found to be caused by a bacterium very similar to *X. fastidiosa*, but differing from North and South American strains in serological properties and housekeeping gene sequences (Leu and Su, 1993; Chen et al., 2006). Hosts such as Asian pear or nashi, were planted as a novelty crop in southern China in the 1980’s. It is not known whether the latter two species have ever been subjected to surveys for *X. fastidiosa*.

**Symptoms and transmission.** For symptoms on different hosts, see http://www.cnr.berkeley.edu/xylella/. In general, early symptoms are a slight chlorosis or bronzing along leaf margin or tip that intensifies and that may become water-soaked before browning and drying. These symptoms are first found on a few branches, then they extend to most or the whole canopy (so-called leaf scorch or nashis, were planted as a novelty crop in southern Europe especially in the 1980’s. It is not known whether the latter two species have ever been subjected to surveys for *X. fastidiosa*.

**Risks.** *X. fastidiosa* is an emerging threat in the south-west USA, mainly due to recent establishment of the glassy-winged sharpshooter (GWSS, *H. vitripennis*), providing much more efficient transmission than local vectors, and leading to very serious outbreaks of PD in grapevine, ALS and OLS. GWSS probably first entered California as eggs deposited in plant tissues. In Central and South America *X. fastidiosa* has become very noxious due to the rapid expansion (most likely via distribution of infected planting material) of CVC in *Citrus*, leading to more than a third of all trees in the area having symptoms of CVC, and CLC in coffee. As to Europe, there are only a few unconfirmed reports of the presence of *X. fastidiosa* in the grapevine in Kosovo (Berisha et al., 1998) and in almond in Turkey (Güldür et al., 2005). Since *X. fastidiosa* has more than 150 hosts and many of them, including *Vitis*, were are imported (often as planting material), risk of introduction (especially in latent form) must not be underestimated. Absence of the diseases caused by *X. fastidiosa* will mainly be due to the absence of suitable vectors. However, introduction of the pathogen and infected vectors with plant material cannot be excluded. Moreover, also local Cicadellidae (see above) could become potential vectors. Therefore, *X. fastidiosa* has the A1 quarantine status in the EPPO region and *H. vitripennis* that has a very large host range and feeds on almond, peach and plum was recently placed on the EPPO alert list. As in the more southern parts of the USA, European *Vitis* varieties are very susceptible to *X. fastidiosa*. This is really a risk if a vector that could survive the winters of southern Europe were introduced, the pathogen would become established in wild hosts (wild and domestic plums and wild cherry are symptomless reservoirs in the USA) and cause spring infections that are most likely to persist over the years. The same risk holds true for citrus (sweet oranges, mandarins, and tangerines) and other hosts, such as almond, plum and peach that are widely grown in southern Europe, especially in the warmer Mediterranean basin, where a disease-favourable combination of warm nights, regular rainfall/high humidity and long growing season, is present. The conclusion is that *X. fastidiosa* is a real threat for Europe, not only for *Vitis* and *Citrus* but also for stone fruits (almond, peach and plum) and oleander (GWSS likes to feed on oleander), that is difficult to prevent from entering and difficult to control once established, deserving more attention than up till now. Resistance in European grapes is scarce or even absent. Vector control proved not to be very effective in the USA. Cultural practices to keep plants in optimum condition are of importance, but not sufficient, and the use of avirulent strains for cross-protection is still in its infancy.

**Citrus canker, caused by Xanthomonas citri pv. citri (X. axonopodis pv. citri).** General. Asian citrus canker, a spot disease of *Citrus* spp. characterized by corky le-
sions on leaves, fruits and twigs, was first described by Stevens in 1914 and the causal bacterium, now named *Xanthomonas citri* pv. *citri* by Hasse in 1915 in the USA. However, citrus canker had been observed earlier in Asia as it originates most probably from China. Symptoms and spread of the bacterium are enhanced by the activity of the citrus leaf miner, *Phyllocnistis citrella*, which occurs in nearly all citrus growing areas of the world. In Europe is is established in the Iberian peninsula, Corsica, Italy, Greece and Montenegro (http://www.bladmineerders.nl/minersf/lepidoptera/ramin/phyllocnistis/citrella/citrella.htm). This insect, however, is not a true vector. Grapefruit (*C. aurantium*), lemon (*C. limon*) and *C. bystrix* are the most susceptible species *X. citri* pv. *citri*, is a Gram-negative bacterium with one polar flagellum, forming yellow colonies on agar media. Over the years, several much related diseases and pathogenic bacteria have been described from *Citrus* and some other Rutaceae spp. in Asia, North and South America:

(i) *X. citri* pv. *citri* (Xcc) (syn. *X. citri* subsp. *citri*, *X. axonopodis* pv. *citri*) formerly named group A or Asiatic strain is the most aggressive form. Other names for this pathogen have been *X. citri*, *X. smitthii* subsp. *citri* and *X. campestris* pv. *citri* (A group) (Schaad et al., 2005, 2006; Ah-You et al., 2009). Strains with a different host range, named pathotype A* strains, infecting Mexican lime and Tahiti lime (*C. latifolia*) but not grapefruit (C. *paradisi*) have been reported from Cambodia, Iran, India, Oman and Saudi Arabia. The so-called Aw strains that infect Mexican lime and alemow (*C. macrophylla*) were described from Florida (Ngoc et al., 2009, 2010; Sun et al., 2004).

(ii) *X. citri* pv. *aurantifolii* (syn. *X. axonopodis* pv. *aurantifolii*, *X. fuscans* pv. *aurantifolii*), formerly named *X. citri* group B or cancrosis B, group C or Mexican lime cancrosis, and group D or Citrus bacteria, causes a relatively mild disease in Mexico and South America. It was also reported from Swingle citromelo rootstock (*C. paradisi* × *Poncirus trifolata*) from Brazil (Jaciani et al., 2009).

(iii) *X. citri* pv. *bilvae* (syn. *X. campestris* pv. *bilvae*), is an ill-defined pathogen, causing shot-hole disease and canker on Bael (*Aegle marmelos*), a member of the family Rutaceae described in India in 1953 (Ngoc et al., 2010).

(iv) *X. alfalfa* subsp. *citrumelonis* (syn. *X. campestris* pv. *citrumelo*), is the cause of citrus bacterial spot, formerly named Group E strains in Florida, USA (Schaad et al., 2005).

Only Xcc, the agent of Asian citrus canker (A, A* and Aw strains) is described here. It is a quarantine organism on the EPPO A1 list and EC Annex II/A list. Main reference sources are Brunings and Gabriel (2003), Das (2003), Gottwald et al. (2002), Rogers et al. (2010), Lowe (2010) and, Ngoc et al., (2009), Golmohammadi et al. (2007) and EPPO (2005b) for diagnostic methods.

**Geographical distribution** Xcc originates from and is widespread in Asia, including Georgia, Iran, Iraq, Oman, Saudi Arabia, UAE and Yemen. Australia (eradicated), Argentina, Belau, Brazil, Caroline Islands, Cocos Islands, Comoros, Congo Democratic Republic, Ivory Coast, Fiji, Gabon, Madagascar, Mauritius, Mozambique (eradicated), Netherlands Antilles, New Zealand (eradicated), Micronesia, Palau, Papua New Guinea, Paraguay, Reunion, Seychelles, South Africa (eradicated), Uruguay, USA (CABI/EPPO, 2006). There are recent reports from Somalia (Balestra et al., 2008) Mali (Traoré et al., 2009) and Ethiopia (A* strains, Desro et al., 2009).

**Hosts.** Cultivated hosts are *Aegle marmelos* (golden apple), *Casimiroa edulis* (white sapote), *C. aurantium* (sour orange), *C. bystrix* (maitius bitter orange), *C. junos* (yuzu), *C. limetta* (sweet lemon tree), *C. limon* (lemon), *C. madurensis* (calamondin), *C. maxima* (pummelo), *C. medica* (citron), *C. natsudaidai* (natsudaidai), *C. reshni* (Cleopatra mandarin), *C. reticulata* (mandarin), *C. reticulata* × *Poncirus trifolata* (citromelo), *C. sinensis* (navel orange), *C. sunki* (sour mandarin), *C. tankan* (tankan mandarin), *Citrus unshiu* (satsuma), *C. x paradisi* (grapefruit), *Eremocitrus glau- ca* (Australian desert lime), *Limonia acidissima* (elephant apple), *Poncirus trifolata* (trifoliate orange or Japanese bitter orange). Minor hosts are *Fortunella japonica* (round kumquat) and *F. margarita* (oval kumquat). Wild hosts are *Ageratum conyzoides* (billy goat weed), *Severinia buxi- folia* (box orange or boxthorn) and *Swinglea glutinosa* (= *Aegle decandra*, *Limonia glutinosa*).

**Symptoms and transmission.** (For pictures of symptoms and of the citrus leaf miner, *P. citrella*, see http://www.plantmanagementnetwork.org/pub/php/review/citruscanker/). Small spots, first visible on the upper leaf blade, appear on the leaves, shoots, twigs and fruits, to become raised pustules or blister-like eruptions (Fig. 3A). With time, the lesions increase size (up to 10 mm) and turn brown and necrotic with a depressed centre, and are sometimes surrounded by a yellow halo. On the fruits, the lesions can be mistaken for scale insects (e.g. the California red scale, *Aonidiella aurantii*)). The bacterium is a wound parasite and, as mentioned, the citrus leaf miner (*P. citrella*) contributes to disease spread and severity. Citrus canker is especially epidemic and damaging on seedlings and young trees, especially after storms (hurricanes) under warm weather conditions, but because of dependence on these weather conditions epidemics are sporadic. Full-grown trees show much less disease and damage (Goto, 1992). The bacterium can survive in a latent form in and on diseased shoots and discoloured bark tissue of the trunk, and may reoccur suddenly after several years (sometimes even as long as 10 years).

**Risks and damage.** Heavy losses were reported in epidemics, due to premature fruit drop and fruits with
spots that cannot be marketed or start rotting, thus must be destroyed. Furthermore, quarantine measures such as burning of trees and destruction of fruits may add to these losses (Goto, 1992). In severe cases, almost 100% of the fruits and leaves of young, susceptible trees may be infected and the plant growth is delayed for a number of years. The direct Government costs in the USA for the eradication activities from 1995 to 2006 were calculated to be more than $1.3 billion. From 2006 to 2009, together with costs for the control of Citrus huanglongbing the amount was $90 million. The citrus acreage in Florida decreased since 1996 by ca. 33% (Lowe, 2010). In January 2006, the USDA determined that canker had become so widespread in Florida that eradication was no longer feasible. Use of healthy planting material and use of other measures, including weather forecasting, in an integrated way have been applied in the control of Citrus canker with some success. Resistance has been found especially in *C. mitis* (calamondin) and *Fortunella* (kumquat). *C. reticulata* (mandarin) is tolerant.

One should remember that epidemics of citrus canker on mature plants are sporadic and very dependent on weather conditions (rainstorms, hurricanes), which are less prevalent in the Mediterranean basin. On the other hand, it should also be realized that the citrus leaf miner is already widespread in this area. Seed transmission has not been observed. Long distance dispersal is by infected planting material or infected fruits. In the past, ornamental *Citrus* have been imported from Asia.

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**Fig. 3.** A. Wart-like excrescences induced by *Xanthomonas citri* pv. *citri* on sweet orange (*Citrus sinensis*). B-E. Symptoms of *Xanthomonas citri* subsp. *mangiferaeindicae* infection on mango (*Mangifera indica*). Necrotic leaf spots (B). Twig canker (C). Fruit spots with gummy exudates (D). Close up of the spots (E) [Gagnevin and Opruvost (2001). Courtesy O. Pruvost]. F. Necrotic spots on pomegranate (*Punica granatum*), caused by *Xanthomonas axonopodis* pv. *punicae* (source Dr. R. Kumar, http://www.nhm.nic.in/Vasanta_Pome.ppt#294,22,Slide 223). G. Necrotic lesions along the main vein of leaves of guava (*Psidium guajava*), caused by *Erwinia psidii* (Source: Prof. M.E.S. Papa, Sao Paulo University Brazil and APSnet Image Resources). H. Watersoaked leaf spots on passion fruit (*Passiflora* spec.), caused by *Xanthomonas campestris* pv. *passiflorae* (Courtesy: Dr. S.O.M. El Tassa, Departamento de Fitosanidade, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil).
to Europe and Xcc has been intercepted on this material (author's personal experience). However, the risk of dispersal by infected fruit was evaluated to be very small or absent if fruits are disinfected before shipment (Gottwald et al., 2009). Without disinfection, survival chances in symptomatic fruits are apparently also small (Shiotani et al., 2009), but not impossible, as shown by the identification of Xcc on imported fruits by the Dutch Plant Protection Service (author's personal experience). Since Xcc is endemic and spreading in countries surrounding the Mediterranean basin, some of which have also huanglongbing problems, and the citrus leaf miner is widespread in the area, the conclusion is that this pathogen is a real and emerging threat.

BACTERIAL DISEASES WITH AN EMERGING CHARACTER, BUT NOT YET PRESENT IN EUROPE OR THE MEDITERRANEAN BASIN

Black spot of mango - Xanthomonas citri pv. mangiferaeindicae. A leaf spot and canker disease of mango (Mangifera indica) called bacterial black spot, was described in South Africa by Doidge (1915) who named the causal agent Bacillus mangiferae [later also named Xanthomonas mangiferaeindicae, and X. campestris (axonopodis) pv. mangiferaeindicae]. Ah-You et al. (2007, 2009) showed that this bacterium is much related to a pathogen from cashew (Anacardium occidentale) and named the two bacteria X. citri subspp. mangiferaeindicae and X. citri pv. anacardii, respectively. Both mango and cashew belong to the family Anacardiaceae. Symptoms of black spot start as small water-soaked spots that become later raised and necrotic, sometimes surrounded by a narrow yellow halo. On the fruits the water-soaked spots become star-shaped and crack and often show exuding gum. Severe infection under influence of rainstorms may lead to premature leaf and fruit drop, twig cankers and twig death (Fig. 3 B-E). Other (rare) hosts are ambarella (Spondias dulcis, syn. S. cytherea) and Brazilian pepper (Schinus terebinthifolius), both belonging to the Anacardiaceae. When other diseases and pests are controlled, black spot is a limiting disease to mango production, because it is very difficult to contain. In most susceptible cultivars up to 100% fruit loss may occur. Many commercial cultivars are very susceptible. In 1996 and 1997, severe black spot epidemics were observed in many mango-growing areas of South Africa, causing almost 100% fruit loss on the most susceptible cultivars and ca. $1 million economic loss. There is production of mango in Europe and the Mediterranean basin (e.g. Spain, Italy, Israel and Portugal). Black spot occurs in Australia, Comoro Islands, many areas in southern and eastern Africa and Asia, Mauritius, New Caledonia, Reunion, Taiwan, and the United Arab Emirates (Gagnevin and Pruvost, 2001). Discrimination of strains from mango and some related hosts and from different geographic origin (Asia, Africa and Brazil) was possible using RFLP (Gagnevin et al., 1997). Long distance dissemination of the pathogen is thought to be by infected planting material. Epiphytic/endophytic populations of the pathogen occur (Pruvost et al., 2009). Seed transmission has not been demonstrated.

Bacterial blight of pomegranate - Xanthomonas axonopodis pv. punicae. A bacterial disease on pomegranate (Punica granatum, family Lythraceae) was observed for the first time in 1952 in New Delhi, India and described by Hingorani and Sing (1959) as being induced by Xanthomonas punicae (later classified as X. campestris pv. punicae and X. axonopodis pv. punicae). First symptoms are water-soaked spots on leaves and fruits (Fig. 3F). On the leaves, spots become necrotic and, when they coalesce, severe leaf drop may occur. Fruits may crack and drop. Black necrotic spots occur on branches, that become weak and may split. Pomegranate is produced mainly by India (50%), Iran (35%), some of the former Soviet Union states and in the Mediterranean basin, especially in Spain (2.5%), Morocco, Egypt and Turkey. Bacterial blight has developed into a very serious disease in India, where it causes very heavy losses (up to 100%) in many pomegranate-growing areas. (Kumar et al., 2006). Dispersal at a site is by rain splash, insects and tools, whereas over long distances is by infected planting material. Like X. citri pv. citri and pv. mangiferaeindicae, stormy weather conditions are important for epidemic outbreaks. X.a. pv. punicae overwinters in infected leaves of neem (Azadirachta indica, family Meliaceae) planted along pomegranate fields (Dhasandar et al., 2004; Sharma et al., 2008; Kumar et al., 2009) Since these former two pathogens have already spread from the more eastern parts of Asia, X. a. pv. punicae can be seen also as a potential threat that may soon show its presence closer to the Mediterranean basin.

Bacterial blight of guava - Erwinia psidii. A vascular disease of guava (Psidium guajava) was reported as bacterial blight from Brazil and the causal agent denoted Erwinia psidii (Neto et al., 1987), was held responsible for the outbreaks in the main production areas of southeastern and central regions of the country (Tokeshi et al., 1980). Symptoms are local and systemic. Leaves show large necrotic lesions along the main veins (Fig. 3G) and at the margins (scorching), or small water-soaked spots, sometimes with a chlorotic halo, that later become necrotic and coalesce causing leaf drop. When bacteria reach the xylem they spread into branches, trunk and roots. In severe cases, trees are defoliated and die. Symptoms on the fruits are not common. This disease could be of importance to guava-growing countries such as Egypt that produces yearly some 230,000 tons of guava fruits, being the fifth producer in the world after India, Pak-
istan, Brazil and Mexico. In Brazil, pathogen dispersal often occurs with contaminated planting material (Marques et al., 2007; Teixeira et al., 2008b).

**Bacterial spot of passion fruit - *Xanthomonas campestris pv. passiflorae***. A destructive disease of cultivated passion fruit (*Passiflora edulis* and *P. edulis* var. *flavicarpa*), was described by Pereira in 1969 in Brazil (El Tassa, 2002). This disease is characterized by the presence of water-soaked, greasy lesions of irregular shape on the leaves, surrounded by chlorotic areas that result in extensive necrosis when they coalesce (Fig. 3H). Fruits display greasy spots that make them unsuitable for consumption and industrial processing. This disease has an emerging character in Brazil, where ca. 25,000 ha are given over to passion fruit and has become a major problem to production. Hosts are *Passiflora alata*, *P. amethystina*, *P. coccinea*, *P. edulis*, *P. edulis* var. *flavicarpa*, *P. maliformis*, *P. nitida* and *P. serrato-digitata* (Neto et al., 1984; Torres Filho and Ponte, 1994; Gonçalves and Rosato, 2000; Lopes et al., 2006).

**BACTERIAL DISEASES/PATHOGENS WITH AN EMERGING CHARACTER, ALREADY OCCURRING IN EUROPE OR THE MEDITERRANEAN BASIN**

**Almond witches’ broom - *Candidatus Phytoplasma phoenicium***. This devastating disease of almond (*Prunus amygdalus*), shows typical witches’ broom symptoms, i.e. small yellow leaves on proliferating shoots that wither and die in later stages (Fig. 4.A-C). It
was reported from Lebanon by Choueiri et al. (2001), and was classified and named by Verdin et al. (2003). *Candidatus* Phyttoplasma phoenicium belongs to the pigeon pea witches’ broom group (16SrIX). In a few years time, more than 100,000 trees were killed in different areas in Lebanon. This phytoplasma was also found on peach and nectarine (Abou-Jawdah et al., 2009) and was recently also reported from Iran on almond (Zirak et al., 2009) and on GF-677 (*Prunus amygdalus*) was recently also reported from Iran on almond (Zirak et al., 2009). Vectors are suspected to be leafhopper which have not yet been identified (Abou-Jawdah et al., 2011). This pathogen is not included in quarantine lists, but certainly deserves attention as an emerging threat for almond. Stem cutting culture with thermotherapy was successfully used for regeneration of phytoplasma-free plantlets of almond (Chalak et al., 2010). Recently Davis et al. (2010) have reported a phytoplasma closely related to *Ca. P. phoenicium* as the cause of witches’ broom affecting *Juniperus occidentalis* (western juniper) in Oregon (USA).

**Potato stolbur - *Candidatus* Phyttoplasma solani.**

*Ca. Phyttoplasma solani* is a non-culturable, insect-transmitted bacterium that induces bushy growth and malformations in solanaceous (Fig. 4D) and other hosts (EPPO/CABI 1996) and is on the EPPO A2 quarantine list. It belongs to the so-called Aster yellows or stolbur (16Sr-XII-A) phytoplasma group, the latter having a very wide host range (‘Bois noir’ of grapevine is caused by a phytoplasma of the same group). Severe outbreaks occur under dry weather conditions when vector populations develop explosively on wild hosts. In some years, substantial damage was reported from south-east Europe and Russia. Spreading in potato is slow. Vectors are mainly leafhoppers of the family Cixiidae, the most important of which are the polyfagous *Hyalostethus obsoletus*, the true bug *Lygus pratensis* (family Miridae) and some other cicadas, such as *Macrosteles quadripunctatus*. Potato stolbur has been reported from Austria, Bulgaria, Czech Republic, Germany, France, Greece, Hungary, Italy (Berger et al., 2009), Israel, Poland, Romania, Russia, Serbia, Switzerland, Turkey and Ukraine. Main hosts for the bacterium are potato, tomato, eggplant, pepper and weeds such as black nightshade, bindweeds (*Convolvulus arvensis, Calystegia sepium*), stinging nettle, *Cardaria* or *Lepidium* and *Lavandula*. Larvae of cicadellids feed on the roots from which they can acquire and transmit the bacterium. Potato stolbur might spread more, when the climate warms up. *H. obsoletus* recently spread in Germany from the Moselle valley and by the end of 2009 stolbur phytoplasma was detected in association with bindweed in Rheinland-Pfalz (EPPO Reporting Service 2010/155). In eastern Europe (Bulgaria, Romania, Serbia), *Ca. phyttoplasma solani* has been causing for many years a disease transmitted by *Reptalus panzerti* (Jovic et al., 2009) called ‘corn reddening’, responsible for 10-90% crop losses. Diseased plants show a red discolouration of stems and main leaf vein and abnormal ears. In Romania the cultivation of potato cv. Lady Rosetta was stopped in disease-sensitive areas, severe losses occurred from 2006-2008, in 2008 circa 75 ha were infected, of which 45 ha suffered total crop loss. Also from Russia, the region Severe damages were reported in 2003 from Russia in an area around Krasnodar (www.costphytoplasma.eu) as well as in the Czech Republic (Navrátil et al., 2009). *Ca. P. solani* does not persist much in potatoes during storage, so that only few diseased plants develop from infected tubers. Late infections do not influence the yield. The disease should not be a problem in a well-tended crop, were healthy, certified planting material is used and where a careful weed and vector control takes place.

**Zebra chip disease of potato or psyllid yellows of solanaceae and carrot - *Candidatus Liberibacter solanacearum* (synonym *Ca. Liberibacter psyllourous*).**

This non-culturable bacterium is closely related to earlier mentioned Liberibacter species occurring in *Citrus*, and has been placed on the EPPO alert list (http://www.eppo.org/QUARANTINE/Alert_List/bacteria/Liberibacter_psyllourous.htm). *Ca. Liberibacter solanacearum* (*CaLS*) was first described in New Zealand in 2008 (Lieftink et al., 2011) as *Ca. L. psyllourous*, and was later found also the USA (Hansen et al., 2008) where it has an emerging character. In the USA it has been reported from Texas in 2010 (French-Monar et al., 2010) and in 2011 from Idaho, Washington state and Oregon (Crosslin et al., 2012). It also occurs in Guatemala, Honduras, Canada and, in tomato, in Mexico (Munyaneza et al., 2010c). The tomato/potato psyllid *Bactericera cockerelli* is a vector that feeds mainly on solanaceous hosts, but it occurs on many other hosts and causes so-called psyllid yellows. It is present in North and Central America and, recently (2000), it appeared in New Zealand. The bacterium can be spread with potato seed, tomato plantlets and fruits. Seed transmission has not been reported. Apparently, *CaLS* is quite versatile and adapted to diverse climatic regions such as desert, steppe, Mediterranean, marine coast, humid continental and humid subtropical. Its hosts are *Capsicum annuum, C. frutescens, Lycopersicon esculentum, Physalis peruviana, Solanum betaceum* and *S. tuberosum* (Fig. 5A). The disease is called Zebra chip because characteristic brown stripes develop when potato tubers are cooked (Fig. 5B). The complete genome sequence of *CaLS* has been determined (Lin et al., 2011). More recent records of *CaLS* in carrot (*Daucus carota*) are from: (i) Finland where the bacterium is transmitted by the psyllid *Triozia apicalis* and causes, together with its vector, substantial crop losses. Infected plants show typical leaf curling, yellow and purple discoloration of the leaves, stunting of roots and shoots, and proliferation of secondary roots (Munyaneza et al., 2010a, 2010b); (ii)
Canary Islands (Tenerife, since 2009) where the bacterium is apparently transmitted by the psyllid Bactericera trigonica (Alfaro-Fernández et al., 2012a). Subsequently it has also been reported from mainland Spain, where it infects celery (Apium graveolens) in several regions, also in mixed infections with Ca. P. solani and, strangely enough, with the bacterium Spiroplasma citri (Alfaro-Fernández et al., 2012b); (iii) Norway (since 2011) where CaLS causes extensive damage to commercial fields with an incidence of 10-100% (Munyaneza et al., 2012a); (iv) Sweden, since 2011 (Munyaneza et al., 2012b). Until now, CaLS has not been reported from potato, probably because the vector B. cockerelli does not occur in Europe.

Other phytoplasmas that could become a threat will not be treated in this review, apart from their mentioning in Table 1. For a recent overview, see COST Action FA0807 “Integrated Management of Phytoplasma Epidemics in Different Crop Systems”.

Harmless (?) “Candidatus Liberibacter europaeus” found in Cacopsylla pyri. An apparent harmless Liberibacter species, named “Candidatus L. europaeus” was reported from Italy to occur in a high percentage of the psyllid Cacopsylla pyri that thrives on pear trees (Pyrus communis). The bacterium could be experimentally transmitted by the psyllid to pear but, apparently, ut remains until now non-pathogenic (Raddadi et al., 2011).

### Table 1. Emerging phytoplasmas reported at the Meeting on “Emerging phytoplasma diseases of stone fruits and other crops and their possible impact on EU countries”, held in 2011 in Istanbul, Turkey.

<table>
<thead>
<tr>
<th>Name</th>
<th>16S rRNA Group</th>
<th>Latin name</th>
<th>Country where reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almond witches’ broom</td>
<td>16SrIX-B</td>
<td>“Ca. P. phoenicium”</td>
<td>Lebanon</td>
</tr>
<tr>
<td>Cassava frog skin</td>
<td>16SrIII-L</td>
<td>“Ca. P. phoenicium”</td>
<td>Colombia</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16SrI-B</td>
<td>“Ca. P. asteris”</td>
<td>Italy, South Africa</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16SrVII-A</td>
<td>“Ca. P. fraxini”</td>
<td>Chile</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16SrXII</td>
<td>“Ca. P. ziziphi”</td>
<td>Iran</td>
</tr>
<tr>
<td>Lime witches’ broom</td>
<td>16SrII-B</td>
<td>“Ca. P. aurantifolia”</td>
<td>China, Korea, Italy</td>
</tr>
<tr>
<td>Potato purple top wilt</td>
<td>16SrVI-A</td>
<td>“Ca. P. trifolii”</td>
<td>USA</td>
</tr>
<tr>
<td>Potato purple top wilt</td>
<td>16SrXVIII-A</td>
<td>“Ca. P. americanum”</td>
<td>USA</td>
</tr>
</tbody>
</table>
planting material and the disease it elicits may result in extensive damage and losses (40-100% in USA and Brazil). Control measures to be implemented are: (i) use of healthy tested seed (seed treatment has not been effective up to now); (ii) seed test by a check on 10,000 seedlings/lot in a greenhouse (so-called sweatbox method); (iii) inspection of plants during the growing season and destruction of infected plant material. In recent years also PCR screening tests have been developed (Bahar et al., 2008; Jing et al., 2011; Woudt et al., 2012). Positive results of all these methods still need to be verified by isolation of the pathogen. Perhaps RT-PCR in combination with mass spectrometry (matrix assisted laser desorption ionization time-of-flight mass spectrometry) could be used to exclude false positives (Wang et al., 2012).

A new strain of the potato stem rot bacterium belonging to the genus Dickeya, provisionally named ‘D. solani’. Bacterial stem rot of potato, caused by different ‘cold tolerant’ biovars of Erwinia chrysanthemi, now named Dickeya dianthicola and D. chrysanthemi, has been reported from many European countries since the 1970s, and is regarded as a quality disease (Janse and Ruissen, 1988; Toth et al., 2011). A taxonomic revision placed Erwinia chrysanthemi biovars into six species of the newly created genus Dickeya, namely D. chrysanthemi biovar chrysanthemi and biovar parthenii, D. dadanti, D. dianthicola, D. dieffenbachiae, D. parasidica and D. zeae (Samson et al., 2005). Among them there are ‘warm tolerant’ species like D. dadanti and D. zeae that were found not only in potato in hot climates, but also in many ornamental plants, often grown in greenhouse in temperate regions (Janse and Ruissen, 1988; Janse and Scheepens, 1990; Samson et al., 1990). Recently a new ‘warm tolerant’ variant of Dickeya was observed, first in the Netherlands since 2000 (Czajkowski, 2009a, 2009b, 2011; J. Van Vaerenbergh, personal communication), then in Poland, Belgium, Finland, France, Israel and most recently also in the UK (2010), Denmark and Sweden (2011). This variant is virulent under warm climatic conditions and is closely related, but not similar to D. dadanti. It has been provisionally denoted D. solani (Czajkowski, 2011). A recent taxonomic study indicates that it is not certain whether this variant is indeed a new species (Van Vaerenbergh et al., 2012). Warm tolerant Dickeya spp. have been isolated from surface waters and D. solani, that shows a strong clonal character, in the Netherlands also from hyacinth and iris in the bulb production area where no potatoes are grown (Toth et al., 2011; Van Vaerenbergh et al., 2012). A theory is that a warm tolerant species (D. dadanti or zeae) escaped from greenhouse wastewater disposals to surface water and mutated to a form that clonally spread in hyacinth, iris and potato. As with the other Dickeya and Pectobacterium species in potato, effective control is possible and regulated via certification schemes. It should be based on the use of tested, healthy seed and careful cultural practices (careful and dry harvesting, proper storage and ventilation, avoiding of desprouting and cutting seed, hygiene on the farm, etc).

**Stem rot and leaf spot of maize and ‘center rot’ of onion - Pantoea ananatis.** Pantoea ananatis can cause a number of diseases on different hosts, e.g. stem rot and leaf spot of maize, internal fruit rot of melon and pink disease of pineapple. It also infects onion (Allium cepa) inducing ‘center rot’. Outbreaks of which have been reported since 1997 in the USA, and was isolated from onion seed in South Africa (Gosczynska, 2006). Seed transmission was confirmed in the USA (Walcott et al., 2002). The thrips Frankiella fusca can transmit the bacterium which apparently survives as a saprophyte on many weed and crop hosts (Gitaitis et al., 2003).

**Stewart’s disease or bacterial wilt - Pantoea stewartii subsp. stewartii.** This vascular disease, characterized by white stripes on the host leaves, is widespread in North America. The main host is maize, especially sugar maize, but also the so-called ‘dent’, ‘flint’, ‘flour’ and popcorn types. The bacterium is mainly spread by Chaetocnema pulicaria, the corn flea beetle. Ps. subsp. stewartii and bacterial wilt have been reported from, but are not established in Austria, Greece, Italy, Poland, Romania and European Russia. Main source of introduction is contaminated seed from North America, but pathogen and disease disappear apparently some years after introduction, due to absence of the vector beetle in Europe. Local vectors do not transmit the bacterium as far as is known. In Italy, substantial damage was reported in the years 1940-1950, and some reoccurrence, but not very damaging in 1983-1984 (Mazzucchi, 1984; www.eppo.org/QUARANTINE/bacteria/Pantoea_stewartii/ERWIST_ds.pdf). Further introductions could occur in the future, but will remain relatively harmless as long as the vector is not introduced or local flea beetles, as potential vectors, do not acquire and transmit the pathogen.

**New outbreaks of bacterial (blight) canker of kiwifruit - Pseudomonas syringae pv. actinidiae.** Bacterial canker of kiwifruit was first observed in Japan in 1984 (Takikawa et al., 1989). It was also reported from China where, as we know now, it occurred already as early as 1984/1985 (Liang et al., 2000). Subsequently it was found in Korea and Italy (1992). Hosts are green kiwi (Actinidia deliciosa), yellow kiwi (Actinidia chinensis), A. arguta, and A. kolomikta. Since 2008 bacterial canker outbreaks have repeatedly been observed in Italy (Emilia-Romagna, Lazio, Piemonte and Veneto) on green kiwi, causing a mild leaf spot and some canker formation on branches and trunk. There was also a re-
port from Iran in 1994 and a recent occurrence of the so-called Asian or mild strain in Australia (EPPO Reporting Service 2011/130). In spring and autumn of 2008 and winter 2008/9, however, severe outbreaks occurred on yellow kiwi characterized by wilting, necrosis, severe leaf spot and canker formation (Fig. 6A-C), especially the cvs Hort 16A and Jin Tao cultivated in central Italy (Latina province). Recently, severe outbreaks have also occurred on green kiwi cv. Hayward (Ferrante and Scortichini 2010) and the disease has spread to Calabria, Campania, and Friuli-Venezia Giulia regions in 2011 (EPPO Reporting Service 2011/131). P. s. pv. actinidiae was recently observed in yellow and green kiwi in France [severe form found in the Rhône-Alp area in 2010 (EPPO Reporting Service 2012/002)], Spain (Abelleira et al., 2011; Balestra et al., 2011), Portugal (Balestra et al., 2010), Switzerland (2011), Chile (2011) (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm) and Turkey, where it is present since 2009 in the Black Sea area (Bastas and Karakaya, 2012). Interestingly, the severe form is also known to occur since 2010 in New Zealand and has spread on the North and South Island (Everett et al., 2011; Young, 2012). In this country another mild strain causing only leaf spots has also been observed, which may be not P. s. pv. actinidiae (Vanneste et al., 2010).

The Asian mild strain was recently reported to be present in Australia (EPPO Reporting Service 2011/130). A PCR test for identification and epidemiological studies was developed by Vanneste et al. (2010). Epidemics of bacterial blight of kiwi occur usually after frost damage. In Japan and Korea bacterial blight is the most limiting

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**Fig. 6.** A-C. Symptoms induced by an aggressive strain of *Psudomonas syringae* pv. *actinidiae* on yellow kiwifruit (*Actinidia chinensis*) in Italy. Wilting and necrosis (A), necrotic leaf spots (B), canker with a reddish ooze (C) (Courtesy M. Scortichini). D-F: Symptoms induced by *Psudomonas syringae* pv. *aesculi* on Horse chestnut (*Aesculus hippocastanum*). Severe wilting and yellowing (D), red ooze from a canker (E), extensive cankers on a branch (F) (Courtesy Aesculaap, NL). G: Leaf spots with a yellow halo and ‘shotholes’ on cherry laurel (*Prunus laurocerasus*) caused by *Xanthomonas arboricola* pv. *pruni* (Source Naktuinbouw, NL).
factor to kiwi crops, although mild strains appear to occur there. From Italy in recent years more than 2 million euro damage was reported. In a recent taxonomic study, with extensive sequence analysis of the diverse strains, Mazzaglia et al. (2012) came to the conclusion that severe strains from Europe and New Zealand indeed differ from the mild strains that occur in Japan, Korea and were also present in Italy in the past, and that are similar to strains from China, the country where the bacterium probably originates. They also showed that New Zealand and European strains differ to a small extent and both are more close to the Chinese strains, implicating a possible origin of these strains in China, probably imported with planting material from that country. Due to the emerging character of the new severe strains of \textit{P. syringae pv. actinidiae}, EPPO has placed it on its alert list (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm).

An emerging disease in forestry: Bleeding canker of horse chestnut - \textit{Pseudomonas syringae pv. aesculi}. Since 2002 an emerging bacterial disease, named bleeding canker of horse chestnut (\textit{Aesculus} spp., especially \textit{A. hippocastanum}) was observed first in the Netherlands, then in Belgium, France, Germany and the UK. It is thought that the disease had longer been present in the countries concerned, with an incidence of 40-60\%, but that symptoms were often mistaken for those of fungal diseases. Progress of the disease was rapid in the countries concerned, with an incidence of 40-60\%, but that symptoms were often mistaken for those of fungal diseases. Progress of the disease was rapid in the early 2000 but the epidemic in the Netherlands seems to have slowed down considerably in recent years (author’s personal observation). The pathogen appears to be a mutant of a bacterium from \textit{Aesculus} earlier described in India as \textit{Pseudomonas syringae pv. aesculi} (Durgapal and Sing, 1980). The bacterium shows a clonal character and adaptation to horse chestnut (Green et al., 2010, 2012). Symptoms are cracks with reddish-brown exudate on trunk and branches, and extensive necrotic cankers in phloem, where death of cambium leads to dieback (Fig. 6D-F). Insect transmission cannot be excluded and flower infection has been observed. Trees aged 10 to 30 years can be killed in a few years time (van Beuningen et al., 2009). It was determined that \textit{P. pv. aesculi} does not survive exposure to 39°C for 24 h and initial experiments treating young trees for 48 h at this temperature were promising (Keijzer et al., 2012). See also: Bultreys et al. (2008); Janse et al. (2006); Schmidt et al. (2008); Webber et al. (2008).

Spreading of bacterial canker and leaf spots of stone fruits - \textit{Xanthomonas arboricola pv. prunii}. Recent outbreaks of bacterial canker induced by the quarantine pathogen \textit{Xanthomonas arboricola pv. prunii} on peach, nectarine (\textit{P. persica var. nectarina}), apricot, cherry and/or plum have been reported from Slovenia (1994), France (1995), Spain (1999) and Iran (2005). The disease is established in Austria, Bulgaria, Italy, Moldova, Montenegro, Russian Federation and Ukraine. On peach, small cankers on twigs and leaf spots are formed; on plum and apricot, holdover cankers on trunk and larger branches are the main problem. On cherry, fruit infection is most damaging (Roselló et al., 2012; Stefani, 2010). \textit{X.a. pv. prunii} would be a risk for north-west Europe should climate change towards higher temperatures continue. The bacterium was identified from plum planting material originating from Asia in 1994 and spread to peach, with an outbreak in 1994 and further ones in later years (Seljak et al., 2001). In France, a severe outbreak took place in 2000 on peach and nectarine (EPPO reporting service 2006/235). \textit{X.a. pv. prunii} was reported from Hungary in propagating material of plum (2004), then in an apricot orchard (Nemeth, 2007). In Italy severe outbreaks occurred in the 1990s on Japanese plum (\textit{P. salicina}) and nectarine, after introduction of the very susceptible cv. Calita. A first report of \textit{X.a. pv. prunii} infection to \textit{Prunus laurocerasus} in a nursery in Toscany was also from Italy in 2005. In Switzerland, the first record was in an apricot orchard in 2005 and in two Japanese plum orchards in 2009, near Martigny (Pothier et al., 2009). In the Netherlands, a first outbreak in \textit{P. laurocerasus} occurred in 2008, in the west of the country, where plants showed shot-hole symptoms in the leaves (Fig. 6G) (EPPO Reporting Service 2009/178). Infections were again identified in different nurseries in 2009-2011 (Bergsma-Vlam et al., 2012). In Spain the bacterium was found on peach in 1999, further outbreaks in almond in 2006 and 2009 in Valencia and Aragon (Palacio-Bielsa et al., 2010, Roselló et al., 2012). Temperatures of 15-28°C, heavy rain and wind in springtime stimulate epidemics. The economic impact of \textit{X.a. pv. prunii} consists of reduced quality and marketability of fruits and reduced productivity of the trees, as well as higher production costs.

Bacterial leaf spot of poinsettia - \textit{Xanthomonas axonopodis pv. poinsettii}. This bacterium was originally found in poinsettia (\textit{Euphorbia pulcherrima}) in India (Patel et al., 1951), then reported from Florida (USA) (Chase, 1985). Other Euphorbiaceae are susceptible, such as \textit{E. heterophylla} (wild poinsettia, mainly occurring in North America, but now widespread in Italy), \textit{E. mili} (crown-of-thorns), \textit{Codiaeum variegatum} (croton) and \textit{Manihot esculenta} (cassava) (Chase, 1985; CABI, 2011). \textit{E. pulcherrima} originates from Mexico and is a very popular Christmas pot plant in European countries, on which \textit{X.a. pv. poinsettii} causes brown to black leaf spots, sometimes surrounded by a yellow halo. Over time the spots may coalesce and the leaves turn completely yellow and drop, severely impairing the commercial value of the plants (Wohanka, 2004). The disease occurs in Cocos Islands (territory of Australia), the Philippines (Quimio, 1974), Taiwan (Lee et al.,
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2006), China (Li et al., 2006b), Venezuela (Hernández and Trujillo, 1997), Australia and New Zealand (Hill, 1979) [see also EPPO (2006)]. In Europe the disease was first reported from Italy in 2003 (Stravato et al., 2004) and again in 2007, and from Germany also in 2003 (Wohanka, 2004). Further reports are from Austria in (2007, in one glasshouse, eradicated), Czech Republic (2007 in one glasshouse, eradicated), Netherlands (several findings since 2006/2007), UK (several findings since 2006), Slovenia (2009 in one glasshouse, eradicated) and Norway (2010 in 16 places of production, Perminow et al. (2011)). The ultimate source of this latter infection has not yet been identified. However, it is known that mother material in some of the infected countries was not only imported from other European countries, but also from South America and Africa. Identification methods have been described by Li et al. (2006b).

CONCLUDING REMARKS

A number of specific and general prevention and control measures for emerging diseases have been listed under Liberibacter spp. The following further measures can be considered important in the management of emerging bacterial diseases:

- Ornamental and wild hosts may play an important role in spreading diseases and maintaining the pathogen and its vectors in the environment. These plants should be included in surveys.

- Rapid and reliable diagnosis remains a key issue, as well as breeding for resistance. All pathogens mentioned are emerging threats, with real risks of introduction and in some cases closely approaching or already present in the Mediterranean basin and/or Europe.

- Introduction of susceptible wild hosts and susceptible cultivars of cultivated hosts must be avoided, as they often lead to introduction of the pathogen or to outbreaks due to a ‘jump’ of local pathogen strains to the very susceptible cultivar. Introduction of vectors should also be prevented as history teaches that the vector(s) appear first and the pathogen a few years later. Examples of these facts are presented in the description of the various disease-pathogen combinations.

- An efficient prevention and control strategy of diseases described in this review should be based on so-called pathway protection. This means it should be based on regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-regulated pests, where the following conditions should be maintained and controlled: (i) place of production should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

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OFFERED REVIEW

BACTERIAL DISEASES THAT MAY OR DO EMERGE, WITH (POSSIBLE) ECONOMIC DAMAGE FOR EUROPE AND THE MEDITERRANEAN BASIN: NOTES ON EPIDEMIOLOGY, RISKS, PREVENTION AND MANAGEMENT ON FIRST OCCURRENCE

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SUMMARY

Bacterial diseases are difficult to control (both chemically and biologically), and are restrained primarily by preventive measures. Most important risk factors for the introduction or spread of bacterial diseases in Europe and the Mediterranean basin are imported planting material and infected insect vectors. In this review the epidemiology, management and main risks of several emerging bacterial diseases approaching or already present in Europe, their causal organisms and vectors will be highlighted, especially: (a) Citrus huanglongbing (= Citrus greening), caused by the heat-tolerant “Candidatus Liberibacter asiaticus” and heat-sensitive “Candidatus L. africanus”. Both liberibacters and the respective psyllid vectors Diaphorina citri and Triozomyrma erytreae are present in the Arabian peninsula, with recent reports of huanglongbing occurring in Iran, Mali, Ethiopia and Somalia. T. erytreae is already present on some Atlantic Ocean islands; (b) leaf scorch and leaf scald diseases of grape and different fruit and ornamental trees, caused by Xylella fastidiosa. For this pathogen, although its presence has not been confirmed in Europe or the Mediterranean basin, local possible vectors such as Cicadella viridis and Philaenus spumarius occur; (c) citrus canker caused by Xanthomonas citri pv. citri, the most severe form of which, the so-called Asiatic, is already present in Iraq, Iran, Oman, Somalia, United Arab Emirates (UAE), Saudi Arabia, Yemen and Reunion. Outbreaks and/or risk and (possible) emerging character of some other bacterial pathogens not yet present in Europe (i-iv) or already present (v-xvi) are also highlighted: (i) black spot of mango, Xanthomonas citri pv. mangiferaeindicae, present in UAE and Reunion; (ii) bacterial blight of pomegranate, X. axonopodis pv. punicae, emerging in India; (iii) bacterial blight of guava, Erwinia psidii, emerging in Brazil; (iv) bacterial spot of passion fruit, X. campestris pv. passiflorae, emerging in Brazil; (v) stem rot and leaf spot of maize and center rot of onion, Pantoceana ananatis, an emerging problem for onion in the USA, isolated from seed in South Africa; (vi) almond witches’ broom, “Candidatus Phytoplasma phoenicium” killing thousands of trees in Lebanon and Iran; (vii) potato stolbur, “Candidatus Phytoplasma solani”, spreading from Eastern Europe westwards; (viii) zebra chip disease of potato and yellows of carrot caused by “Candidatus Liberibacter solanacearum” synonym Ca. Liberibacter psylllourous). The potato strains occur only in North and Central America and New Zealand, but the carrot pathogen has been reported from several Scandinavian countries and Spain with the respective psyllid vectors Bactericera cockerelli and Triozomyrma apicalis; (ix) an apparently ‘harmless’ “Candidatus Liberibacter europaeaus” found in the pear psyllid (Capopsylla pyri) in Italy; (x) bacterial fruit blotch of cucurbits, Acidovorax citrulli, seed-transmitted and the cause of outbreaks in Europe, Turkey and Israel; (xi) a new strain of the potato stem rot bacterium, provisionally named Dickeya solani, emerging in several north-western European countries and Israel; (xii) Stewart’s disease or bacterial wilt of maize, P. stewartii subsp. stewartii, spread by the corn flea beetle Chaetocnema pulicaria, observed in several European countries in which it has not become established due to the absence of vector; (xiii) renewed outbreaks from 2008 of Pseudomonas syringae pv. actinidiae, the agent of bacterial blight, especially on Actinidia chinensis (yellow kiwifruit) but also on A. delicosa in central Italy and, since 2010, in France; (xiv) bleeding canker of horse chestnut, P. syringae pv. aesculi, emerging in western Europe; (xv) bacterial canker of stone fruits caused by X. arboricola pv. pruni, with recent outbreaks in Switzerland, Spain (on almond) and in the Netherlands on cherry-laurel (Prunus laurocerasus); (xvi) bacterial leaf spot of poinsettia, X. axonopodis pv. poinsetticola observed in greenhouses in several north-western European countries. Ornamental and wild hosts may play an important role in spreading diseases and maintaining the pathogens and their vectors in the environment. These plants should be included in surveys. Rapid and reliable diagnosis remains a key issue, as well as breeding for resistance. All pathogens mentioned are emerging threats, with real risks of introduction and, in some cases, closely approaching or already present in the...
Mediterranean basin and/or Europe. Introduction of susceptible wild hosts and susceptible cultivars of cultivated hosts must be avoided, as they often lead to introduction of pathogens or to outbreaks due to a ‘jump’ of local pathogen strains to very susceptible cultivars. Introduction of vectors should also be prevented as history teaches that vectors appear first and the pathogens a few years later. Examples of these events are given in the description of the various disease-pathogen combinations. An efficient prevention and control strategy of the diseases mentioned in this review should be based on the so-called pathway protection, i.e. regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-regulated pests, where the following conditions should be enforced and controlled: (i) place of production should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

Key words: quarantine, risk assessment, geographical distribution, host plants, plant pathogens.

INTRODUCTION

Bacterial diseases are often a destructive and/or a major constraint for many crops. Substantial killing of the host and yield losses, often over 50% result from infestations caused by established pathogens such as Erwinia amylovora, Pseudomonas syringae pv. syringae and pv. morsprunorum, Agrobacterium tumefaciens (syn. Rhizobium tumefaciens) Xanthomonas arboricola pv. pruni, Candidatus Phytoplasma mali, Grapevine flavescence dorée phytoplasma, Grapevine bois noir phytoplasma, “Candidatus Phytoplasma pyri” and “Candidatus P. prunorum” (European stone fruit yellows). Quarantine pathogens sometimes spread out of contained loci, presenting a long-term threat to other EU countries (e.g. Xanthomonas arboricola pv. pruni spreading out of France and Italy) and others are an emerging threat outside Europe (Xylella fastidiosa, Candidatus Liberibacter spp. agents of citrus huanglongbing).

Bacterial diseases are difficult to control (both chemically and biologically) and are restrained primarily by preventive measures such as hygiene, use of healthy planting material, good cultural practices and avoidance of risky planting sites. Moreover, they are easily spread by (surface) water, planting material and contaminated agricultural implements/machines and by aspecific or specific insect vectors. Most important risk factors for the introduction of bacterial diseases into Europe are imported infected planting material and naturally spreading insect vectors. Therefore, early detection and correct identification/diagnosis are of utmost importance. In recent reviews, current classic and molecular methods for detection and identification of bacterial pathogens of fruit trees and nuts, including Xylella fastidiosa, have been described (Janse, 2010; Janse and Obradovic, 2010; Janse et al., 2012). In this contribution the epidemiology and main risks in the framework of prevention and management in case of first occurrence of some emerging bacterial diseases approaching the Mediterranean basin and Europe, their causal organisms and vectors, will be highlighted, with special reference to: (i) Citrus huanglongbing (HLB, formerly called Citrus greening), caused by the heat tolerant “Candidatus Liberibacter asiaticus” and the heat sensitive “Candidatus L. africanus”. Both liberibacters and the respective psyllid vectors Diaphorina citri and Triozoa erytreae are present in the Arabian peninsula, with recent reports of huanglongbing occurring in Iran, Mali, Ethiopia and Somalia and T. erytreae already present on some Atlantic Ocean islands (for references see under Geographical distribution of the specific pathogens later in the text). Furthermore, in less detail: (ii) leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by Xylella fastidiosa. For this pathogen, although its presence in Europe and the Mediterranean basin has not been confirmed, local possible vectors such as Cricadella viridis and Philaenus spumarius occur; (iii) Citrus canker, caused by Xanthomonas citri pv. citri (syn. X. axonopodis pv. citri, X. citri subsp. citri), the most severe form of which, the so-called Asiatic, is already present in Iraq, Iran, Oman, Somalia, UAE, Saudi Arabia, Yemen and Reunion (for references see under Geographical distribution of the specific pathogens later in the text).

Outbreaks and the possible emerging character of some other bacterial pathogens will be dealt with (e.g. Xanthomonas citri pv. mangiferaeindicae approaching the Mediterranean basin like X. c. pv. citri and the devastating “Candidatus Phytoplasma phoenicium” that occurs on almond in Lebanon). Furthermore, some diseases already present in the area which show an emerging character, such as the bacterial canker of kiwifruit (Actinidia spp.) caused by Pseudomonas syringae pv. actinidiae in Italy and France in the last three years, will also be addressed.

Since initial management and risk avoiding measures following an introduction are more or less the same for most of the above-mentioned pathogens, they will be detailed for HLB. It will be argued that the diseases addressed in this presentation are emerging threats, with real risks of introduction and economic damage and in some cases closely approaching the Mediterranean basin. The aim of this contribution is to create awareness of the risks of these diseases, thus enabling prevention, early detection and proper actions once introduction has occurred.
MAIN EMERGING BACTERIAL DISEASES

Citrus huanglongbing caused by “Candidatus Liberibacter” species. General. Huanglongbing or HLB (Chinese for yellow shoot disease) of citrus is caused by a non-culturable, fastidious, phloem-inhabiting, Gram-negative bacterium belonging to the α-Proteobacteria and to the genus “Candidatus Liberibacter” [originally the genus was named Liberobacter (Bové, 2006)]. This disease (Fig. 1A-C) was formally known as Citrus greening yellow dragon disease, mottle leaf disease, ‘likubin’ or vein phloem degeneration. It is one of the most destructive diseases of cultivated citrus for which no effective control is available with the consequence that citrus production declines in all areas where the disease occurs. Lin (1956) determined that this disease is graft-transmissible and described it for the first time in China. The origin of huanglongbing is not clear, probably Asia (India or China) or Africa (Gottwald et al., 2007). For a thorough general review on the disease and its causal organism see Bové (2006) and for a review on its epidemiology see Gottwald (2010).

Three different Liberibacter species causing Huanglongbing have been described based only on 16S rRNA and whole genome sequences information, because these organisms cannot be cultivated in vitro:

1. “Candidatus Liberibacter asiaticus” (Las), originally described as Liberobacter asiaticum, the most aggressive species, is heat-tolerant and stands temperatures above 30°C. The disease caused by this species is found mainly in lowlands where it is transmitted by the heat-tolerant psyllid Diaphorina citri (Kuwayama (Fig. 1D,E) [a recent report mentions the black psyllid, Diaphorina communis, as a vector in Bhutan (Donovan et al., 2011)]. Las is widespread in Asia, the Arabian peninsula, Mauritius and Reunion islands and, since 2004, in Brazil, Sao Paulo state (in Brazil some 10% of the infected plants are infected by this Liberibacter species, others are infected by the third species, Ca. L. africanus (Teixeira et al., 2005)). It occurs since 2005 in Florida (USA) (Hilbert, 2005).

2. “Candidatus Liberibacter africanus” (Laf), described as Liberobacter africanus, less aggressive and heat-sensitive. The disease induced by Laf is suppressed after exposure to temperatures above 30°C, therefore it occurs in the tropics at elevations higher than 700 metres above sea level. Laf is widespread in Africa (Somalia, Ethiopia and Cameroon) and occurs also in Reunion, Mauritius, and Yemen. Its vector is the heat-sensitive psyllid Trioza erytreae Del. Guercio.

3. “Candidatus L. americanus” (Lam), closely related to Las but heat-sensitive. It occurs in Brazil and is transmitted by the psyllid D. citri.

A recently described subspecies, “Candidatus Liberibacter africanus” subsp. capensis, was isolated from a symptomless ornamental rutaceous tree (Calodendrum capense) in the Western Cape province (South Africa). In the article describing this subspecies, the genus name Liberobacter was changed for all species into Liberibacter (Garnier et al., 2000). Ca. L. africanus subsp. capensis appears to be widespread in C. capense in South Africa, but is not transmitted to citrus (Phahladira et al., 2012) The complete genome sequence of Las is available (Duan et al., 2009) and, recently, differences between the Asiatic (China) and North American strains of Las were reported (Chen et al., 2010). Liberibacter species are apparently not seed-transmissible (Hartung et al., 2010; Hilf, 2011).

Some biological traits of the psyllid vectors. Both Diaphorina citri and Trioza erytreae feed on the phloem and are experimentally able to transmit both the Asian and African HLB. Fourth and fifth instar nymphs and adults can acquire and transmit the bacteria during their whole life, after a latent period of ca. 10 days, after feeding times varying from 15 to 20 min for D. citri to 24 h for T. erytreae. Multiplication in the vector has not clearly been demonstrated, the bacteria, however, can be found in large amounts in the haemolymph and salivary glands. For T. erytreae there is evidence that transovarial transmission occurs. Other psyllids that thrive on citrus were not found to transmit the bacteria, except for the black psyllid (Diaphorina communis) which is a Las vector in Bhutan (Donovan et al., 2011). Psyllids like to feed on new vegetation flushes the presence of which constitutes a risk for transmission. Although D. citri does not tolerate frost very well, it survived frosty days of up to -5°C in Florida (Bové, 2006; Bralinsky and Rogers, 2007, 2008; Gottwald, 2010; Gottwald et al., 2007; Halbert and Manjunath, 2004; Manjunath et al., 2008).

Geographical distribution of bacteria and vectors. Main sources: Anonymous (2007); EPPO/CABI (1997a, 1997b, 1998a, 1998b); CABI (2011); Halbert and Manjunath (2004) and http://www.aphis.usda.gov/plant_health/plant_pest_info/citrus_greening/background.shtml (i) Bacteria. Las (transmitted by D. citri in all countries mentioned). South east Asia: Cambodia, China (including Hong Kong), Indonesia, spreading along southern islands of Japan (Shinohara et al., 2006), Laos, Malaysia, Myanmar, Philippines, Taiwan, east Timor, Thailand, and Vietnam; Indian subcontinent: Bangladesh, Bhutan, India, Nepal, and Pakistan; Western Asia: Iran in Sistan-Baluchistan and Hormozgan provinces (Faghihi et al., 2009; Salehi et al., 2012); Indian Ocean: Comoros Islands, Madagascar, Mauritius, Re-
union and Sri Lanka; Arabian peninsula: Saudi Arabia, Yemen in the south west along the Red Sea; Africa: Northern Ethiopia (De Bac et al., 2010); South America: Brazil, mainly Sao Paulo state, since 2004; Caribbean: Cuba (Martinez et al., 2009), Dominican Republic (Matos et al., 2009), Belize (Manjunath et al., 2010); North America: USA [Florida since 2005; Louisiana, 2008; Georgia and South Carolina, 2009; California 2012]; Mexico, 2009. (http://www.pestalert.org/oprDetail.cfm?oprID=382; http://www.pestalert.org/oprDetail.cfm?oprID=516, http://www.pestalert.org/oprDetail.cfm?oprID=401)


Laf and Las. Ethiopia, Mauritius, Reunion, Saudi Arabia and Yemen host both vectors (D. citri and T. erytreae) and both pathogens.

Lam. Brazil. Lam has also been reported (but not confirmed) from one of 97 citrus leaf samples from eight provinces of southern China (Lou et al., 2008).

(ii) Vectors. D. citri (EPPO, 2005c). South-east Asia: Cambodia, China (including Hong Kong), Indonesia, southern islands of Japan, Macau, Malaysia, Myanmar, Philippines, Taiwan, Thailand, and Vietnam; Indian sub-continent: Afghanistan, Bangladesh, Bhutan, India, Nepal, and Pakistan; Indian Ocean: Comoros Islands, Madagascar, Mauritius, Reunion and Sri Lanka; Arabian peninsula: Saudi Arabia (Wooler et al., 1974), Yemen, Oman; South America: Argentina [since 1984 in northeast, since 2006 in north-west, Ramallo et al. (2008)], Brazil (since the 1940’s), Venezuela; Central America: Honduras, Belize, Costa Rica; Caribbean: Cuba (1999), Haiti (2000), Guadeloupe (1998) (Étienne et al., 1998), Bahamas (1999), Cayman Islands (2000), Virgin Islands and Dominican Republic (2001), Puerto Rico (2002) (Halbert and Núñez, 2004); North America: USA Florida (1998), Alabama, Georgia, Mississippi, South Carolina, Louisiana, California (USDA 2010), Texas (2001) (French et al., 2001); Mexico (2009); Pacific Ocean: Hawaii, Maui (Conant et al., 2007)

T. erytreae. Africa: Burundi, Cameroon, Central African Republic, Ethiopia, Kenya, Malawi, Nigeria, Rwanda, Somalia, South Africa, Sudan, Swaziland,
Liberibacter species. Rutaceae: Most Citrus species and forms are or can be host of Liberibacters. Sweet oranges, mandarins and tangelos (C. reticulata x C. paradisi) are generally the most susceptible showing severe symptoms. Lemons, grapefruits, C. limonia, C. limettioides, rough lemons, kumquats (Fortunella spp.) and citrons are less severely affected. Symptoms are mild on limes and pummelos (C. grandis). However, in Brazil and the USA all commercial Citrus species have a very similar susceptibility. Other confirmed hosts are Limo-
nia acidissima, Murraya paniculata or orange jasmine (often used as ornamental and for hedges) and the related or similar M. exotica, M. (Bergera) koenigii, Severinia buxifolia and Vepris lanceolata (= V. undulata = Todella lanceolata)

(i) Vectors (D. citri and T. erytreae). Rutaceae, all cultivated and wild species listed above and Clausena anisata (= C. inaequalis) and Zanthoxylum capense (= Fagara capensis).

Symptomatology. Symptoms (Fig. 1A–C) may be confused with those of other diseases and disorders. More typically, the first symptoms are one or more yellowing shoots. Leaves are asymmetrical when the two leaf-halves are compared and exhibit well-defined yellow areas, called blotchy motting, that may also occur on fruits. With time, yellow spots may intensify, resembling very much zinc deficiency. Veins may become corky, giving the leaves a thicker appearance. Fruits are often smaller, asymmetrically misshaped (lopsided) and show persistent green areas, especially at the stylar end. When infected fruits are cut, yellow-brown vascular bundles and necrotic seeds may be observed. The final stages of the diseases are characterized by severe leaf and fruit drop, twig and stem die-back and death of the trees (especially when infected by Las). For photos of symptoms and psyllids, see http://anrcatalog.ucdavis.edu/ pdf/8205.pdf; http://entnemdept.ufl.edu/creatures/citrus/acpsyllid.htm; http://www.forestryimages.org/browse/subthumb.cfm?sub=4695&start=8; http://www.eppo.org.

Detection and diagnosis. In the early years detection and diagnosis was mainly based on symptoms, electron microscopy and biological indexing. A monoclonal antiserum was developed but proved too specific. PCR (classical, nested, real-time, qualitative real-time and multiplex) is now the main confirmatory test and is rou-
tinely used in many areas, allowing also detection in insects and of latent infections in plants (Benyon et al., 2008a; Li et al., 2006a, 2007; Teixeira et al., 2008). Okuda et al. (2005) have developed a species-specific multiplex TaqMan (real-time) PCR for Las, Laf and Lam (with COX primers/probe for the host plant cytochrome oxidase gene as internal control). For a very sensitive combination of nested PCR and TaqMan (real-time) PCR in a single-tube test see Lin et al. (2010) and a real-time PCR protocol based on prophage genes, that is useful for world-wide detection, also in difficult hosts, see Morgan et al. (2012).

Epidemiology. Main sources: Bové (2006); Gottwald (2010); Gottwald et al., (2007). HLB epidemics develop rather slowly in time (several to more than 12 years), but the disease may spread quite rapidly in a grove (in several years trees may show severe symptoms and within 7-10 years orchards may become unproductive, depending on the age of the trees and the presence and number of vectors). A complicating factor is that many trees may have been already latently infected for a considerable time before symptoms become obvious. The number of latent infected trees may be two- to manifold as compared to symptomatic trees, which hinders early detection of HLB and frustrates eradication campaigns. Spread of vectors and disease has a tree-to-tree pattern although also further spread (a few kilometres) from an infection focus may be possible. Psyllids migrate mostly when host plants are flushing. Long distance dispersal is by infected planting material or by infected psyllids accidently transported by man on plant material or otherwise. The presence of HLB in a tree may be sectorial, thus the bacteria may not be detectable in the symptomless part. How far psyllids can be carried by high air streams and spread over long distances is still unknown, but its occurrence cannot be excluded. Seed transmission of virulent bacteria has not yet been demonstrated.

Damage and losses. Since resistance against HLB is a dangerous and devastating disease and, as yet, no sources of resistance have been found or created. Yield is decreased, mainly because of reduced growth and fruit drop, and the fruit quality impaired. Yield losses may be 30-100% and, within 7-10 years from planting, the groves may lose productivity completely. It has been calculated that ca. 100 million trees have been killed in south-east Asia, India, Arabian peninsula, and South Africa, leading to decline of the citrus industry in these areas. In southwestern Saudi Arabia, sweet orange and mandarin have practically disappeared already during the 1970s. Since 2004, ca. 1 million trees have been destroyed in Brazil due to HLB infection. In Florida HLB was detected for the first time in 2005 and by 2009 it has spread to most citrus-growing areas, so that eradication efforts were given up (Bové, 2006; Gottwald, 2010) and the select agent status for all three Liberibacter species by USDA APHIS abandoned.
Main risk factors. HLB and its vectors have not yet been reported from the European mainland or the Mediterranean basin, where there are no local psyllid vectors known. Long distance spread of Liberibacter spp. and vectors is possible and will mainly be with planting material. In Kenya, infected breeding material from abroad was suspected to be the source of initial infection (Magomere et al., 2009). Note that HLB is approaching the Mediterranean basin mainly from three sides: (i) Saudi Arabia, where both Laf and Las are present and could move further north (e.g. also with people travelling to and from Mecca), possibly also for Las and its vector should global warming continue. Note that Las and its vector could also establish in subtropical Florida; (ii) Africa, where Laf and its vector are already present in Ethiopia and Somalia and Las is established in north Ethiopia; (iii) Iran, where Las has been reported in 2009. Note that D. citri was present in Florida since 1998 and HLB followed in 2004 (also see Bové, 2006), and that the Laf vector is already present in the Atlantic, i.e. the Canary, Madeira, Porto Santos and St. Helena islands. Murraya paniculata (orange jasmine), a popular landscape plant, played an important role in spread of HLB and D. citri in Florida (and probably also to other states), also via garden centres (Manjunath et al., 2008). Murraya species of Asian origin have been marketed (also in bonsai form) in Europe for quite some years. Infections may go unnoticed for several years after they became established due to latency or overlooking of the early symptoms. HLB is suspected to have been present several years before its official detection in Florida in 2005 (Gottwald et al., 2007; Gottwald, 2010).

Prevention and management following first introduction. Once established, HLB bacteria and vectors are very difficult to control. Main strategies are geographical isolation and certification/indexing programs for budwood sources and nursery production in insect-proof greenhouses, chemical and/or biological vector control and intensive surveying (by visual inspection and laboratory testing) with removal and destruction of infected trees (both visually and latently infected trees) wherever possible. Healthy budwood can be obtained by shoot-tip grafting, or alternatively by heat treatment, e.g. water-saturated hot air at 49°C for 50 min and fumigation of budwood against HLB vectors is possible (EPPO, 1988). The visual inspections in the groves should be very intensive and performed from moving inspection platforms. Chemical sprays, for vector control, to be effective, should also be performed frequently. Antibiotic treatment has been abandoned completely as it was not effective and is not human and environmentally friendly. Biological control was effective in Reunion Island, with hymenopterous ectoparasites Tamarixia dryi for Laf and T. radiae for Las, probably because hyperparasites of the parasite were absent. At present the only successful prevention/control methods appears to be the one followed in São Paulo state in Brazil, following the introduction of huanglongbing in 2004. The control program that was immediately installed in 2004 by responsible organisations, became very successful after eight years, when disease incidence on more than 200,000 ha was reduced to less than 1%. This was because: (i) at the beginning of the program the disease incidence was still low (less than 8%) and (ii) it could be executed on large, easy to handle farms (>500 ha). The basic principles of the so-called Three-Pronged System (TPS) are: (i) intensive vector control, (ii) reduction of inoculum by tracing, identifying and removing infected trees, and (iii) production of healthy nursery stock in covered, insect proof nurseries and their use for re-planting (Bové, 2012; Belasque et al., 2010).

Genetic resistance development has started, but is still in its infancy (Grosser et al., 2008). Still, breeding for resistance will remain one of the most important ways to control this disease and those described in the following paragraphs.

HLB bacteria and their vectors should be prevented from entering the Mediterranean basin by strict inspection and laboratory testing of imported breeding material from risk areas. In the European Union the importation of citrus planting material from third (non-EU) countries is already prohibited (Annex III A of Directive 2000/29/EC) and EPPO (EPPO, 1988) and the introduction of plants for planting and cut citrus branches from countries where HLB or its vectors occur should also be prohibited. However, when such material is imported, it should be fumigated and quarantined for at least two years. Since Liberibacter spp., D. citri and T. erythreae are quarantine organisms included in the EPPO A1 list and EC Annex II/1a list, the following additional measures can be enforced [see also Bassanezi et al., 2008; Bové, 2006; EPPO, 1988; Gottwald, 2010; USDA, 2010. For (pro-active) measures advised by the Australian government, see http://www.agnet.org/library/eb/607/]:

- Murraya spp. and ornamental citrus spp. already present in garden centres or in plantations should be surveyed, including vector sampling. In suspect areas this could also been done in (young) citrus orchards.
- Importations from risk areas of these plants strictly inspected and laboratory-tested.
- Proactive training of diagnosticians, surveyors, nursery managers and producers.
- Production in citrus nurseries should be in secure, insect-proof greenhouses.
- Once an introduction has occurred, infected trees should be destroyed and vectors controlled by chemical insecticides.
- When executing severe eradication programs, governments should consider compensation or facilitate insurance to secure full cooperation also from small growers and private tree-owners.
Further training of all parties involved and development of a public information program.

Leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by *Xylella fastidiosa*.

**General.** The bacterium *Xylella fastidiosa* is a xylem-inhabiting, vector-transmitted, very slow growing, Gram-negative bacterium. It was cultured and described for the first time in 1987 in the USA as the cause of Pierce’s disease (PD) of grapevine (disease observed already in 1884) and as the cause of phony peach disease (PPD) in peach, *Prunus persica* (disease observed in 1890 in the USA). In 1993 *X. fastidiosa* was identified as the cause of citrus variegated chlorosis (CVC) or citrus X disease in Brazil. The bacterium also causes a number of so-called leaf scorch diseases to *Prunus* spp. (including almond leaf scorch or ALS in *P. amygdalus* and plum leaf scald or PLS in *P. domestica*), *Acer* spp., *Carya illinoensis* (pecan), *Coffea arabica* (CLC, in Brazil isolated in 1995 and also pathogenic to citrus), *Hedera helix*, *Morus rubra*, *Nerium oleander* (OLS), *Platanus occidentalis* (Fig. 2A), *Quercus* spp. and *Ulmus americana*. *X. fastidiosa* is also the agent of alfalfa dwarf and of wilting of *Vinca major*. Many wild plants such as grasses, sedges and trees may carry the pathogen, often without showing symptoms. None of these diseases is apparently seed-borne. Seed transmission, however, has been reported in sweet orange, *C. sinensis* (Li et al., 2003). They occur mainly in tropical/subtropical areas, although leaf scorch diseases are present also in much colder climate, e.g. oak leaf scorch in eastern North America up to Canada.

Several pathogenic varieties of the bacterium have been described, that are often host-specific (e.g., the PD strain will not cause disease if introduced to peach or plum). The following subspecies have been described:

(i) *Xylella fastidiosa* subsp. *fastidiosa* (erroneously named *X. f. subsp. piercei*), PD and LSA, strains from cultivated grape, alfalfa, almond (two), and maple; (ii) *X. fastidiosa* subsp. *multiplex*, PPD and PLS, strains from peach, elm, plum, pigeon grape, sycamore, almond and recently pecan (Melanson et al., 2012); (iii) *X. fastidiosa* subsp. *pauca*, CVC, strains from citrus and probably those from coffee (CLC); (iv) *X. fastidiosa* subsp. *sandy*, strains from *Nerium oleander* (OLS); (v) *X. fastidiosa* subsp. *tashke*, strains from the ornamental tree *Chitalpa tashkentensis*.

*X. fastidiosa* isolates are genetically very similar, but studies on their biological traits have indicated differences in virulence and symptomatology. As mentioned, taxonomic analyses have identified several subspecies, and phylogenetic analyses of housekeeping genes have shown broad host-based genetic differences. However, results are still inconclusive for genetic differentiation of isolates within subspecies. In a recent study, sequences of nine non-housekeeping genes were used to study 54 *X. fastidiosa* isolates infecting different host plants. Strains could again be divided into the known *X. fastidiosa* subspecies, but also showed new within-subspecies differentiation, including geographic differentiation, and some host-based isolate variation and specificity (Parker et al., 2012).

*Xylella fastidiosa* is a quarantine organism on the EPPO A1 list and EC Annex II/Ia list. For further and extensive data on biology, hosts, geographical distribution and epidemiology see recent reviews (Purcell, 1997; Chatterjee et al., 2008; Janse, 2010; Janse and Obradovic, 2010) and the extensive *X. fastidiosa* website, edited and maintained by A. Purcell and R. Almeida: http://www.cnr.berkeley.edu/xylella/. For diagnostic methods, see EPPO 2005a and Janse et al. (2012).

**Hosts.** A full host list can be found on http://...
Symptoms and transmission. For symptoms on different hosts, see http://www.cnr.berkeley.edu/xylella/. In general, early symptoms are a slight chlorosis or bronzing along leaf margin or tip that intensifies and that may become water-soaked before browning and drying. These symptoms are first found on a few branches, then they extend to most or the whole canopy (so-called leaf scorch). A narrow chlorotic band that becomes especially clear in autumn delineates the affected area. A premature defoliation may take place with new malformed leaves are formed. Fruits can be abnormally shaped and stems may show internal and external discolorations, dieback and abnormal growth, leading to eventual death of the host. Vectors are mainly sharpshooters and froghoppers or spittlebugs (Cicadellidae) that lack a latent period, and have no transstadial or transovarial transmission of the bacterium. The pathogen shows persistence in the vector adults, and or transovarial transmission of the bacterium. The sharpshooters and froghoppers or spittlebugs (Cicadellidae) extend to most or the whole canopy (so-called leaf scorch). CVC vectors in Brazil are Acrogonia terminalis that lays eggs externally on the leaves, Dilobopterus costalimai and Oncometopia fascialis. Local possible vectors for Europe are Cicadella viridis and Philaenus spumarius (meadow spittlebug) (Fig. 2B). Because of the presence of these potential vectors the establishment of the pathogen in Europe is not strictly connected to introduction of foreign recognized vectors.

Risks. X. fastidiosa is an emerging threat in the south-west USA, mainly due to recent establishment of the glassy-winged sharpshooter (GWSS, H. vitripennis), providing much more efficient transmission than local vectors, and leading to very serious outbreaks of PD in grapevine, ALS and OLS. GWSS probably first entered California as eggs deposited in plant tissues. In Central and South America X. fastidiosa has become very noxious due to the rapid expansion (most likely via distribution of infected planting material) of CVC in Citrus. leading to more than a third of all trees in the area having symptoms of CVC, and CLC in coffee. As to Europe, there are only a few unconfirmed reports of the presence of X. fastidiosa in the grapevine in Kosovo (Berisha et al., 1998) and in almond in Turkey (Güldür et al., 2005). Since X. fastidiosa has more than 150 hosts and many of them, including Vitis, were are imported (often as planting material), risk of introduction (especially in latent form) must not be underestimated. Absence of the diseases caused by X. fastidiosa will mainly be due to the absence of suitable vectors. However, introduction of the pathogen and infected vectors with plant material cannot be excluded. Moreover, also local Cicadellidae (see above) could become potential vectors. Therefore, X. fastidiosa has the A1 quarantine status in the EPPO region and H. vitripennis that has a very large host range and feeds on almond, peach and plum was recently placed on the EPPO alert list. As in the more southern parts of the USA, European Vitis varieties are very susceptible to X. fastidiosa. This is really a risk if a vector that could survive the winters of southern Europe were introduced, the pathogen would become established in wild hosts (wild and domestic plums and wild cherry are symptomless reservoirs in the USA) and cause spring infections that are most likely to persist over the years. The same risk holds true for citrus (sweet oranges, mandarins, and tangerines) and other hosts, such as almond, plum and peach that are widely grown in southern Europe, especially in the warmer Mediterranean basin, where a disease-favourable combination of warm nights, regular rainfall/high humidity and long growing season, is present. The conclusion is that X. fastidiosa is a real threat for Europe, not only for Vitis and Citrus but also for stone fruits (almond, peach and plum) and oleander (GWSS likes to feed on oleander), that is difficult to prevent from entering and difficult to control once established, deserving more attention than up till now. Resistance in European grapes is scarce or even absent. Vector control proved not to be very effective in the USA. Cultural practices to keep plants in optimum condition are of importance, but not sufficient, and the use of avirulent strains for cross-protection is still in its infancy.

Citrus canker, caused by Xanthomonas citri pv. citri (X. axonopodis pv. citri). General. Asian citrus canker, a spot disease of Citrus spp. characterized by corky le-
sions on leaves, fruits and twigs, was first described by Stevens in 1914 and the causal bacterium, now named Xanthomonas citri pv. citri by Hasse in 1915 in the USA. However, citrus canker had been observed earlier in Asia as it originates most probably from China. Symptom formation and spread of the bacterium are enhanced by the activity of the citrus leaf miner, Phyllocnistis citrella, which occurs in nearly all citrus growing areas of the world. In Europe is is established in the Iberian peninsula, Corsica, Italy, Greece and Montenegro (http://www.bladmineerders.nl/minersf/lepidoptera/ramin/phyllocnistis/citrella/citrella.htm). This insect, however, is not a true vector. Grapefruit (C. paradisi), Mexican/Key lime (C. aurantifolia), lemon (C. limon) and C. brytrix are the most susceptible species X. citri pv. citri, is a Gram-negative bacterium with one polar flagellum, forming yellow colonies on agar media. Over the years, several much related diseases and pathogenic bacteria have been described from Citrus and some other Rutaceae spp. in Asia, North and South America:

(i) X. citri pv. citri (Xcc) (syn. X. citri subsp. citri, X. axonopodis pv. citri) formerly named group A or Asiatic strain is the most aggressive form. Other names for this pathogen have been X. citri, X. smithii subsp. citri and X. campestris pv. citri (A group) (Schaad et al., 2005, 2006; Ah-You et al., 2009). Strains with a different host range, named pathotype A* strains, infecting Mexican lime and Tahiti lime (C. latifolia) but not grapefruit (C. paradisi) have been reported from Cambodia, Iran, India, Oman and Saudi Arabia. The so-called Aw strains that infect Mexican lime and alemow (C. macrophylla) were described from Florida (Ngoc et al., 2009, 2010; Sun et al., 2004).

(ii) X. citri pv. aurantiifoli (syn. X. axonopodis pv. aurantiifoli, X. fuscans pv. aurantiifoli) formerly named X. citri group B or cancerxis, group C or Mexican lime cancerosis, and group D or Citrus bacteriosis, causes a relatively mild disease in Mexico and South America. It was also reported from Swingle citromelo rootstock (C. paradisi x Poncirus trifoliata) from Brazil (Jaciani et al., 2009).

(iii) X. citri pv. bilvae (syn. X. campestris pv. bilvae), is an ill-defined pathogen, causing shoot-hole disease and canker on Bael (Aegle marmelos), a member of the family Rutaceae described in India in 1953 (Ngoc et al., 2010).

(iv) X. alfalfa subsp. citrusmelonis (syn. X. campestris pv. citrusmelon), is the cause of citrus bacterial spot, formerly named Group E strains in Florida, USA (Schaad et al., 2005).

Only Xcc, the agent of Asian citrus canker (A, A* and Aw strains) is described here. It is a quarantine organism on the EPPO A1 list and EC Annex II/1a list. Main reference sources are Brunings and Gabriel (2003), Das (2003), Gottwald et al. (2002), Rogers et al. (2010), Lowe (2010) and, Ngoc et al., (2009), Golmohammadi et al. (2007) and EPPO (2005b) for diagnostic methods.

Geographical distribution Xcc originates from and is widespread in Asia, including Georgia, Iran, Iraq, Oman, Saudi Arabia, UAE and Yemen. Australia (eradicated), Argentina, Belau, Brazil, Caroline Islands, Cocos Islands, Comoros, Congo Democratic Republic, Ivory Coast, Fiji, Gabon, Madagascar, Mauritius, Mozambique (eradicated), Netherlands Antilles, New Zealand (eradicated), Micronesia, Palau, Papua New Guinea, Paraguay, Reunion, Seychelles, South Africa (eradicated), Uruguay, USA (CABI/EPPO, 2006). There are recent reports from Somalia (Balestra et al., 2008) Mali (Traoré et al., 2009) and Ethiopia (A* strains, Derсо et al., 2009).

Hosts. Cultivated hosts are Aegle marmelos (golden apple), Casimiroa edulis (white sapote), Citrus aurantiifolia (lime), C. aurantium (sour orange), C. brytrix (mauritius bitter orange), C. junos (yuzu), C. limetta (sweet lemon tree), C. limon (lemon), C. madurensis (calamondin), C. maxima (pummelo), C. medica (citron), C. natsudaidai (natsudaidai), C. reshni (Cleopatra mandarin), C. reticulata (mandarin), C. reticulata x Poncirus trifoliata (citrumelo), C. sinensis (navel orange), C. sunki (sour mandarin), C. tankan (tankan mandarin), Citrus unshiu (satssuma), Citrus x paradisi (grapefruit), Eremocitrus glauca (Australian desert lime), Limonia acidissima (elephant apple), Poncirus trifoliata (trifoliolate orange or Japanese bitter orange). Minor hosts are Fortunella japonica (round kumquat) and F. margarita (oval kumquat). Wild hosts are Ageratum coryzoides (billy goat weed), Severinia buxi-folia (box orange or boxthorn) and Swinglea glutinosa (= Aegle decandra, Limonia glutinosa).

Symptoms and transmission. (For pictures of symptoms and of the citrus leaf miner, P. citrella, see http://www.plantmanagementnetwork.org/pub/php/review/citruscanker/). Small spots, first visible on the upper leaf blade, appear on the leaves, shoots, twigs and fruits, to become raised pustules or blister-like eruptions (Fig. 3A). With time, the lesions increase size (up to 10 mm) and turn brown and necrotic with a depressed centre, and are sometimes surrounded by a yellow halo. On the fruits, the lesions can be mistaken for scale insects (e.g. the California red scale, Aonidiella auranti). The bacterium is a wound parasite and, as mentioned, the citrus leaf miner (P. citrella) contributes to disease spread and severity. Citrus canker is especially epidemic and damaging on seedlings and young trees, especially after storms (hurricanes) under warm weather conditions, but because of dependence on these weather conditions epidemics are sporadic. Full-grown trees show much less disease and damage (Goto, 1992). The bacterium can survive in a latent form in and on diseased shoots and discoloured bark tissue of the trunk, and may reoccur suddenly after several years (sometimes even as long as 10 years).

Risks and damage. Heavy losses were reported in epidemics, due to premature fruit drop and fruits with
spots that cannot be marketed or start rotting, thus must be destroyed. Furthermore, quarantine measures such as burning of trees and destruction of fruits may add to these losses (Goto, 1992). In severe cases, almost 100% of the fruits and leaves of young, susceptible trees may be infected and the plant growth is delayed for a number of years. The direct Government costs in the USA for the eradication activities from 1995 to 2006 were calculated to be more than $1.3 billion. From 2006 to 2009, together with costs for the control of Citrus huanglongbing the amount was $90 million. The citrus acreage in Florida decreased since 1996 by ca. 33% (Lowe, 2010). In January 2006, the USDA determined that canker had become so widespread in Florida that eradication was no longer feasible. Use of healthy planting material and use of other measures, including weather forecasting, in an integrated way have been applied in the control of Citrus canker with some success. Resistance has been found especially in *C. mitus* (calamondin) and *Fortunella* (kumquat). *C. reticulata* (mandarin) is tolerant.

One should remember that epidemics of citrus canker on mature plants are sporadic and very dependent on weather conditions (rainstorms, hurricanes), which are less prevalent in the Mediterranean basin. On the other hand, it should also be realized that the citrus leaf miner is already widespread in this area. Seed transmission has not been observed. Long distance dispersal is by infected planting material or infected fruits. In the past, ornamental *Citrus* have been imported from Asia.
to Europe and Xcc has been intercepted on this material (author’s personal experience). However, the risk of dispersal by infected fruit was evaluated to be very small or absent if fruits are disinfected before shipment (Gottwald et al., 2009). Without disinfecion, survival chances in symptomatic fruits are apparently also small (Shiotani et al., 2009), but not impossible, as shown by the identification of Xcc on imported fruits by the Dutch Plant Protection Service (author’s personal experience). Since Xcc is endemic and spreading in countries surrounding the Mediterranean basin, some of which have also huanglongbing problems, and the citrus leaf miner is widespread in the area, the conclusion is that this pathogen is a real and emerging threat.

BACTERIAL DISEASES WITH AN EMERGING CHARACTER, BUT NOT YET PRESENT IN EUROPE OR THE MEDITERRANEAN BASIN

Black spot of mango - Xanthomonas citri pv. mangiferaeindicata. A leaf spot and canker disease of mango (Mangifera indica) called bacterial black spot, was described in South Africa by Doidge (1915) who named the causal agent Bacillus mangiferae [later also named Xanthomonas mangiferae, and X. campestris (axonopodis) pv. mangiferae]. Ah-You et al. (2007, 2009) showed that this bacterium is much related to a pathogen from cashew (Anacardium occidentale) and named the two bacteria X. citri subsp. mangiferaeindicata and X. citri pv. anacardii, respectively. Both mango and cashew belong to the family Anacardiaceae. Symptoms of black spot start as small water-soaked spots that become later raised and necrotic, sometimes surrounded by a narrow yellow halo. On the fruits the water-soaked spots become star-shaped and cracked and often show exuding gum. Severe infection under influence of rainstorms may lead to premature leaf and fruit drop, twig cankers and twig death (Fig. 3 B-E). Other (rare) hosts are ambarella (Spondias dulcis, syn. S. cytherea) and Brazilian pepper (Schinus terebinthifolius), both belonging to the Anacardiaceae. When other diseases and pests are controlled, black spot is a limiting disease to mango production, because it is very difficult to contain. In most susceptible cultivars up to 100% fruit loss may occur. Many commercial cultivars are very susceptible. In 1996 and 1997, severe black spot epidemics were observed in many mango-growing areas of South Africa, causing almost 100% fruit loss on the most susceptible cultivars and ca. $1 million economic loss. There is production of mango in Europe and the Mediterranean basin (e.g. Spain, Italy, Israel and Portugal). Black spot occurs in Australia, Comoro Islands, many areas in southern and eastern Africa and Asia, Mauritius, New Caledonia, Reunion, Taiwan, and the United Arab Emirates (Gagnevin and Pruvost, 2001). Discrimination of strains from mango and some related hosts and from different geographic origin (Asia, Africa and Brazil) was possible using RFLP (Gagnevin et al., 1997). Long distance dissemination of the pathogen is thought to be by infected planting material. Epiphytic/endophytic populations of the pathogen occur (Pruvost et al., 2009). Seed transmission has not been demonstrated.

Bacterial blight of pomegranate - Xanthomonas axonopodis pv. punicae. A bacterial disease on pomegranate (Punica granatum, family Lythraceae) was observed for the first time in 1952 in New Delhi, India and described by Hingorani and Singh (1959) as being induced by Xanthomonas punicae (later classified as X. campestris pv. punicae and X. axonopodis pv. punicae). First symptoms are water-soaked spots on leaves and fruits (Fig. 3F). On the leaves, spots become necrotic and, when they coalesce, severe leaf drop may occur. Fruits may crack and drop. Black necrotic spots occur on branches, that become weak and may split. Pomegranate is produced mainly by India (50%), Iran (35%), some of the former Soviet Union states and in the Mediterranean basin, especially in Spain (2.5%), Morocco, Egypt and Turkey. Bacterial blight has developed into a very serious disease in India, where it causes very heavy losses (up to 100%) in many pomegranate-growing areas. (Kumar et al., 2006). Dispersal at a site is by rain splash, insects and tools, whereas over long distances is by infected plant material. Like X. citri pv. citri and pv. mangiferaeindicata, stormy weather conditions are important for epidemic outbreaks. X.a. pv. punicae overwinters in infected leaves of neem (Azadirachta indica, family Meliaceae) planted along pomegranate fields (Dhasandar et al., 2004; Sharma et al., 2008; Kumar et al., 2009) Since these former two pathogens have already spread from the more eastern parts of Asia, X. a. pv. punicae can be seen also as a potential threat that may soon show its presence closer to the Mediterranean basin.

Bacterial blight of guava - Erwinia psidii. A vascular disease of guava (Psidium guajava) was reported as bacterial blight from Brazil and the causal agent denoted Erwinia psidii (Neto et al., 1987), was held responsible for the outbreaks in the main production areas of southeastern and central regions of the country (Tokeshi et al., 1980). Symptoms are local and systemic. Leaves show large necrotic lesions along the main veins (Fig. 3G) and at the margins (scorching), or small water-soaked spots, sometimes with a chlorotic halo, that later become necrotic and coalesce causing leaf drop. When bacteria reach the xylem they spread into branches, trunk and roots. In severe cases, trees are defoliated and die. Symptoms on the fruits are not common. This disease could be of importance to guava-growing countries such as Egypt that produces yearly some 230,000 tons of guava fruits, being the fifth producer in the world after India, Pak-
istan, Brazil and Mexico. In Brazil, pathogen dispersal often occurs with contaminated planting material (Marques et al., 2007; Teixeira et al., 2008b).

**Bacterial spot of passion fruit - Xanthomonas campestris pv. passiflorae.** A destructive disease of cultivated passion fruit (*Passiflora edulis* and *P. edulis* var. *flavicarpa*), was described by Pereira in 1969 in Brazil (El Tassa, 2002). This disease is characterized by the presence of water-soaked, greasy lesions of irregular shape on the leaves, surrounded by chlorotic areas that result in extensive necrosis when they coalesce (Fig. 3H). Fruits display greasy spots that make them unsuitable for consumption and industrial processing. This disease has an emerging character in Brazil, where ca. 25,000 ha are given over to passion fruit and has become a major problem to production. Hosts are *Passiflora alata*, *P. amethystina*, *P. coccinea*, *P. edulis*, *P. edulis* var. *flavicarpa*, *P. maliformis*, *P. nitida* and *P. serrato-digitata* (Neto et al., 1984; Torres Filho and Ponte, 1994; Gonçalves and Rosato, 2000; Lopes et al., 2006).

**BACTERIAL DISEASES/PATHOGENS WITH AN EMERGING CHARACTER, ALREADY OCCurring IN EUROPE OR THE MEDITERRANEAN BASIN**

**Almond witches’ broom - Candidatus Phytoplasma phoenicium.** This devastating disease of almond (*Prunus amygdalus*), shows typical witches’ broom symptoms, i.e. small yellow leaves on proliferating shoots that wither and die in later stages (Fig. 4.A-C).

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Fig. 4. Symptoms of witches’ broom in almond. on flowering twigs (A), mature branches (B) and tree (C) caused by “*Candidatus Phytoplasma phoenicium*” (courtesy E. Choueiri). D. Malformations of tomato (*Lycopersicon esculentum*), caused by potato stolbur, “*Candidatus phytoplasma solani*” (Source EPPO).
was reported from Lebanon by Choueiri et al. (2001), and was classified and named by Verdin et al. (2003). *Candidatus* Phytoplasma phoenicium belongs to the pigeon pea witches' broom group (16SrIX). In a few years time, more than 100,000 trees were killed in different areas in Lebanon. This phytoplasma was also found on peach and nectarine (Abou-Jawdah et al., 2009) and was recently also reported from Iran on almond (Zirak et al., 2009) and on GF-677 (*Prunus amygdalus* × *Prunus persica*) (Salehi et al., 2011). Vectors are suspected to be leafhopper which have not yet been identified (Abou-Jawdah et al., 2011). This pathogen is not included in quarantine lists, but certainly deserves attention as an emerging threat for almond. Stem cutting culture with thermotherapy was successfully used for regeneration of phytoplasma-free plantlets of almond (Chalak et al., 2005). Recently Davis et al. (2010) have reported a phytoplasma closely related to *Ca. P. phoenicium* as the cause of witches’ broom affecting *Juniperus occidentalis* (western juniper) in Oregon (USA).

**Potato stolbur - *Candidatus* Phytoplasma solani.** *Ca. Phytoplasma solani* is a non-culturable, insect-transmitted bacterium that induces bushy growth and malformations in solanaceous (Fig. 4D) and other hosts (EPPO/CABI 1996) and is on the EPPO A2 quarantine list. It belongs to the so-called Aster yellows or stolbur (16Sr-XII-A) phytoplasma group, the latter having a very wide host range (‘Bois noir’ of grapevine is caused by a phytoplasma of the same group). Severe outbreaks occur under dry weather conditions when vector populations develop explosively on wild hosts. In some years, substantial damage was reported from south-east Europe and Russia. Spreading in potato is slow. Vectors are mainly leafhoppers of the family Cicadidae, the most important of which are the polyfagous *Hyalosthes obsolutus*, the true bug *Lygus pratensis* (family Miridae) and some other cicadas, such as *Macrosteles quadripunctatus*. Potato stolbur has been reported from Austria, Bulgaria, Czech Republic, Germany, France, Greece, Hungary, Italy (Berger et al., 2009), Israel, Poland, Romania, Russia, Serbia, Switzerland, Turkey and Ukraine. Main hosts for the bacterium are potato, tomato, eggplant, pepper and weeds such as black nightshade, bindweeds (*Convolvulus arvensis*, *Calystegia sepium*), stinging nettle, *Cardaria* or *Lepidium* and *Lavandula*. Larvae of cicadellids feed on the roots from which they can acquire and transmit the bacterium. Potato stolbur might spread more, when the climate warms up. *H. obsolutus* recently spread in Germany from the Moselle valley and by the end of 2009 potato stolbur phytoplasma was detected in association with bindweed in Rheinland-Pfalz (EPPO Reporting Service 2010/155). In eastern Europe (Bulgaria, Romania, Serbia), *Ca. phytoplasma solani* has been causing for many years a disease transmitted by *Reptalus panzeri* (Jovic et al., 2009) called ‘corn reden-
Canary Islands (Tenerife, since 2009) where the bacterium is apparently transmitted by the psyllid Bactericera trigonica (Alfaro-Fernández et al., 2012a). Subsequently it has also been reported from mainland Spain, where it infects celery (Apium graveolens) in several regions, also in mixed infections with Ca. P. solani and, strangely enough, with the bacterium Spiroplasma citri (Alfaro-Fernández et al., 2012b); (iii) Norway (since 2011) where CaLS causes extensive damage to commercial fields with an incidence of 10-100% (Munyaneza et al., 2012a); (iv) Sweden, since 2011 (Munyaneza et al., 2012b). Until now, CaLS has not been reported from potato, probably because the vector B. cockerelli does not occur in Europe.

Other phytoplasmas that could become a threat will not be treated in this review, apart from their mention in Table 1. For a recent overview, see COST Action FA0807 “Integrated Management of Phytoplasma Epidemics in Different Crop Systems”

Harmless (?) “Candidatus Liberibacter europaeus” found in Cacopsylla pyri. An apparent harmless Liberibacter species, named “Candidatus L. europaeus” was reported from Italy to occur in a high percentage of the psyllid Cacopsylla pyri that thrives on pear trees (Pyrus communis). The bacterium could be experimentally transmitted by the psyllid to pear but, apparently, ut remains until now non-pathogenic (Raddadi et al., 2011).

Bacterial fruit blotch of Cucurbitaceae - Acidovorax citrulli (syn. A. avenae subsp. citrulli). Symptoms of bacterial fruit blotch are water-soaked leaf spots on seedlings that may coalesce in later stages and kill the plantlets. Mature plants show reddish brown streaks along main veins and, most characteristically, dark green spots on the fruit surface (watermelon, Fig. 5C) or inside the fruit (melon and pumpkin) which are accompanied by rind depressions and cracks from which bacteria may ooze. Hosts are watermelon (Citrullus lanatus) and melon (Cucumis melo), but also Cucumis sativus (cucumber), Cucurbita pepo (squash), and C. moschata, which show symptoms only on the leaves, and wild cucurbits, e.g. Citrullus lanatus var. citroides. Deng et al. (2010) reported A. citrulli outbreaks on Piper betle (betelvine, Piperaceae) in Taiwan. Strain diversity, i.e. mild strains on hosts other than watermelon and more aggressive strains from watermelon, has been observed in the USA (Walcott et al., 2004). Recent outbreaks of bacterial fruit blotch were reported in Europe from Greece (2005), Hungary (2007, apparently with watermelon transplants from Turkey), Israel (2000 and 2003, melon and watermelon), Turkey (1995, Marmara region and 2005 Mediterranean region) and Italy (2009, melon) [see also: http://www.eppo.org/QUARANTINE/Alert_List/bacteria/Acidovorax_citrulli.htm; Holeva (2009); Hopkins et al. (2001); Hopkins and Thomson (2002)]. The bacterium is mainly spread with seed and

![Fig. 5. A. Malformations, leaf rolling and necrosis in potato caused by “Candidatus Liberibacter psyllaurous”. B. Characteristic so-called zebra stripes, caused by the same bacterium when potatoes are fried (Courtesy J.M. Crosslin). C. Water-soaked spots on melon (Cucumis melo), caused by Acidovorax citrulli (Courtesy M.Holeva).](image_url)

### Table 1. Emerging phytoplasmas reported at the Meeting on “Emerging phytoplasma diseases of stone fruits and other crops and their possible impact on EU countries”, held in 2011 in Istanbul, Turkey.

<table>
<thead>
<tr>
<th>Name</th>
<th>16S rRNA Group</th>
<th>Latin name</th>
<th>Country where reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almond witches’ broom</td>
<td>16S rRNA Group</td>
<td>“Ca. P. phoenicum”</td>
<td>Lebanon</td>
</tr>
<tr>
<td>Cassava frog skin</td>
<td>16S rRNA Group</td>
<td>“Ca. P. phoenicum”</td>
<td>Colombia</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16S rRNA Group</td>
<td>“Ca. P. asteris”</td>
<td>Italy, South Africa</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16S rRNA Group</td>
<td>“Ca. P. fraxini”</td>
<td>Chile</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16S rRNA Group</td>
<td>“Ca. P. ziziphi”</td>
<td>China, Korea, Italy</td>
</tr>
<tr>
<td>Lime witches’ broom</td>
<td>16S rRNA Group</td>
<td>“Ca. P. aurantifolia”</td>
<td>Oman/Iran</td>
</tr>
<tr>
<td>Potato purple top wilt</td>
<td>16S rRNA Group</td>
<td>“Ca. P. trifolii”</td>
<td>USA</td>
</tr>
<tr>
<td>Potato purple top wilt</td>
<td>16S rRNA Group</td>
<td>“Ca. P. americanum”</td>
<td>USA</td>
</tr>
</tbody>
</table>

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planting material and the disease it elicits may result in extensive damage and losses (40-100% in USA and Brazil). Control measures to be implemented are: (i) use of healthy tested seed (seed treatment has not been effective up to now); (ii) seed test by a check on 10,000 seedlings/lot in a greenhouse (so-called sweatbox method); (iii) inspection of plants during the growing season and destruction of infected plant material. In recent years also PCR screening tests have been developed (Bahar et al., 2008; Jing et al., 2011; Woudt et al., 2012). Positive results of all these methods still need to be verified by isolation of the pathogen. Perhaps RT-PCR in combination with malditof (matrix assisted laser desorption ionization time-of-flight mass spectrometry) could be used to exclude false positives (Wang et al., 2012).

A new strain of a the potato stem rot bacterium belonging to the genus *Dickeya*, provisionally named *D. solani*. Bacterial stem rot of potato, caused by different ‘cold tolerant’ biovars of *Erwinia chrysanthemi*, now named *Dickeya dianthicola* and *D. chrysanthemi*, has been reported from many European countries since the 1970s, and is regarded as a quality disease (Janse and Ruissen, 1988; Toth et al., 2011). A taxonomic revision placed *Erwinia chrysanthemi* biovars into six species of the newly created genus *Dickeya*, namely *D. chrysanthemi* biovar *chrysanthemi* and biovar *parthenii*, *D. dadanti*, *D. dianthicola*, *D. dieffenbachiae*, *D. paradisiaca* and *D. zeae* (Samson et al., 2005). Among them there are ‘warm tolerant’ species like *D. dadanti* and *D. zeae* that were found not only in potato in hot climates, but also in many ornamental plants, often grown in greenhouse in temperate regions (Janse and Ruissen, 1988; Janse and Scheepens, 1990; Samson et al., 1990). Recently a new ‘warm tolerant’ variant of *Dickeya* was observed, first in the Netherlands since 2000 (Czajkowski, 2009a, 2009b, 2011; J. Van Vaerenbergh, personal communication), then in Poland, Belgium, Finland, France, Israel and most recently also in the UK (2010), Denmark and Sweden (2011). This variant is virulent under warm climatic conditions and is closely related, but not similar to *D. dadanti*, it has been provisionally denoted *D. solani* (Czajkowski, 2011). A recent taxonomic study indicates that it is not certain whether this variant is indeed a new species (Van Vaerenbergh et al., 2012). Warm tolerant *Dickeya* spp. have been isolated from surface waters and *D. solani*, that shows a strong clonal character, in the Netherlands also from hyacinth and iris in the bulb production area where no potatoes are grown (Toth et al., 2011; Van Vaerenbergh et al., 2012). A theory is that a warm tolerant species (*D. dadanti* or *zeae*) escaped from greenhouse wastewater disposals to surface water and mutated to a form that clonally spread in hyacinth, iris and potato. As with the other *Dickeya* and *Pectobacterium* species in potato, effective control is possible and regulated via certification schemes. It should be based on the use of tested, healthy seed and careful cultural practices (careful and dry harvesting, proper storage and ventilation, avoiding of desprouting and cutting seed, hygiene on the farm, etc).

**Stem rot and leaf spot of maize and ‘center rot’ of onion - *Pantoea ananatis***. *Pantoea ananatis* can cause a number of diseases on different hosts, e.g. stem rot and leaf spot of maize, internal fruit rot of melon and pink disease of pineapple. It also infects onion (*Allium cepa*) inducing ‘center rot’, outbreaks of which have been reported since 1997 in the USA, and was isolated from onion seed in South Africa (Goszczynska, 2006). Seed transmission was confirmed in the USA (Walcott et al., 2002). The thrips *Franklinella fusca* can transmit the bacterium which apparently survives as a saprophyte on many weed and crop hosts (Gitaitis et al., 2003).

**Stewart’s disease or bacterial wilt - *Pantoea stewartii* subsp. stewartii**. This vascular disease, characterized by white stripes on the host leaves, is widespread in North America. The main host is maize, especially sugar maize, but also the so-called ‘dent’, ‘flint’, ‘flour’ and popcorn types. The bacterium is mainly spread by *Chaetocnema pulicaria*, the corn flea beetle. *P. stewartii* and bacterial wilt have been reported from, but are not established in Austria, Greece, Italy, Poland, Romania and European Russia. Main source of introduction is contaminated seed from North America, but pathogen and disease disappear apparently some years after introduction, due to absence of the vector beetle in Europe. Local vectors do not transmit the bacterium as far as is known. In Italy, substantial damage was reported in the years 1940-1950, and some reoccurrence, but not very damaging in 1983-1984 (Mazzuchl, 1984; www.eppo.org/QUARANTINE/bacteria/Pantoea_stewartii/ERWIST_ds.pdf). Further introductions could occur in the future, but will remain relatively harmless as long as the vector is not introduced or local flea beetles, as potential vectors, do not acquire and transmit the pathogen.

**New outbreaks of bacterial (blight) canker of kiwifruit - *Pseudomonas syringae pv. actinidiae***. Bacterial canker of kiwifruit was first observed in Japan in 1984 (Takikawa et al., 1989). It was also reported from China where, as we know now, it occurred already as early as 1984/1985 (Liang et al., 2000). Subsequently it was found in Korea and Italy (1992). Hosts are green kiwifruit (*Actinidia deliciosa*), yellow kiwifruit (*Actinidia chinensis*), *A. arguta*, and *A. kolomikta*. Since 2008 bacterial canker outbreaks have repeatedly been observed in Italy (Emilia-Romagna, Lazio, Piemonte and Veneto) on green kiwi, causing a mild leaf spot and some canker formation on branches and trunk. There was also a re-
Port from Iran in 1994 and a recent occurrence of the so-called Asian or mild strain in Australia (EPPO Reporting Service 2011/130). In spring and autumn of 2008 and winter 2008/9, however, severe outbreaks occurred on yellow kiwi characterized by wilting, necrosis, severe leaf spot and canker formation (Fig. 6A-C), especially the cvs Hort 16A and Jin Tao cultivated in central Italy (Latina province). Recently, severe outbreaks have also occurred on green kiwi cv. Hayward (Ferrante and Scortichini 2010) and the disease has spread to Calabria, Campania, and Friuli-Venezia Giulia regions in 2011 (EPPO Reporting Service 2011/131). *P. s. pv. actinidiae* was recently observed in yellow and green kiwi in France [severe form found in the Rhône-Alp area in 2010 (EPPO Reporting Service 2012/002)], Spain (Abelleira *et al.*, 2011; Balestra *et al.*, 2010), Portugal (Balestra *et al.*, 2010), Switzerland (2011), Chile (2011) (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm) and Turkey, where it is present since 2009 in the Black Sea area (Bastas and Karakaya, 2012). Interestingly, the severe form is also known to occur since 2010 in New Zealand and has spread on the North and South Island (Everett *et al.*, 2011; Young, 2012). In this country another mild strain causing only leaf spots has also been observed, which may be not *P. s. pv. actinidiae* (Vanneste *et al.*, 2010). The Asian mild strain was recently reported to be present in Australia (EPPO Reporting Service 2011/130). A PCR test for identification and epidemiological studies was developed by Vanneste *et al.* (2010). Epidemics of bacterial blight of kiwi occur usually after frost damage. In Japan and Korea bacterial blight is the most limiting

![Symptoms induced by an aggressive strain of *Psudomonas syringae pv. actinidiae* on yellow kiwifruit (*Actinidia chinensis*) in Italy. Wilting and necrosis (A), necrotic leaf spots (B), canker with a reddish ooze (C) (Courtesy M. Scortichini). D-F: Symptoms induced by *Pseudomonas syringae pv. aesculi* on Horse chestnut (*Aesculus hippocastanum*). Severe wilting and yellowing (D), red ooze from a canker (E), extensive cankers on a branch (F) (Courtesy Aesculaap, NL). G: Leaf spots with a yellow halo and ‘shotholes’ on cherry laurel (*Prunus laurocerasus*) caused by *Xanthomonas arboricola pv. pruni* (Source Naktuinbouw, NL).]

**Fig. 6.** A-C. Symptoms induced by an aggressive strain of *Psudomonas syringae pv. actinidiae* on yellow kiwifruit (*Actinidia chinensis*) in Italy. Wilting and necrosis (A), necrotic leaf spots (B), canker with a reddish ooze (C) (Courtesy M. Scortichini). D-F: Symptoms induced by *Pseudomonas syringae pv. aesculi* on Horse chestnut (*Aesculus hippocastanum*). Severe wilting and yellowing (D), red ooze from a canker (E), extensive cankers on a branch (F) (Courtesy Aesculaap, NL). G: Leaf spots with a yellow halo and ‘shotholes’ on cherry laurel (*Prunus laurocerasus*) caused by *Xanthomonas arboricola pv. pruni* (Source Naktuinbouw, NL).
factor to kiwi crops, although mild strains appear to occur there. From Italy in recent years more than 2 million euro damage was reported. In a recent taxonomic study, with extensive sequence analysis of the diverse strains, Mazzaglia et al. (2012) came to the conclusion that severe strains from Europe and New Zealand indeed differ from the mild strains that occur in Japan, Korea and were also present in Italy in the past, and that are similar to strains from China, the country where the bacterium probably originates. They also showed that New Zealand and European strains differ to a small extent and both are more close to the Chinese strains, implicating a possible origin of these strains in China, probably imported with planting material from that country. Due to the emerging character of the new severe strains of \( P. syringae \) \( pv. \) actinidiae, EPPO has placed it on its alert list (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm).

**An emerging disease in forestry: Bleeding canker of horse chestnut - \( Pseudomonas syringae \) \( pv. aesculi \).** Since 2002 an emerging bacterial disease, named bleeding canker of horse chestnut (\( Aesculus \) spp., especially \( A. hippocastanum \)) was observed first in the Netherlands, then in Belgium, France, Germany and the UK. It is thought that the disease had longer been present in the countries concerned, with an incidence of 40-60%, but that symptoms were often mistaken for those of fungal diseases. Progress of the disease was rapid in the early 2000 but the epidemic in the Netherlands seems to have slowed down considerably in recent years (author’s personal observation). The pathogen appears to be a mutant of a bacterium from \( Aesculus \) earlier described in India as \( Pseudomonas syringae \) \( pv. aesculi \) (Durgapal and Sing, 1980). The bacterium shows a clonal character and adaptation to horse chestnut (Green et al., 2010, 2012). Symptoms are cracks with reddish-brown exudate on trunk and branches, and extensive necrotic cankers in phloem, where death of cambium leads to dieback (Fig. 6D-F). Insect transmission cannot be excluded, such as with rain and wind in springtime stimulate epidemics. The economic impact of \( X.a. \) \( pv. pruni \) consists of reduced quality and marketability of fruits and reduced productivity of the trees, as well as higher production costs.

**Bacterial leaf spot of poinsettia - \( Xanthomonas axonopodis \) \( pv. poineartricola \).** This bacterium was originally found in poinsettia (\( Euphorbia pulcherrima \)) in India (Patel et al., 1951), then reported from Florida (USA) (Chase, 1985). Other Euphorbiaceae are susceptible, such as \( E. heterophylla \) (wild poinsettia, mainly occurring in North America, but now widespread in Italy), \( E. milii \) (crown-of-thorns), \( Codiaeum variegatum \) (croton) and \( Manihot esculenta \) (cassava) (Chase, 1985; CABI, 2011). \( E. pulcherrima \) originates from Mexico and is a very popular Christmas pot plant in European countries, on which \( X.a. \) \( pv. poineartricola \) causes brown to black leaf spots, sometimes surrounded by a yellow halo. Over time the spots may coalesce and the leaves turn completely yellow and drop, severely impairing the commercial value of the plants (Wohanka, 2004). The disease occurs in Cocos Islands (territory of Australia), the Philippines (Quimio, 1974), Taiwan (Lee et al.,...
CONCLUDING REMARKS

A number of specific and general prevention and control measures for emerging diseases have been listed under Liberibacter spp. The following further measures can be considered important in the management of emerging bacterial diseases:

- Ornamental and wild hosts may play an important role in spreading diseases and maintaining the pathogen and its vectors in the environment. These plants should be included in surveys.

- Rapid and reliable diagnosis remains a key issue, as well as breeding for resistance. All pathogens mentioned are emerging threats, with real risks of introduction and in some cases closely approaching or already present in the Mediterranean basin and/or Europe.

- Introduction of susceptible wild hosts and susceptible cultivars of cultivated hosts must be avoided, as they often lead to introduction of the pathogen or to outbreaks due to a ‘jump’ of local pathogen strains to the very susceptible cultivar. Introduction of vectors should also be prevented as history teaches that the vector(s) appear first and the pathogen a few years later. Examples of these facts are presented in the description of the various disease-pathogen combinations.

- An efficient prevention and control strategy of diseases described in this review should be based on so-called pathway protection. This means it should be based on regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-regulated pests, where the following conditions should be maintained and controlled: (i) place of production should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

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subsp. aurantifolii (ex Gabriel 1989) sp. nov. nom. rev. comb.
comb. nov., and *X. alfalfae* subsp. citruscello (ex Riker and
Jones) Gabriel et al., 1989 sp. nov. nom. rev. comb. nov.; *X.
campestris pv. malvaecarum* (ex Smith 1901) Dye 1978 as *X.
smitii* subsp. smithii nov. comb. nov. nom. nov.; *X. campestris
campestris* subsp. alfalfae (ex Riker and Jones, 1935) Dye 1978
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doi:10.1371/journal.pone.0035738.

Verdin E., Salar P., Danet J.L., Choueiri E., Jreijiri F., Zammah


Mediterranean basin and/or Europe. Introduction of susceptible wild hosts and susceptible cultivars of cultivated hosts must be avoided, as they often lead to introduction of pathogens or to outbreaks due to a ‘jump’ of local pathogen strains to very susceptible cultivars. Introduction of vectors should also be prevented as history teaches that vectors appear first and the pathogens a few years later. Examples of these events are given in the description of the various disease-pathogen combinations. An efficient prevention and control strategy of the diseases mentioned in this review should be based on the so-called pathway protection, i.e. regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-regulated pests, where the following conditions should be enforced and controlled: (i) place of production should have integrated pest management practices; (ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

Key words: quarantine, risk assessment, geographical distribution, host plants, plant pathogens.

INTRODUCTION

Bacterial diseases are often a destructive and/or a major constraint for many crops. Substantial killing of the host and yield losses, often over 50% result from infestations caused by established pathogens such as Erwinia amylovora, Pseudomonas syringae pv. syringae and pv. morrisonorum, Agrobacterium tumefaciens (syn. Rhizobium tumefaciens) Xanthomonas axonopodis pv. prunorum, Candidatus Phytoplasma mali, Grapevine flavescence dorée phytoplasma, Grapevine bois noir phytoplasma, "Candidatus Phytoplasma pyri" and "Candidatus P. prunorum" (European stone fruit yellows). Quarantine pathogens sometimes spread out of contained loci, presenting a long-term threat to other EU countries (e.g. Xanthomonas arboricola pv. prunorum spreading out of France and Italy) and others are an emerging threat outside Europe (Xylella fastidiosa, Candidatus Liberibacter spp. agents of citrus huanglongbing).

Bacterial diseases are difficult to control (both chemically and biologically) and are restrained primarily by preventive measures such as hygiene, use of healthy planting material, good cultural practices and avoidance of risky planting sites. Moreover, they are easily spread by (surface) water, planting material and contaminated agricultural implements/machines and by aspecific or specific insect vectors. Most important risk factors for the introduction of bacterial diseases into Europe are imported infected planting material and naturally spreading insect vectors. Therefore, early detection and correct identification/diagnosis are of utmost importance. In recent reviews, current classic and molecular methods for detection and identification of bacterial pathogens of fruit trees and nuts, including Xylella fastidiosa, have been described (Janse, 2010; Janse and Obradovic, 2010; Janse et al., 2012). In this contribution the epidemiology and main risks in the framework of prevention and management in case of first occurrence of some emerging bacterial diseases approaching the Mediterranean basin and Europe, their causal organisms and vectors, will be highlighted, with special reference to: (i) Citrus huanglongbing (HLB, formerly called Citrus greening), caused by the heat tolerant "Candidatus Liberibacter asiaticus" and the heat sensitive "Candidatus L. africanus". Both liberibacters and the respective psyllid vectors Diaphorina citri and Trioza erytreae are present in the Arabian peninsula, with recent reports of huanglongbing occurring in Iran, Mali, Ethiopia and Somalia and T. erytreae already present on some Atlantic Ocean islands (for references see under Geographical distribution of the specific pathogens later in the text). Furthermore, in less detail: (ii) leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by Xylella fastidiosa. For this pathogen, although its presence in Europe and the Mediterranean basin has not been confirmed, local possible vectors such as Cacidaella viridis and Philaenus spumarius occur; (iii) Citrus canker, caused by Xanthomonas citri pv. citri (syn. X. axonopodis pv. citri, X. citri subsp. citri), the most severe form of which, the so-called Asiatic, is already present in Iraq, Iran, Oman, Somalia, UAE, Saudi Arabia, Yemen and Reunion (for references see under Geographical distribution of the specific pathogens later in the text).

Outbreaks and the possible emerging character of some other bacterial pathogens will be dealt with (e.g. Xanthomonas citri pv. mangiferaeindicae approaching the Mediterranean basin like X. c. pv. citri and the devastating "Candidatus Phytoplasma phoenicium" that occurs on almond in Lebanon). Furthermore, some diseases already present in the area which show an emerging character, such as the bacterial canker of kiwifruit (Actinidia spp.) caused by Pseudomonas syringae pv. actinidiae in Italy and France in the last three years, will also be addressed.

Since initial management and risk avoiding measures following an introduction are more or less the same for most of the above-mentioned pathogens, they will be detailed for HLB. It will be argued that the diseases addressed in this presentation are emerging threats, with real risks of introduction and economic damage and in some cases closely approaching the Mediterranean basin. The aim of this contribution is to create awareness of the risks of these diseases, thus enabling prevention, early detection and proper actions once introduction has occurred.
MAIN EMERGING BACTERIAL DISEASES

Citrus huanglongbing caused by “Candidatus Liberibacter” species. General. Huanglongbing or HLB (Chinese for yellow shoot disease) of citrus is caused by a non-culturable, fastidious, phloem-inhabiting, Gram-negative bacterium belonging to the α-Proteobacteria and to the genus “Candidatus Liberibacter” [originally the genus was named Liberobacter (Bové, 2006)]. This disease (Fig. 1A-C) was formerly known as Citrus greening yellow dragon disease, mottle leaf disease, ‘likubin’ or vein phloem degeneration. It is one of the most destructive diseases of cultivated citrus for which no effective control is available with the consequence that citrus production declines in all areas where the disease occurs. Lin (1956) determined that this disease is graft-transmissible and described it for the first time in China. The origin of huanglongbing is not clear, probably Asia (India or China) or Africa (Gottwald et al., 2007). For a thorough general review on the disease and its causal organism see Bové (2006) and for a review on its epidemiology see Gottwald (2010).

Three different Liberibacter species causing Huanglongbing have been described based only on 16S rRNA and whole genome sequences information, because these organisms cannot be cultivated in vitro:
a. “Candidatus Liberibacter asiaticus” (Las), originally described as Liberobacter asiaticum, the most aggressive species, is heat-tolerant and stands temperatures above 30°C. The disease caused by this species is found mainly in lowlands where it is transmitted by the heat-tolerant psyllid Diaphorina citri Kuwayama (Fig. 1D,E) (a recent report mentions the black psyllid, Diaphorina communi, as a vector in Bhutan (Donovan et al., 2011)]. Las is widespread in Asia, the Arabian peninsula, Mauritius and Reunion islands and, since 2004, in Brazil, Sao Paulo state (in Brazil some 10% of the infected plants are infected by this Liberibacter species, others are infected by the third species, Ca. L. africanus (Teixeira et al., 2005)). It occurs since 2005 in Florida (USA) (Halbert, 2005).
b. “Candidatus L. africanus” (Laf), described as Liberobacter africanus, less aggressive and heat-sensitive. The disease induced by Laf is suppressed after exposure to temperatures above 30°C, therefore it occurs in the tropics at elevations higher than 700 metres above sea level. Laf is widespread in Africa (Somalia, Ethiopia and Cameroon) and occurs also in Reunion, Mauritius, and Yemen. Its vector is the heat-sensitive psyllid Trioza erytreae Del Guercio. c. “Candidatus L. americanus” (Lam), closely related to Las but heat-sensitive. It occurs in Brazil and is transmitted by the psyllid D. citri.

A recently described subspecies, “Candidatus Liberibacter africanus” subsp. capensis, was isolated from a symptomless ornamental rutaceous tree (Caldendrum capense) in the Western Cape province (South Africa). In the article describing this subspecies, the genus name Liberobacter was changed for all species into Liberibacter (Garnier et al., 2000). Ca. L. africanus subsp. capensis appears to be widespread in C. capense in South Africa, but is not transmitted to citrus (Phahladira et al., 2012) The complete genome sequence of Las is available (Duan et al., 2009) and, recently, differences between the Asiatic (China) and North American strains of Las were reported (Chen et al., 2010). Liberibacter species are apparently not seed-transmissible (Hartung et al., 2010; Hilf, 2011).

Some biological traits of the psyllid vectors. Both Diaphorina citri and Trioza erytreae feed on the phloem and are experimentally able to transmit both the Asian and African HLB. Fourth and fifth instar nymphs and adults can acquire and transmit the bacteria during their whole life, after a latent period of ca. 10 days, after feeding times varying from 15 to 20 min for D. citri to 24 h for T. erytreae. Multiplication in the vector has not clearly been demonstrated, the bacteria, however, can be found in large amounts in the haemolymph and salivary glands. For T. erytreae there is evidence that transovarial transmission occurs. Other psyllids that thrive on citrus were not found to transmit the bacteria, except for the black psyllid (Diaphorina communis) which is a Las vector in Bhutan (Donovan et al., 2011). Psyllids like to feed on new vegetation flushes the presence of which constitutes a risk for transmission. Although D. citri does not tolerate frost very well, it survived frosty days of up to -5°C in Florida (Bové, 2006; Bransky and Rogers, 2007; Gottwald, 2010; Gottwald et al., 2007; Halbert and Manjunath, 2004; Manjunath et al., 2008).


(i) Bacteria. Las (transmitted by D. citri in all countries mentioned). South east Asia: Cambodia, China (including Hong Kong), Indonesia, spreading along southern islands of Japan (Shinohara et al., 2006), Laos, Malaysia, Myanmar, Philippines, Taiwan, east Timor, Thailand, and Vietnam; Indian subcontinent: Bangladesh, Bhutan, India, Nepal, and Pakistan; Western Asia: Iran in Sistan-Baluchistan and Hormozgan provinces (Faghhi et al., 2009; Salehi et al., 2012); Indian Ocean: Comoros Islands, Madagascar, Mauritius, Re-
union and Sri Lanka; Arabian peninsula: Saudi Arabia, Yemen in the south west along the Red Sea; Africa: Northern Ethiopia (De Bac et al., 2010); South America: Brazil, mainly Sao Paulo state, since 2004; Caribbean: Cuba (Martinez et al., 2009), Dominican Republic (Matos et al., 2009), Belize (Manjunath et al., 2010); North America: USA [Florida since 2005; Louisiana, 2008; Georgia and South Carolina, 2009; California 2012]; Mexico, 2009. (http://www.pestalert.org/oprDetail.cfm?oprID=382; http://www.pestalert.org/oprDetail.cfm?oprID=321&keyword=citrus%20greening); http://www.pestalert.org/oprDetail.cfm?oprID=516, http://www.pestalert.org/oprDetail.cfm?oprID=401)


Laf and Las. Ethiopia, Mauritius, Reunion, Saudi Arabia and Yemen host both vectors (D. citri and T. erytreae) and both pathogens.

Lam. Brazil. Lam has also been reported (but not confirmed) from one of 97 citrus leaf samples from eight provinces of southern China (Lou et al., 2008).


T. erytreae. Africa: Burundi, Cameroon, Central African Republic, Ethiopia, Kenya, Malawi, Nigeria, Rwanda, Somalia, South Africa, Sudan, Swaziland,

Fig. 1. A. Sweet orange with typical symptoms of huanglongbing or citrus greening (source EPPO, J. Bové). B. sweet orange leaves with more or less typical huanglongbing-induced yellowing. Spiroplasma citri or zinc deficiency can cause similar symptoms (source EPPO, J. Bové). C. Mandarin fruits with typical greening (courtesy of J. Gottwald). D. Adult Diaphorina citri vector of "Candidatus Liberibacter asiaticus", the Asian huanglongbing strain. E. Nymph of D. citri. Size of the adult 3-4 mm [Source Conant et al. (2007)].

(i) Liberibacter species. Rutaceae: Most Citrus species and forms are or can be host of Liberibacters. Sweet oranges, mandarins and tangos (C. reticulata x C. paradisi) are generally the most susceptible showing severe symptoms. Lemons, grapefruits, C. limonia, C. limettioides, rough lemons, kumquats (Fortunella spp.) and citrons are less severely affected. Symptoms are mild on limes and pummelos (C. grandis). However, in Brazil and the USA all commercial Citrus species have a very similar susceptibility. Other confirmed hosts are Limo-

(ii) Vectors (D. citri and T. erytreae). Rutaceae, all cultivated and wild species listed above and Clausena anisata (= C. inaequalis) and Zanthoxylum capense (= Fagra capensis).

Symptomatology. Symptoms (Fig. 1 A-C) may be confused with those of other diseases and disorders. More typically, the first symptoms are one or more yellowing shoots. Leaves are asymmetrical when the two leaf-halves are compared and exhibit well-defined yellow areas, called blotchy mottling, that may also occur on fruits. With time, yellow spots may intensify, resembling very much zinc deficiency. Veins may become corky, giving the leaves a thicker appearance. Fruits are often smaller, asymmetrically misshaped (lopsided) and show persistent green areas, especially at the stylar end. When infected fruits are cut, yellow-brown vascular bundles and necrotic seeds may be observed. The final stages of the diseases are characterized by severe leaf and fruit drop, and the fruit quality impaired. Yield losses may be 30-100% and, within 7-10 years from planting, the groves may lose productivity completely. It has been calculated that ca. 100 million trees have been killed in south-east Asia, India, Arabian peninsula, and South Africa, leading to decline of the citrus industry in these areas. In southwestern Saudi Arabia, sweet orange and mandarin have practically disappeared already during the 1970s. Since 2004, ca. 1 million trees have been destroyed in Brazil due to HLB infection. In Florida HLB was detected for the first time in 2005 and by 2009 it has spread to most citrus-growing areas, so that eradication efforts were given up (Bové, 2006; Gottwald, 2010) and the select agent status for all three Liberibacter species by USDA APHIS abandoned. 

Damage and losses. Since resistance against HLB is a dangerous and devastating disease and, as yet, no sources of resistance have been found or created. Yield is decreased, mainly because of reduced growth and fruit drop, and the fruit quality impaired. Yield losses may be 30-100% and, within 7-10 years from planting, the groves may lose productivity completely. It has been calculated that ca. 100 million trees have been killed in south-east Asia, India, Arabian peninsula, and South Africa, leading to decline of the citrus industry in these areas. In southwestern Saudi Arabia, sweet orange and mandarin have practically disappeared already during the 1970s. Since 2004, ca. 1 million trees have been destroyed in Brazil due to HLB infection. In Florida HLB was detected for the first time in 2005 and by 2009 it has spread to most citrus-growing areas, so that eradication efforts were given up (Bové, 2006; Gottwald, 2010) and the select agent status for all three Liberibacter species by USDA APHIS abandoned.

Uganda, Tanzania, and Zimbabwe; Indian Ocean: Madagascar, Mauritius and Reunion; Arabian peninsula: Saudi Arabia, Yemen; Atlantic Ocean: Canary Islands [Tenerife, La Gomera, La Palma and El Hierro since 2002 (Perez Padron and Hernandez, 2002)], Madeira (1994) and Porto Santo Island (Fernandes and Franco-


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Main risk factors. HLB and its vectors have not yet been reported from the European mainland or the Mediterranean basin, where there are no local psyllid vectors known. Long distance spread of Liberibacter spp. and vectors is possible and will mainly be with planting material. In Kenya, infected breeding material from abroad was suspected to be the source of initial infection (Magomere et al., 2009). Note that HLB is approaching the Mediterranean basin mainly from three sides: (i) Saudi Arabia, where both Laf and Las are present and could move further north (e.g. also with people travelling to and from Mecca), possibly also for Las and its vector should global warming continue. Note that Las and its vector could also establish in subtropical Florida; (ii) Africa, where Laf and its vector are already present in Ethiopia and Somalia and Las is established in north Ethiopia; (iii) Iran, where Las has been reported in 2009. Note that D. citri was present in Florida since 1998 and HLB followed in 2004 (also see Bové, 2006), and that the Laf vector is already present in the Atlantic, i.e. the Canary, Madeira, Porto Santos and St. Helena islands. Murraya paniculata (orange jasmine), a popular landscape plant, played an important role in spread of HLB and D. citri in Florida (and probably also to other states), also via garden centres (Manjunath et al., 2008). Murraya species of Asian origin have been marketed (also in bonsai form) in Europe for quite some years. Infections may go unnoticed for several years after they became established due to latency or overlooking of the early symptoms. HLB is suspected to have been present several years before its official detection in Florida in 2005 (Gottwald et al., 2007; Gottwald, 2010).

Prevention and management following first introduction. Once established, HLB bacteria and vectors are very difficult to control. Main strategies are geographical isolation and certification/indexing programs for budwood sources and nursery production in insect-proof greenhouses, chemical and/or biological vector control and intensive surveying (by visual inspection and laboratory testing) with removal and destruction of infected trees (both visually and latently infected trees) wherever possible. Healthy budwood can be obtained by shoot-tip grafting, or alternatively by heat treatment, e.g. water-saturated hot air at 49°C for 50 min and fumigation of budwood against HLB vectors is possible (EPPO, 1988). The visual inspections in the groves should be very intensive and performed from moving inspection platforms. Chemical sprays, for vector control, to be effective, should also be performed frequently. Antibiotic treatment has been abandoned completely as it was not effective and is not human and environmentally friendly. Biological control was effective in Reunion Island, with hymenopterous ectoparasites Tamaria dryi for Laf and T. radiates for Las, probably because hyperparasites of the parasite were absent. At present the only successful prevention/control methods appears to be the one followed in São Paulo state in Brazil, following the introduction of huanglongbing in 2004. The control program that was immediately installed in 2004 by responsible organisations, became very successful after eight years, when disease incidence on more than 200,000 ha was reduced to less then 1%. This was because: (i) at the beginning of the program the disease incidence was still low (less then 8%) and (ii) it could be executed on large, easy to handle farms (>500 ha). The basic principles of the so-called Three-Pronged System (TPS) are: (i) intensive vector control, (ii) reduction of inoculum by tracing, identifying and removing infected trees, and (iii) production of healthy nursery stock in covered, insect proof nurseries and their use for re-planting (Bové, 2012; Belasque et al., 2010).

Genetic resistance development has started, but is still in its infancy (Grosser et al., 2008). Still, breeding for resistance will remain one of the most important ways to control this disease and those described in the following paragraphs.

HLB bacteria and their vectors should be prevented from entering the Mediterranean basin by strict inspection and laboratory testing of imported breeding material from risk areas. In the European Union the importation of citrus planting material from third (non-EU) countries is already prohibited (Annex III A of Directive 2000/29/EC) and EPPO (EPPO, 1988) and the introduction of plants for planting and cut citrus branches from countries where HLB or its vectors occur should also be prohibited. However, when such material is imported, it should be fumigated and quarantined for at least two years. Since Liberibacter spp., D. citri and T. erythraeae are quarantine organisms included in the EPPO A1 list and EC Annex II/la list, the following additional measures can be enforced [see also Bassanezi et al., 2008; Bové, 2006; EPPO, 1988; Gottwald, 2010; USDA, 2010]. For (pro-active) measures advised by the Australian government, see http://www.agnet.org/library/eb/607/):

- Murraya spp. and ornamental citrus spp. already present in garden centres or in plantations should be surveyed, including vector sampling. In suspect areas this could also been done in (young) citrus orchards.
- Importations from risk areas of these plants strictly inspected and laboratory-tested.
- Proactive training of diagnosticians, surveyors, nursery managers and producers.
- Production in citrus nurseries should be in secure, insect-proof screenhouses.
- Once an introduction has occurred, infected trees should be destroyed and vectors controlled by chemical insecticides.
- When executing severe eradication programs, governments should consider compensation or facilitate insurance to secure full cooperation also from small growers and private tree-owners.
Further training of all parties involved and development of a public information program.

Leaf scorch and leaf scald diseases of diverse fruit and ornamental trees, caused by *Xylella fastidiosa*. General. The bacterium *Xylella fastidiosa* is a xylem-inhabiting, vector-transmitted, very slow growing, Gram-negative bacterium. It was cultured and described for the first time in 1987 in the USA as the cause of Pierce’s disease (PD) of grapevine (disease observed already in 1884) and as the cause of phony peach disease (PPD) in peach, *Prunus persica* (disease observed in 1890 in the USA). In 1993 *X. fastidiosa* was identified as the cause of citrus variegated chlorosis (CVC) or citrus X disease in Brazil. The bacterium also causes a number of so-called leaf scorch diseases to *Prunus* spp. (including almond leaf scorch or ALS in *P. amygdalus* and plum leaf scald or PLS in *P. domestica*), *Acer*, *Carya illinoinsensis* (pecan), *Coffea arabica* (CLC, in Brazil isolated in 1995 and also pathogenic to citrus), *Hedera helix*, *Morus rubra*, *Nerium oleander* (OLS), *Platanus occidentalis* (Fig. 2A), *Quercus* spp. and *Ulmus americana*. *X. fastidiosa* is also the agent of alfalfa dwarf and of wilting of *Vinca major*. Many wild plants such as grasses, sedges and trees may carry the pathogen, often without showing symptoms. None of these diseases is apparently seed-borne. Seed transmission, however, has been reported in sweet orange, *C. sinensis* (Li et al., 2003). They occur mainly in tropical/subtropical areas, although leaf scorch diseases are present also in much colder climate, e.g. oak leaf scorch in eastern North America up to Canada.

Several pathogenic varieties of the bacterium have been described, that are often host-specific (e.g., the PD strain will not cause disease if introduced to peach or plum). The following subspecies have been described:

(i) *Xylella fastidiosa* subsp. *fastidiosa* (erroneously named *X. f. subsp. piercei*), PD and LSA, strains from cultivated grape, alfalfa, almond (two), and maple; (ii) *X. fastidiosa* subsp. *multiplex*, PPD and PLS, strains from peach, elm, plum, pigeon grape, sycamore, almond and recently pecan (Melanson et al., 2012); (iii) *X. fastidiosa* subsp. *pauca*, CVC, strains from citrus and probably those from coffee (CLC); (iv) *X. fastidiosa* subsp. *sandy*, strains from *Nerium oleander* (OLS); (v) *X. fastidiosa* subsp. *tasbke*, strains from the ornamental tree *Chitalpa tasbentensis*.

*X. fastidiosa* isolates are genetically very similar, but studies on their biological traits have indicated differences in virulence and symptomatology. As mentioned, taxonomic analyses have identified several subspecies, and phylogenetic analyses of housekeeping genes have shown broad host-based genetic differences. However, results are still inconclusive for genetic differentiation of isolates within subspecies. In a recent study, sequences of nine non-housekeeping genes were used to study 54 *X. fastidiosa* isolates infecting different host plants. Strains could again be divided into the known *X. fastidiosa* subspecies, but also showed new within-subspecies differentiation, including geographic differentiation, and some host-based isolate variation and specificity (Parker et al., 2012).

*Xylella fastidiosa* is a quarantine organism on the EPPO A1 list and EC Annex II/Ia list. For further and extensive data on biology, hosts, geographical distribution and epidemiology see recent reviews (Purcell, 1997; Chatterjee et al., 2008; Janse, 2010; Janse and Obradovic, 2010) and the extensive *X. fastidiosa* website, edited and maintained by A. Purcell and R. Almeida: http://www.cnr.berkeley.edu/xylella/. For diagnostic methods, see EPPO 2005a and Janse et al. (2012).

Hosts. A full host list can be found on http://
www.cnr.berkeley.edu/xylella/. Some hosts relevant for the Mediterranean basin are: Nerium oleander, Platanus occidentalis (sycamore), Quercus spp. (oak), Ulmus americana (elm tree), Ambrosia artimisiifolia (ragweed), Morus alba (white mulberry), Prunus angustifolia (Chickasaw plum) and Ribes spp. In Taiwan a pear leaf scorch was described in 1990 on Pyrus pyrifolia (Japanese pear), cv. Hengshan and P. serotina (Asian pear), which was found to be caused by a bacterium very similar to *X. fastidiosa*, but differing from North and South American strains in serological properties and housekeeping gene sequences (Leu and Su, 1993; Chen et al., 2006). Hosts such as Asian pear were recently introduced in central Europe (Romania) and Japanese pears, or nashi, were planted as a novelty crop in southern Europe especially in the 1980’s. It is not known whether the latter two species have ever been subjected to surveys for *X. fastidiosa*.

**Symptoms and transmission.** For symptoms on different hosts, see http://www.cnr.berkeley.edu/xylella/. In general, early symptoms are a slight chlorosis or bronzing along leaf margin or tip that intensifies and that may become water-soaked before browning and drying. These symptoms are first found on a few branches, then they extend to most or the whole canopy (so-called leaf scorch or scald symptoms). A narrow chlorotic band that becomes especially clear in autumn delineates the affected area. A premature defoliation may take place with new malformed leaves are formed. Fruits can be abnormally shaped and stems may show internal and external discolorations, dieback and abnormal growth, leading to eventual death of the host. Vectors are mainly sharpshooters and froghoppers or spittlebugs (Cicadellidae) that lack a latent period, and have no transstadial or transovarial transmission of the bacterium. The pathogen shows persistence in the vector adults, and the main vectors (for PD unless indicated) are *Philaenus spumarius* (meadow spittlebug), *Graphocephala atropunctata* (blue-green sharpshooter), *Dilobopterus costalimai* (PPD), *H. insolita* (PPD); *Homalodisca vitripennis* (formerly *H. coagulata*) the glassy-winged sharpshooter, *G. versuta* (PPD); *Hordnia circellata*, very efficient; *H. insolita* (PPD), *Oncometopia nigricans*, *O. orbona* (PPD), *Xypbon* (formerly *Carmenecephala*) fulgida (red-headed sharpshooter), CVC vectors in Brazil are *Acrogonia terminalis* that lays eggs externally on the leaves, *Dilobopterus costalimai* and *Oncometopia fascialis*. Local possible vectors for Europe are *Cicadella viridis* and *Philaenus spumarius* (meadow spittlebug) (Fig. 2B). Because of the presence of these potential vectors the establishment of the pathogen in Europe is not strictly connected to introduction of foreign recognized vectors.

**Risks.** *X. fastidiosa* is an emerging threat in the south-west USA, mainly due to recent establishment of the glassy-winged sharpshooter (*GWSS*, *H. vitripennis*), providing much more efficient transmission than local vectors, and leading to very serious outbreaks of PD in grapevine, ALS and OLS. GWSS probably first entered California as eggs deposited in plant tissues. In Central and South America *X. fastidiosa* has become very noxious due to the rapid expansion (most likely via distribution of infected planting material) of CVC in *Citrus*, leading to more than a third of all trees in the area having symptoms of CVC, and CLC in coffee. As to Europe, there are only a few unconfirmed reports of the presence of *X. fastidiosa* in the grapevine in Kosovo (Berisha et al., 1998) and in almond in Turkey (Güldür et al., 2005). Since *X. fastidiosa* has more than 150 hosts and many of them, including *Vitis*, were and are import (often as planting material), risk of introduction (especially in latent form) must not be underestimated. Absence of the diseases caused by *X. fastidiosa* will mainly be due to the absence of suitable vectors. However, introduction of the pathogen and infected vectors with plant material cannot be excluded. Moreover, also local Cicadellidae (see above) could become potential vectors. Therefore, *X. fastidiosa* has the A1 quarantine status in the EPPO region and *H. vitripennis* that has a very large host range and feeds on almond, peach and plum was recently placed on the EPPO alert list. As in the more southern parts of the USA, European *Vitis* varieties are very susceptible to *X. fastidiosa*. This is really a risk if a vector that could survive the winters of southern Europe were introduced, the pathogen would become established in wild hosts (wild and domestic plums and wild cherry are symptomless reservoirs in the USA) and cause spring infections that are most likely to persist over the years. The same risk holds true for *Citrus* (sweet oranges, mandarins, and tangerines) and other hosts, such as almond, plum and peach that are widely grown in southern Europe, especially in the warmer Mediterranean basin, where a disease-favourable combination of warm nights, regular rainfall/high humidity and long growing season, is present. The conclusion is that *X. fastidiosa* is a real threat for Europe, not only for *Vitis* and *Citrus* but also for stone fruits (almond, peach and plum) and oleander (*GWSS* likes to feed on oleander), that is difficult to prevent from entering and difficult to control once established, deserving more attention than up till now. Resistance in European grapes is scarce or even absent. Vector control proved not to be very effective in the USA. Cultural practices to keep plants in optimum condition are of importance, but not sufficient, and the use of avirulent strains for cross-protection is still in its infancy.

**Citrus canker, caused by Xanthomonas citri pv. citri** (*X. axonopodis pv. citri*). General. Asian citrus canker, a spot disease of *Citrus* spp. characterized by corky le-
sions on leaves, fruits and twigs, was first described by Stevens in 1914 and the causal bacterium, now named *Xanthomonas citri* pv. *citri* by Hasse in 1915 in the USA. However, citrus canker had been observed earlier in Asia as it originates most probably from China. Symptom formation and spread of the bacterium are enhanced by the activity of the citrus leaf miner, *Phyllocnistis citrella*, which occurs in nearly all citrus growing areas of the world. In Europe it is established in the Iberian peninsula, Corsica, Italy, Greece and Montenegro (http://www.bladmineerders.nl/minersf/lepidoptera/ramin/phyllocnistis/citrella/citrella.htm). This insect, however, is not a true vector. Grapefruit (*C. paradisi*), Mexican/Key lime (*C. aurantiifolia*), lemon (*C. limon*) and *C. bystrix* are the most susceptible species. *X. citri* pv. *citri*, is a Gram-negative bacterium with one polar flagellum, forming yellow colonies on agar media. Over the years, several related diseases and pathogenic bacteria have been described from *Citrus* and some other Rutaceae spp. in Asia, North and South America:

(i) *X. citri* pv. *citri* (Xcc) (syn. *X. citri* subsp. *citri*, *X. axonopodis* pv. *citri*) formerly named group A or Asiatic strain is the most aggressive form. Other names for this pathogen have been *X. citri*, *X. smithii* subsp. *citri* and *X. campestris* pv. *citri* (A group) (Schaad et al., 2005, 2006; Ah-You et al., 2009). Strains with a different host range, named pathotype A* strains, infecting Mexican lime and Tahiti lime (*C. latifolia*) but not grapefruit (*C. paradisi*) have been reported from Cambodia, Iran, India, Oman and Saudi Arabia. The so-called Aw strains that infect Mexican lime and alemow (*C. macrophylla*) were described from Florida (Ngoc et al., 2009, 2010; Sun et al., 2004).

(ii) *X. citri* pv. *aurantifolii* (syn. *X. axonopodis* pv. *aurantifolii*, *X. fuscans* pv. *aurantifolii*), formerly named *X. citri* group B or cancerosis B, group C or Mexican lime cancerosis, and group D or *Citrus* bacteriosis, causes a relatively mild disease in Mexico and South America. It was also reported from Swingle citromelo rootstock (*C. paradisi* x *Poncirus trifolata*) from Brazil (Jaciani et al., 2009).

(iii) *X. citri* pv. *bilvae* (syn. *X. campestris* pv. *bilvae*), is an ill-defined pathogen, causing shot-hole disease and canker on Bael (*Aegle marmelos*), a member of the family Rutaceae described in India in 1953 (Ngoc et al., 2010).

(iv) *X. alfalfa* subsp. *citrumeloni* (syn. *X. campestris* pv. *citrumelo*), is the cause of citrus bacterial spot, formerly named group E strains in Florida, USA (Schaad et al., 2005).

Only Xcc, the agent of Asian citrus canker (A, A* and Aw strains) is described here. It is a quarantine organism on the EPPO A1 list and EC Annex II/1a list. Main reference sources are Brunings and Gabriel (2003), Das (2003), Gottwald et al. (2002), Rogers et al. (2010), Lowe (2010) and, Ngoc et al., (2009), Golmohammadi et al. (2007) and EPPO (2005b) for diagnostic methods.

**Geographical distribution** Xcc originates from and is widespread in Asia, including Georgia, Iran, Iraq, Oman, Saudi Arabia, UAE and Yemen. Australia (eradicated), Argentina, Belau, Brazil, Caroline Islands, Cocos Islands, Comoros, Congo Democratic Republic, Ivory Coast, Fiji, Gabon, Madagascar, Mauritius, Mozambique (eradicated), Netherlands Antilles, New Zealand (eradicated), Micronesia, Palau, Papua New Guinea, Paraguay, Reunion, Seychelles, South Africa (eradicated), Uruguay, USA (CABI/EPPO, 2006). There are recent reports from Somalia (Balestra et al., 2008) Mali (Traoré et al., 2009) and Ethiopia (A* strains, Derso et al., 2009).

**Hosts.** Cultivated hosts are Aegle marmelos (golden apple), Casimiroa edulis (white sapote), Citrus aurantiifolia (lime), *C. aurantium* (sour orange), *C. bystrix* (mauritius bitter orange), *C. junos* (yuzu), *C. limetta* (sweet lemon tree), *C. limon* (lemon), *C. madurensis* (calamondin), *C. maxima* (pummelo), *C. medica* (citron), *C. natsudaidai* (natsudaidai), *C. reshni* (Cleopatra mandarin), *C. reticulata* (mandarin), *C. reticulata* x *Poncirus trifolata* (citrusmelo), *C. sinensis* (navel orange), *C. sunki* (sour mandarin), *C. tankan* (tankan mandarin), *Citrus unshiu* (satsuma), *Citrus x paradoisi* (grapefruit), *Eremocitrus glauca* (Australian desert lime), *Limonia acidissima* (elephant apple), *Poncirus trifolata* (trifoliate orange or Japanese bitter orange). Minor hosts are Fortunella japonica (round kumquat) and *F. margarita* (oval kumquat). Wild hosts are *Ageratum coryzoides* (billy goat weed), *Severinia buxifolia* (box orange or bokthorn) and *Swinglea glutinosa* (= *Aegle decandra*, *Limonia glutinosa*).

**Symptoms and transmission.** (For pictures of symptoms and of the citrus leaf miner, *P. citrella*, see http://www.plantmanagementnetwork.org/pub/php/review/citruscanker/). Small spots, first visible on the upper leaf blade, appear on the leaves, shoots, twigs and fruits, to become raised pustules or blister-like eruptions (Fig. 3A). With time, the lesions increase size (up to 10 mm) and turn brown and necrotic with a depressed centre, and are sometimes surrounded by a yellow halo. On the fruits, the lesions can be mistaken for scale insects (e.g. the California red scale, *Aonidiella aurantii*). The bacterium is a wound parasite and, as mentioned, the citrus leaf miner (*P. citrella*) contributes to disease spread and severity. Citrus canker is especially epidemic and damaging on seedlings and young trees, especially after storms (hurricanes) under warm weather conditions, but because of dependence on these weather conditions epidemics are sporadic. Full-grown trees show much less disease and damage (Goto, 1992). The bacterium can survive in a latent form in and on diseased shoots and discoloured bark tissue of the trunk, and may reoccur suddenly after several years (sometimes even as long as 10 years).

**Risks and damage.** Heavy losses were reported in epidemics, due to premature fruit drop and fruits with
spots that cannot be marketed or start rotting, thus must be destroyed. Furthermore, quarantine measures such as burning of trees and destruction of fruits may add to these losses (Goto, 1992). In severe cases, almost 100% of the fruits and leaves of young, susceptible trees may be infected and the plant growth is delayed for a number of years. The direct Government costs in the USA for the eradication activities from 1995 to 2006 were calculated to be more than $1.3 billion. From 2006 to 2009, together with costs for the control of Citrus huanglongbing the amount was $90 million. The citrus acreage in Florida decreased since 1996 by ca. 33% (Lowe, 2010). In January 2006, the USDA determined that canker had become so widespread in Florida that eradication was no longer feasible. Use of healthy planting material and use of other measures, including weather forecasting, in an integrated way have been applied in the control of Citrus canker with some success. Resistance has been found especially in *C. mitius* (calamondin) and *Fortunella* (kumquat). *C. reticulata* (mandarin) is tolerant.

One should remember that epidemics of citrus canker on mature plants are sporadic and very dependent on weather conditions (rainstorms, hurricanes), which are less prevalent in the Mediterranean basin. On the other hand, it should also be realized that the citrus leaf miner is already widespread in this area. Seed transmission has not been observed. Long distance dispersal is by infected planting material or infected fruits. In the past, ornamental *Citrus* have been imported from Asia.

![Symptoms of bacterial diseases](image-url)
to Europe and Xcc has been intercepted on this material (author’s personal experience). However, the risk of dispersal by infected fruit was evaluated to be very small or absent if fruits are disinfected before shipment (Gottwald et al., 2009). Without disinfection, survival chances in symptomatic fruits are apparently also small (Shiotani et al., 2009), but not impossible, as shown by the identification of Xcc on imported fruits by the Dutch Plant Protection Service (author’s personal experience). Since Xcc is endemic and spreading in countries surrounding the Mediterranean basin, some of which have also huanglongbing problems, and the citrus leaf miner is widespread in the area, the conclusion is that this pathogen is a real and emerging threat.

BACTERIAL DISEASES WITH AN EMERGING CHARACTER, BUT NOT YET PRESENT IN EUROPE OR THE MEDITERRANEAN BASIN

Black spot of mango - Xanthomonas citri pv. mangiferaeindicae. A leaf spot and canker disease of mango (Mangifera indica) called bacterial black spot, was described in South Africa by Doidge (1915) who named the causal agent Bacillus mangiferae (later also named Xanthomonas mangiferaeindicae, and X. campestris (axonopodis) pv. mangiferaeindicae]. Ah-You et al. (2007, 2009) showed that this bacterium is much related to a pathogen from cashew (Anacardium occidentale) and named the two bacteria X. citri subsp. mangiferaeindicae and X. citri pv. anacardi, respectively. Both mango and cashew belong to the family Anacardiaceae. Symptoms of black spot start as small water-soaked spots that become later raised and necrotic, sometimes surrounded by a narrow yellow halo. On the fruits the water-soaked spots become star-shaped and crack and often show exuding gum. Severe infection under influence of rainstorms may lead to premature leaf and fruit drop, twig cankers and twig death (Fig. 3 B-E). Other (rare) hosts are ambarrella (Spondias dulcis, syn. S. cytherea) and Brazilian pepper (Schinus terebinthifolius), both belonging to the Anacardiaceae. When other diseases and pests are controlled, black spot is a limiting disease to mango production, because it is very difficult to contain. In most susceptible cultivars up to 100% fruit loss may occur. Many commercial cultivars are very susceptible. In 1996 and 1997, severe black spot epidemics were observed in many mango-growing areas of South Africa, causing almost 100% fruit loss on the most susceptible cultivars and ca. $1 million economic loss. There is production of mango in Europe and the Mediterranean basin (e.g. Spain, Italy, Israel and Portugal). Black spot occurs in Australia, Comoro Islands, many areas in southern and eastern Africa and Asia, Mauritius, New Caledonia, Reunion, Taiwan, and the United Arab Emirates (Gagnevin and Pruvo, 2001). Discrimination of strains from mango and some related hosts and from different geographic origin (Asia, Africa and Brazil) was possible using RFLP (Gagnevin et al., 1997). Long distance dissemination of the pathogen is thought to be by infected planting material. Epiphytic/endophytic populations of the pathogen occur (Pruvo et al., 2009). Seed transmission has not been demonstrated.

Bacterial blight of pomegranate - Xanthomonas axonopodis pv. punicae. A bacterial disease on pomegranate (Punica granatum, family Lythraceae) was observed for the first time in 1952 in New Delhi, India and described by Hingorani and Singh (1959) as being induced by Xanthomonas punicae (later classified as X. campestris pv. punicae and X. axonopodis pv. punicae). First symptoms are water-soaked spots on leaves and fruits (Fig. 3F). On the leaves, spots become necrotic and, when they coalesce, severe leaf drop may occur. Fruits may crack and drop. Black necrotic spots occur on branches, that become weak and may split. Pomegranate is produced mainly by India (50%), Iran (35%), some of the former Soviet Union states and in the Mediterranean basin, especially in Spain (2.5%), Morocco, Egypt and Turkey. Bacterial blight has developed into a very serious disease in India, where it causes very heavy losses (up to 100%) in many pomegranate-growing areas. (Kumar et al., 2006). Dispersal at a site is by rain splash, insects and tools, whereas over long distances is by infected plant material. Like X. citri pv. citri and pv. mangiferaeindicae, stormy weather conditions are important for epidemic outbreaks. X. a. pv. punicae overwinters in infected leaves of neem (Azadirachta indica, family Meliaceae) planted along pomegranate fields (Dhasandar et al., 2004; Sharma et al., 2008; Kumar et al., 2009) Since these former two pathogens have already spread from the more eastern parts of Asia, X. a. pv. punicae can be seen also as a potential threat that may soon show its presence closer to the Mediterranean basin.

Bacterial blight of guava - Erwinia psidii. A vascular disease of guava (Psidium guajava) was reported as bacterial blight from Brazil and the causal agent denoted Erwinia psidii (Neto et al., 1987), was held responsible for the outbreaks in the main production areas of southeastern and central regions of the country (Tokeshi et al., 1980). Symptoms are local and systemic. Leaves show large necrotic lesions along the main veins (Fig. 3G) and at the margins (scorching), or small water-soaked spots, sometimes with a chlorotic halo, that later become necrotic and coalesce causing leaf drop. When bacteria reach the xylem they spread into branches, trunk and roots. In severe cases, trees are defoliated and die. Symptoms on the fruits are not common. This disease could be of importance to guava-growing countries such as Egypt that produces yearly some 230,000 tons of guava fruits, being the fifth producer in the world after India, Pak-
istan, Brazil and Mexico. In Brazil, pathogen dispersal often occurs with contaminated planting material (Marques et al., 2007; Teixeira et al., 2008b).

Bacterial spot of passion fruit - *Xanthomonas campestris pv. passiflorae*. A destructive disease of cultivated passion fruit (*Passiflora edulis* and *P. edulis* var. *flavicarpa*), was described by Pereira in 1969 in Brazil (El Tassa, 2002). This disease is characterized by the presence of water-soaked, greasy lesions of irregular shape on the leaves, surrounded by chlorotic areas that result in extensive necrosis when they coalesce (Fig. 3H). Fruits display greasy spots that make them unsuitable for consumption and industrial processing. This disease has an emerging character in Brazil, where ca. 25,000 ha are given over to passion fruit and has become a major problem to production. Hosts are *Passiflora alata*, *P. amethystina*, *P. coccinea*, *P. edulis*, *P. edulis* var. *flavicarpa*, *P. maliformis*, *P. nitida* and *P. serrato-digita* (Neto et al., 1984; Torres Filho and Ponte, 1994; Gonçalves and Rosato, 2000; Lopes et al., 2006).

**BACTERIAL DISEASES/PATHOGENS WITH AN EMERGING CHARACTER, ALREADY OCCURRING IN EUROPE OR THE MEDITERRANEAN BASIN**

Almond witches’ broom - *Candidatus Phytoplasma phoenicium*. This devastating disease of almond (*Prunus amygdalus*), shows typical witches’ broom symptoms, i.e. small yellow leaves on proliferating shoots that wither and die in later stages (Fig. 4.A-C). It

![Symptoms of witches' broom in almond. (A) Mature branches (B) and tree (C) caused by "Candidatus Phytoplasma phoenicium" (courtesy E. Choueiri). (D) Malformations of tomato (*Lycopersicon esculentum*), caused by potato stolbur, "Candidatus phytoplasma solani" (Source EPPO).]
was reported from Lebanon by Choueiri et al. (2001), and was classified and named by Verdin et al. (2003). Candidatus Phytoplasma phoenicium belongs to the pigeon pea witches' broom group (16SrIX). In a few years time, more than 100,000 trees were killed in different areas in Lebanon. This phytoplasma was also found on peach and nectarine (Abou-Jawdah et al., 2009) and was recently also reported from Iran on almond (Zirak et al., 2009) and on GF-677 (Prunus amygdalus x Prunus persica) (Salehi et al., 2011). Vectors are suspected to be leafhopper which have not yet been identified (Abou-Jawdah et al., 2011). This pathogen is not included in quarantine lists, but certainly deserves attention as an emerging threat for almond. Stem cutting culture with thermotherapy was successfully used for regeneration of phytoplasma-free plantlets of almond (Chalak et al., 2011). Recently Davis et al. (2010) have reported a phytoplasma closely related to Ca. P. phoenicium as the cause of witches' broom affecting Juniperus occidentalis (western juniper) in Oregon (USA).

Potato stolbur - Candidatus Phytoplasma solani. Ca. Phytoplasma solani is a non-culturable, insect-transmitted bacterium that induces bushy growth and malformations in solanaceous (Fig. 4D) and other hosts (EPPO/CABI 1996) and is on the EPPO A2 quarantine list. It belongs to the so-called Aster yellows or stolbur (16Sr-XII-A) phytoplasma group, the latter having a very wide host range (‘Bois noir’ of grapevine is caused by a phytoplasma of the same group). Severe outbreaks occur under dry weather conditions when vector populations develop explosively on wild hosts. In some years, substantial damage was reported from south-east Europe and Russia. Spreading in potato is slow. Vectors are mainly leafhoppers of the family Cixiidae, the most important of which are the polyfagous Halyomides obsouletus, the true bug Lygus pratensis (family Miridae) and some other cicadas, such as Macrosteles quadrifimbriatus. Potato stolbur has been reported from Austria, Bulgaria, Czech Republic, Germany, France, Greece, Hungary, Italy (Berger et al., 2009), Israel, Poland, Romania, Russia, Serbia, Switzerland, Turkey and Ukraine. Main hosts for the bacterium are potato, tomato, eggplant, pepper and weeds such as black nightshade, bindweeds (Convolvulus arvensis, Calystegia sepium), stinging nettle, Cardaria or Lepidium and Lavandula. Larvae of cicadellids feed on the roots from which they can acquire and transmit the bacterium. Potato stolbur might spread more, when the climate warms up. H. obsouletus recently spread in Germany from the Moselle valley and by the end of 2009 stolbur phytoplasma was detected in association with bindweed in Rheinland-Pfalz (EPPO Reporting Service 2010/155). In eastern Europe (Bulgaria, Romania, Serbia), Ca. phytoplasma solani has been causing for many years a disease transmitted by Reptalus panzeri (Jovic et al., 2009) called ‘corn reddening’, responsible for 10-90% crop losses. Diseased plants show a red discolouration of stems and main leaf vein and abnormal ears. In Romania the cultivation of potato cv. Lady Rosetta was stopped in disease-sensitive areas, severe losses occurred from 2006-2008, in 2008 circa 75 ha were infected, of which 45 ha suffered total crop loss. Also from Russia, the region Severe damages were reported in 2003 from Russia in an area around Krasnodar (www.copphytoplasma.eu) as well as in the Czech Republic (Navrtil et al., 2009). Ca. P. solani does not persist much in potatoes during storage, so that only few diseased plants develop from infected tubers. Late infections do not influence the yield. The disease should not be a problem in a well-tended crop, were healthy, certified planting material is used and where a careful weed and vector control takes place.

Zebra chip disease of potato or psyllid yellows of solanaceae and carrot - Candidatus Liberibacter solanacearum (synonym Ca. Liberibacter psylllaurous). This non-culturable bacterium is closely related to earlier mentioned Liberibacter species occurring in Citrus, and has been placed on the EPPO alert list (http://www.eppo.org/QUARANTINE/Alert_List/bacteria/Liberibacter_psylllaurous.htm). Ca. Liberibacter solanacearum (CaLS) was first described in New Zealand in 2008 (Liefert et al., 2011) as Ca. L. psylllaurous, and was later found also the USA (Hansen et al., 2008) where it has an emerging character. In the USA it has been reported from Texas in 2010 (French-Monar et al., 2010) and in 2011 from Idaho, Washington state and Oregon (Crosslin et al., 2012). It also occurs in Guatemala, Honduras, Canada and, in tomato, in Mexico (Munyaneza et al., 2010c). The tomato/potato psyllid Bactericera cockerelli is a vector that feeds mainly on solanaceous hosts, but it occurs on many other hosts and causes so-called psyllid yellows. It is present in North and Central America and, recently (2000), it appeared in New Zealand. The bacterium can spread with potato seed, tomato plantlets and fruits. Seed transmission has not been reported. Apparently, CaLS is quite versatile and adapted to diverse climatic regions such as desert, steppe, Mediterranean, marine coast, humid continental and humid subtropical. Its hosts are Capsicum annuum, C. frutescens, Lycopersicon esculentum, Physalis peruviana, Solanum betaceum and S. tuberosum (Fig. 5A). The disease is called Zebra chip because characteristic brown stripes develop when potato tubers are cooked (Fig. 5B). The complete genome sequence of CaLS has been determined (Lin et al., 2011). More recent records of CaLS in carrot (Daucus carota) are from: (i) Finland where the bacterium is transmitted by the psyllid Trioza apicalis and causes, together with its vector, substantial crop losses. Infected plants show typical leaf curling, yellow and purple discoloration of the leaves, stunting of roots and shoots, and proliferation of secondary roots (Munyaneza et al., 2010a, 2010b); (ii)
Canary Islands (Tenerife, since 2009) where the bacterium is apparently transmitted by the psyllid Bactericera trigonica (Alfaro-Fernández et al., 2012a). Subsequently it has also been reported from mainland Spain, where it infects celery (Apium graveolens) in several regions, also in mixed infections with Ca. P. solani and, strangely enough, with the bacterium Spiroplasma citri (Alfaro-Fernández et al., 2012b); (iii) Norway (since 2011) where CaLS causes extensive damage to commercial fields with an incidence of 10-100% (Munyaneza et al., 2012a); (iv) Sweden, since 2011 (Munyaneza et al., 2012b). Until now, CaLS has not been reported from potato, probably because the vector B. cockerelli does not occur in Europe.

Other phytoplasmas that could become a threat will not be treated in this review, apart from their mention in Table 1. For a recent overview, see COST Action FA0807 “Integrated Management of Phytoplasma Epidemics in Different Crop Systems”

Harmless (?) “Candidatus Liberibacter europaeus” found in Cacopsylla pyri. An apparent harmless Liberibacter species, named “Candidatus L. europaeus” was reported from Italy to occur in a high percentage of the psyllid Cacopsylla pyri that thrives on pear trees (Pyrus communis). The bacterium could be experimentally transmitted by the psyllid to pear but, apparently, it remains until now non-pathogenic (Raddadi et al., 2011).

Bacterial fruit blotch of Cucurbitaceae - Acidovorax citrulli (syn. A. avenae subsp. citrulli). Symptoms of bacterial fruit blotch are water-soaked leaf spots on seedlings that may coalesce in later stages and kill the plantlets. Mature plants show reddish brown streaks along main veins and, most characteristically, dark green spots on the fruit surface (watermelon, Fig. 5C) or inside the fruit (melon and pumpkin) which are accompanied by rind depressions and cracks from which bacteria may ooze. Hosts are watermelon (Citrullus lanatus) and melon (Cucumis melo), but also Cucumis sativus (cucumber), Cucurbita pepo (squash), and C. moschata, which show symptoms only on the leaves, and wild cucurbits, e.g. Citrullus lanatus var. citroides. Deng et al. (2010) reported A. citrulli outbreaks on Piper betle (betel vine, Piperaceae) in Taiwan. Strain diversity, i.e. mild strains on hosts other than watermelon and more aggressive strains from watermelon, has been observed in the USA (Walcott et al., 2004). Recent outbreaks of bacterial fruit blotch were reported in Europe from Greece (2005), Hungary (2007, apparently with watermelon transplants from Turkey), Israel (2000 and 2003, melon and watermelon), Turkey (1995, Marmara region and 2005 Mediterranean region) and Italy (2009, melon) [see also: http://www.eppo.org/QUARANTINE/Alert_List/bacteria/Acidovorax_citrulli.htm; Holeva (2009); Hopkins et al. (2001); Hopkins and Thomson (2002)]. The bacterium is mainly spread with seed and

Table 1. Emerging phytoplasmas reported at the Meeting on “Emerging phytoplasma diseases of stone fruits and other crops and their possible impact on EU countries”, held in 2011 in Istanbul, Turkey.

<table>
<thead>
<tr>
<th>Name</th>
<th>16S rRNA Group</th>
<th>Latin name</th>
<th>Country where reported</th>
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<tbody>
<tr>
<td>Almond witches’ broom</td>
<td>16SrIX-B</td>
<td>“Ca. P. phoenicium”</td>
<td>Lebanon</td>
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<tr>
<td>Cassava frog skin</td>
<td>16SrIII-L</td>
<td></td>
<td>Colombia</td>
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<tr>
<td>Grapevine yellows</td>
<td>16SrI-B</td>
<td>“Ca. P. asteris”</td>
<td>Italy, South Africa</td>
</tr>
<tr>
<td>Grapevine yellows</td>
<td>16SrVII-A</td>
<td>“Ca. P. fraxini”</td>
<td>Chile</td>
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<tr>
<td>Grapevine yellows</td>
<td>16SrXII</td>
<td>“Ca. P. ziziphi”</td>
<td>Iran</td>
</tr>
<tr>
<td>Lime witches’ broom</td>
<td>16SrI-B</td>
<td>“Ca. P. aurantifolia”</td>
<td>China, Korea, Italy</td>
</tr>
<tr>
<td>Potato purple top wilt</td>
<td>16SrVI-A</td>
<td>“Ca. P. trifolii”</td>
<td>USA</td>
</tr>
<tr>
<td>Potato purple top wilt</td>
<td>16SrVIII-A</td>
<td>“Ca. P. americanum”</td>
<td>USA</td>
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planting material and the disease it elicits may result in extensive damage and losses (40-100% in USA and Brazil). Control measures to be implemented are: (i) use of healthy tested seed (seed treatment has not been effective up to now); (ii) seed test by a check on 10,000 seedlings/lot in a greenhouse (so-called sweaterbox method); (iii) inspection of plants during the growing season and destruction of infected plant material. In recent years also PCR screening tests have been developed (Bahar et al., 2008; Jing et al., 2011; Woudt et al., 2012). Positive results of all these methods still need to be verified by isolation of the pathogen. Perhaps RT-PCR in combination with MALDI-TOF (matrix assisted laser desorption ionization time-of-flight mass spectrometry) could be used to exclude false positives (Wang et al., 2012).

A new strain of a potato stem rot bacterium belonging to the genus Dickeya, provisionally named ‘D. solani’. Bacterial stem rot of potato, caused by different ‘cold tolerant’ biovars of Erwinia chrysanthemi, now named Dickeya dianthicola and D. chrysanthemi, has been reported from many European countries since the 1970s, and is regarded as a quality disease (Janse and Ruisien, 1988; Toth et al., 2011). A taxonomic revision placed Erwinia chrysanthemi biovars into six species of the newly created genus Dickeya, namely D. chrysanthemi biovar chrysanthemi and biovar parthenii, D. dadanti, D. dianthicola, D. dieffenbachiae, D. paradisiaca and D. zae (Samson et al., 2005). Among them there are ‘warm tolerant’ species like D. dadanti and D. zae that were found not only in potato in hot climates, but also in many ornamental plants, often grown in greenhouses in temperate regions (Janse and Ruisien, 1988; Janse and Scheepens, 1990; Samson et al., 1990). Recently a new ‘warm tolerant’ variant of Dickeya was observed, first in the Netherlands since 2000 (Czajkowski, 2009a, 2009b, 2011; J. Van Vaerenbergh, personal communication), then in Poland, Belgium, Finland, France, Israel and most recently also in the UK (2010), Denmark and Sweden (2011). This variant is virulent under warm climatic conditions and is closely related, but not similar to D. dadanti, it has been provisionally denoted D. solani (Czajkowski, 2011). A recent taxonomic study indicates that it is not certain whether this variant is indeed a new species (Van Vaerenbergh et al., 2012). Warm tolerant Dickeya spp. have been isolated from surface waters and D. solani, that shows a strong clonal character, in the Netherlands also from hyacinth and iris in the bulb production area where no potatoes are grown (Toth et al., 2011; Van Vaerenbergh et al., 2012). A theory is that a warm tolerant species (D. dadanti or zae) escaped from greenhouse wastewater disposals to surface water and mutated to a form that clonally spread in hyacinth, iris and potato. As with the other Dickeya and Pectobacterium species in potato, effective control is possible and regulated via certification schemes. It should be based on the use of tested, healthy seed and careful cultural practices (careful and dry harvesting, proper storage and ventilation, avoiding of desprouting and cutting seed, hygiene on the farm, etc).

Stem rot and leaf spot of maize and ‘center rot’ of onion - Pantoea ananatis. Pantoea ananatis can cause a number of diseases on different hosts, e.g. stem rot and leaf spot of maize, internal fruit rot of melon and pink disease of pineapple. It also infects onion (Allium cepa) inducing ‘center rot’, outbreaks of which have been reported since 1997 in the USA, and was isolated from onion seed in South Africa (Goszczynska, 2006). Seed transmission was confirmed in the USA (Walcott et al., 2002). The thrips Franklinella fusca can transmit the bacterium which apparently survives as a saprophyte on many weed and crop hosts (Gitaitis et al., 2003).

Stewart’s disease or bacterial wilt - Pantoea stewartii subsp. stewartii. This vascular disease, characterized by white stripes on the host leaves, is widespread in North America. The main host is maize, especially sugar maize, but also the so-called ‘dent’, ‘flint’, ‘flour’ and popcorn types. The bacterium is mainly spread by Chaetocnema pulicaria, the corn flea beetle. P. subsp. stewartii and bacterial wilt have been reported from, but are not established in Austria, Greece, Italy, Poland, Romania and European Russia. Main source of introduction is contaminated seed from North America, but pathogen and disease disappear apparently some years after introduction, due to absence of the vector beetle in Europe. Local vectors do not transmit the bacterium as far as is known. In Italy, substantial damage was reported in the years 1940-1950, and some reoccurrence, but not very damaging in 1983-1984 (Mazzuchi, 1984; www.eppo.org/QUARANTINE/bacteria/Pantoea_stewartii/ERWIST_ds.pdf). Further introductions could occur in the future, but will remain relatively harmless as long as the vector is not introduced or local flea beetles, as potential vectors, do not acquire and transmit the pathogen.

New outbreaks of bacterial (blight) canker of kiwifruit - Pseudomonas syringae pv. actinidiae. Bacterial canker of kiwifruit was first observed in Japan in 1984 (Takikawa et al., 1989). It was also reported from China where, as we know now, it occurred already as early as 1984/1985 (Liang et al., 2000). Subsequently it was found in Korea and Italy (1992). Hosts are green kiwi (Actinidia deliciosa), yellow kiwi (Actinidia chinensis), A. arguta, and A. kolomikta. Since 2008 bacterial canker outbreaks have repeatedly been observed in Italy (Emilia-Romagna, Lazio, Piemonte and Veneto) on green kiwi, causing a mild leaf spot and some canker formation on branches and trunk. There was also a re-
port from Iran in 1994 and a recent occurrence of the so-called Asian or mild strain in Australia (EPPO Reporting Service 2011/130). In spring and autumn of 2008 and winter 2008/9, however, severe outbreaks occurred on yellow kiwi characterized by wilting, necrosis, severe leaf spot and canker formation (Fig. 6A-C), especially the cvs Hort 16A and Jin Tao cultivated in central Italy (Latina province). Recently, severe outbreaks have also occurred on green kiwi cv. Hayward (Ferrante and Scortichini 2010) and the disease has spread to Calabria, Campania, and Friuli-Venezia Giulia regions in 2011 (EPPO Reporting Service 2011/131). *P. s. pv. actinidiae* was recently observed in yellow and green kiwi in France [severe form found in the Rhône-Alp area in 2010 (EPPO Reporting Service 2012/002)], Spain (Abelleira *et al*., 2011; Balestra *et al*., 2010), Portugal (Balestra *et al*., 2010), Switzerland (2011), Chile (2011) (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm) and Turkey, where it is present since 2009 in the Black Sea area (Bastas and Karakaya, 2012). Interestingly, the severe form is also known to occur since 2010 in New Zealand and has spread on the North and South Island (Everett *et al*., 2011; Young, 2012). In this country another mild strain causing only leaf spots has also been observed, which may be not *P. s. pv. actinidiae* (Vanneste *et al*., 2010).

The Asian mild strain was recently reported to be present in Australia (EPPO Reporting Service 2011/130). A PCR test for identification and epidemiological studies was developed by Vanneste *et al*. (2010). Epidemics of bacterial blight of kiwi occur usually after frost damage. In Japan and Korea bacterial blight is the most limiting

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**Fig. 6.** A-C. Symptoms induced by an aggressive strain of *Psudomonas syringae pv. actinidiae* on yellow kiwifruit (*Actinidia chinensis*) in Italy. Wilting and necrosis (A), necrotic leaf spots (B), canker with a reddish ooze (C) (Courtesy M. Scortichini). D-F. Symptoms induced by *Psudomonas syringae pv. aesculi* on Horse chestnut (*Aesculus hippocastanum*). Severe wilting and yellowing (D), red ooze from a canker (E), extensive cankers on a branch (F) (Courtesy Aesculaap, NL). G. Leaf spots with a yellow halo and ‘shotholes’ on cherry laurel (*Prunus laurocerasus*) caused by *Xanthomonas arboricola pv. pruni* (Source Naktuinbouw, NL).
factor to kiwi crops, although mild strains appear to occur there. From Italy in recent years more than 2 million euro damage was reported. In a recent taxonomic study, with extensive sequence analysis of the diverse strains, Mazzaglia et al. (2012) came to the conclusion that severe strains from Europe and New Zealand indeed differ from the mild strains that occur in Japan, Korea and were also present in Italy in the past, and that are similar to strains from China, the country where the bacterium probably originates. They also showed that New Zealand and European strains differ to a small extent and both are more close to the Chinese strains, implicating a possible origin of these strains in China, probably imported with planting material from that country. Due to the emerging character of the new severe strains of \textit{P. syringae pv. actinidiae}, EPPO has placed it on its alert list (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm).

\textbf{An emerging disease in forestry: Bleeding canker of horse chestnut - \textit{Pseudomonas syringae pv. aesculi}.}\n
Since 2002 an emerging bacterial disease, named bleeding canker of horse chestnut (\textit{Aesculus} spp., especially \textit{A. hippocastanum}) was observed first in the Netherlands, then in Belgium, France, Germany and the UK. It is thought that the disease had longer been present in the countries concerned, with an incidence of 40-60%, but that symptoms were often mistaken for those of fungal diseases. Progress of the disease was rapid in the countries concerned, with an incidence of 40-60%, and were also present in Italy in the past, and that are similar to strains from China, the country where the bacterium probably originates. They also showed that New Zealand and European strains differ to a small extent and both are more close to the Chinese strains, implicating a possible origin of these strains in China, probably imported with planting material from that country. Due to the emerging character of the new severe strains of \textit{P. syringae pv. actinidiae}, EPPO has placed it on its alert list (http://www.eppo.int/QUARANTINE/Alert_List/bacteria/P_syringae_pv_actinidiae.htm).

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\textbf{Spreading of bacterial canker and leaf spots of stone fruits - \textit{Xanthomonas axonopodis pv. pruni}.}\n
Recent outbreaks of bacterial canker induced by the quarantine pathogen \textit{Xanthomonas axonopodis pv. pruni} on peach, nectarine (\textit{P. persica var. nectarina}), apricot, cherry and/or plum have been reported from Slovenia (1994), France (1995), Spain (1999) and Iran (2005). The disease is established in Austria, Bulgaria, Italy, Moldova, Montenegro, Russian Federation and Ukraine. On peach, small cankers on twigs and leaf spots are formed; on plum and apricot, holdover cankers on trunk and larger branches are the main problem. On cherry, fruit infection is most damaging (Roselló et al., 2012; Stefani, 2010). \textit{X. a. pv. pruni} would be a risk for north-west Europe should climate change towards higher temperatures continue. The bacterium was identified from plum planting material originating from Asia in 1994 and spread to peach, with an outbreak in 1994 and further ones in later years (Seljak et al., 2001). In France, a severe outbreak took place in 2000 on peach and nectarine (EPPO reporting service 2006/235). \textit{X. a. pv. pruni} was reported from Hungary in propagating material of plum (2004), then in an apricot orchard (Nemeth, 2007). In Italy severe outbreaks occurred in the 1990s on Japanese plum (\textit{P. salicina}) and nectarine, after introduction of the very susceptible cv. Calita. A first report of \textit{X. a. pv. pruni} infection to \textit{Prunus laurocerasus} in a nursery in Toscany was also from Italy in 2005. In Switzerland, the first record was in an apricot orchard in 2005 and in two Japanese plum orchards in 2009, near Martigny (Pothier et al., 2009). In the Netherlands, a first outbreak in \textit{P. laurocerasus} occurred in 2008, in the west of the country, where plants showed shoot-hole symptoms in the leaves (Fig. 6G) (EPPO Reporting Service 2009/178). Infections were again identified in different nurseries in 2009-2011 (Bergsma-Vlami et al., 2012). In Spain the bacterium was found on peach in 1999, further outbreaks in almond in 2006 and 2009 in Valencia and Aragon (Palacio-Bielsa et al., 2010, Roselló et al., 2012). Temperatures of 15-28°C, heavy rain and wind in springtime stimulate epidemics. The economic impact of \textit{X. a. pv. pruni} consists of reduced quality and marketability of fruits and reduced productivity of the trees, as well as higher production costs.

\textbf{Bacterial leaf spot of poinsettia - \textit{Xanthomonas axonopodis pv. poinsettii cola}.}\n
This bacterium was originally found in poinsettia (\textit{Euphorbia pulcherrima}) in India (Patel et al., 1951), then reported from Florida (USA) (Chase, 1985). Other Euphorbiaceae are susceptible, such as \textit{E. heterophylla} (wild poinsettia, mainly occurring in North America, but now widespread in Italy), \textit{E. milii} (crown-of-thorns), \textit{Codiaeum variegatum} (crotone) and \textit{Manihot esculenta} (cassava) (Chase, 1985; CABI, 2011). \textit{E. pulcherrima} originates from Mexico and is a very popular Christmas pot plant in European countries, on which \textit{X. a. pv. poinsettii cola} causes brown to black leaf spots, sometimes surrounded by a yellow halo. Over time the spots may coalesce and the leaves turn completely yellow and drop, severely impairing the commercial value of the plants (Wohanka, 2004). The disease occurs in Cocos Islands (territory of Australia), the Philippines (Quimio, 1974), Taiwan (Lee et al.,
Rapid and reliable diagnosis remains a key issue, as emerging bacterial diseases: can be considered important in the management of Liberibacter spp. The following further measures control measures for emerging diseases have been listed. (2006b).

America and Africa. Identification methods have been from other European countries, but also from South some of the infected countries was not only imported identified. However, It is known that mother material in places of production, Perminow eradicated (Dreo 2003, 2007 in one glasshouse, eradicated), Nethertria in (2007, in one glasshouse, eradicated), Czech Re-2003 (Wohanka, 2004). Further reports are from Aus-1979) [see also EPPO (2006)]., 2006b), Venezuela (Hernández A,, 2006b), First report of citrus canker caused by Pseudomonas syringae pv. actinidiae in Spain. Plant Disease 95: 1583.


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CONCLUDING REMARKS

A number of specific and general prevention and control measures for emerging diseases have been listed under Liberibacter spp. The following further measures can be considered important in the management of emerging bacterial diseases:

- Ornamental and wild hosts may play an important role in spreading diseases and maintaining the pathogen and its vectors in the environment. These plants should be included in surveys.

- Rapid and reliable diagnosis remains a key issue, as well as breeding for resistance. All pathogens men-
tioned are emerging threats, with real risks of intro-
duction and in some cases closely approaching or already present in the Mediterranean basin and/or Europe.

- Introduction of susceptible wild hosts and suscepti-
ble cultivars of cultivated hosts must be avoided, as they often lead to introduction of the pathogen or to outbreaks due to a ‘jump’ of local pathogen strains to the very susceptible cultivar. Introduction of vectors should also be prevented as history teaches that the vector(s) appear first and the pathogen a few years later. Examples of these facts are presented in the de-
scription of the various disease-pathogen combina-
tions.

- An efficient prevention and control strategy of dis-

eas described in this review should be based on so-
called pathway protection. This means it should be based on regulatory systems ensuring importation of plant material free of all quarantine and regulated non-quarantine pests and practically free of non-reg-
ulated pests, where the following conditions should be maintained and controlled: (i) place of production should have integrated pest management practices;

(ii) pre-export treatments if necessary; (iii) clean growing media associated with plants; (iv) proper waste management; (v) availability of expert diagnostic services; (vi) inspections at growing sites and (vii) clean packing practices.

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