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Canker and decline diseases caused by soil- and airborne *Phytophthora* species in forests and woodlands

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Key words

disease management
epidemic
forest dieback
invasive pathogens
nursery infestation
root rot

Abstract Most members of the oomycete genus *Phytophthora* are primary plant pathogens. Both soil- and airborne *Phytophthora* species are able to survive adverse environmental conditions with enduring resting structures, mainly sexual oospores, vegetative chlamydospores and hyphal aggregations. Soilborne *Phytophthora* species infect fine roots and the bark of suberized roots and the collar region with motile biflagellate zoospores released from sporangia during wet soil conditions. Airborne *Phytophthora* species infect leaves, shoots, fruits and bark of branches and stems with caducous sporangia produced during humid conditions on infected plant tissues and dispersed by rain and wind splash. During the past six decades, the number of previously unknown *Phytophthora* declines and diebacks of natural and semi-natural forests and woodlands has increased exponentially, and the vast majority of them are driven by introduced invasive *Phytophthora* species. Nurseries in Europe, North America and Australia show high infestation rates with a wide range of mostly exotic *Phytophthora* species. Planting of infested nursery stock has proven to be the main pathway of *Phytophthora* species between and within continents. This review provides insights into the history, distribution, aetiology, symptomatology, dynamics and impact of the most important canker, decline and dieback diseases caused by soil- and airborne *Phytophthora* species in forests and natural ecosystems of Europe, Australia and the Americas.

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INTRODUCTION

The oomycete genus *Phytophthora* belongs to the *Peronosporaceae*, order *Peronosporales*, class *Peronosporomycetes*, kingdom *Stramenipila* (Dick 2001, Hulvey et al. 2010, Beakes et al. 2014, Thines & Choi 2016). Initially, based on phylogenetic analysis of ITS rDNA sequences from 50 *Phytophthora* species, the genus was structured in 10 phylogenetic clades (Cooke et al. 2000). Although increasing numbers of *Phytophthora* species and increasing numbers of nuclear and mitochondrial gene regions were used in subsequent phylogenetic studies, the basic structure of the genus remained unaltered (Martin & Tooley 2003, Kroon et al. 2004, 2012, Blair et al. 2008, Martin et al. 2014, Yang et al. 2017). However, recently the number of clades was expanded to 12 in order to accommodate the growing number of species in the *P. quercina* clade and the unique position of *P. lilii* (Rahman et al. 2015, Jung et al. 2017b). Several phylogenetic studies demonstrated that the genus *Phytophthora* is monophyletic with the 19 downy mildew genera residing within *Phytophthora* (Cooke et al. 2000, Kroon et al. 2004, Göker et al. 2007, Runge et al. 2011, Martin et al. 2014, Thines & Choi 2016). Recently, a closely related sister genus of *Phytophthora*, *Nothophytophthora*, which shares many phenotypic and ecological characters with *Phytophthora*, has been described from natural ecosystems in Asia, Europe and South America (Jung et al. 2017d).

Most *Phytophthora* (Greek for 'plant destroyer') species have a hemibiotrophic or necrotrophic lifestyle as primary plant pathogens, although for many aquatic *Phytophthora* species from phylogenetic Clades 6 and 9 a lifestyle as saprophytes and opportunistic necrotrophic pathogens seems likely (Erwin & Ribeiro 1996, Brasier et al. 2003, Jung et al. 2011, Nechwatal et al. 2013). In contrast, all c. 600 downy mildew species are host specific, obligate biotrophic plant pathogens (Göker et al. 2007, Runge et al. 2011, Beakes et al. 2012, Thines & Choi 2016). *Phytophthora* species are renowned as primary parasites on thousands of tree, shrub and crop species across the world. Depending on whether the lifecycle occurs mainly above- or below-ground a distinction is made between soilborne *Phytophthora* species causing fine root losses, root and collar rots and bleeding bark cankers, and airborne *Phytophthora* species causing leaf necrosis, shoot blights, fruit rots and also bleeding bark cankers (Erwin & Ribeiro 1996). However, several *Phytophthora* species have both a soil- and an airborne lifecycle. *Phytophthora cactorum*, for example, is causing root and collar rot in strawberries and a range of fruit and forest trees but can also infect the foliage and shoots of many ornamental plants and cause aerial bleeding cankers on European beech trees (Mircetich & Matheron 1983, Wilcox & Ellis 1989, Erwin & Ribeiro 1996, Jung 2009, Jung et al. 2016). In *P. pseudosyringae* the rate of sporangial caducity is highly variable between isolates enabling the pathogen to cause both fine root infections and aerial bleeding cankers on oaks, beech and other forest trees (Wickland et al. 2008, Jung et al. 2003b, 2013b, Jung 2009, Scanu & Webber 2016, Hansen et al. 2017).

It was estimated that, on a global scale, more than 66 % of all fine root diseases and more than 90 % of all collar rots of woody plants are caused by *Phytophthora* species (Tsao

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1990). However, in many cases, abiotic factors or secondary pathogens instead of the primary *Phytophthora* pathogens are considered as the causal agents of disease. The reasons for such misidentifications are mainly based on the specific life-cycles of *Phytophthora* spp. Highly specific isolation methods are required in order to break dormancy of resting spores and exclude fungi and other oomycetes like *Pythium* or *Phytopythium* which are usually much faster growing than *Phytophthora* species (Ribeiro 1978, Tsao 1983, Erwin & Ribeiro 1996, Jung et al. 1996, De Cock et al. 2015). *Phytophthora*-specific isolation approaches include, amongst others, a wide range of specific baiting tests, *Phytophthora*-specific isolation media containing various antibiotics and fungicides, low or high incubation temperature depending on the *Phytophthora* species, leaching of polyphenols from necrotic bark samples, and drying and re-moistening of infested soil samples (Tsao 1983, 1990, Jeffers & Aldwinckle 1987, Erwin & Ribeiro 1996, Jung et al. 1996, 2013a, 2016, 2017c, Jung 2009). Another problem for *Phytophthora* isolations can be fluctuations of inoculum levels depending on the phase of the disease. When first symptoms become visible in the crown of a mature tree, the destruction of the fine root system is already in an advanced stage resulting in a continuous decrease of *Phytophthora* inoculum. As a consequence, a secondary disease process caused by high populations of secondary pathogenic and saprophytic fungi is masking the primary cause of the disease (Tsao 1990, Erwin & Ribeiro 1996, Jung et al. 1996). Similarly, isolation tests of *Phytophthora* species from bleeding bark lesions are only reliable at the active advancing lesion fronts whereas slightly older parts of the lesions are quickly colonised by secondary pathogenic fungi preventing the isolation of the primary *Phytophthora* pathogen (Erwin & Ribeiro 1996, Jung & Blaschke 2004, Jung 2009). In recent years, an array of highly sensitive, high-throughput, species-specific molecular detection methods have been developed, which facilitate the diagnosis of *Phytophthora* diseases significantly, and are particularly useful for routine screening of high numbers of samples for harmful and emerging *Phytophthora* pathogens (Schubert et al. 1999, Nechwatal et al. 2001, Schena et al. 2006, Martin et al. 2012, Scibetta et al. 2012, Sikora et al. 2012, Than et al. 2013, King et al. 2015, Schenck et al. 2016). Recent metagenomic approaches provide an efficient tool for large-scale surveys of *Phytophthora* diversity (Vettraino et al. 2012, Català et al. 2015, Sapkota & Nicolaisen 2015, Burgess et al. 2017).

Detailed descriptions and schematic illustrations of the life-cycles of airborne and soilborne *Phytophthora* species were given by several authors (Hickman 1958, Ribeiro 1978, Erwin & Ribeiro 1996, Jung 1998, Hansen et al. 2000, Agrios 2005, Grünwald et al. 2008). Both soil- and airborne *Phytophthora* species are able to survive unsuitable environmental conditions over several years with dormant resting structures (oospores, chlamydospores and hyphal aggregations) in the soil or in infected plant tissues. When environmental conditions become suitable (high moisture and temperature higher than the minimum temperature required by the respective *Phytophthora* species) the resting spores germinate by forming sporangia. In soilborne *Phytophthora* species, the sporangia release motile, biflagellate zoospores into the soil water which are then chemotactically attracted by a gradient of organic acids released from the elongation zone of young fine roots. In airborne species, the caducous sporangia are spread by wind and rain splash onto above-ground plant tissues where they either germinate directly or release zoospores. After penetrating the rhizodermis, exodermis or periderm of roots or the cuticle and epidermis of leaves, shoots and fruits *Phytophthora* grows as a hemibiotroph or necrotroph inter- and intracellular in the infected tissue with typical coralloid to irregular, non-septate hyphae. Nutrient depletion, competition by secondary antagonistic fungi or strong defence reactions by the host plant stimulate the production of resting structures. After decomposition of the necrotic tissue by saprophytes the resting spores are released into the soil, and the cycle starts again. Via the continuous production of sporangia on infected roots, leaves and fruits, *Phytophthora* pathogens can prolifically increase and disseminate their inoculum from initially very low levels during a relatively short time of favourable environmental conditions. Therefore, *Phytophthora*-induced fine root, leaf and fruit diseases are considered to be multicyclic (Ribeiro 1978, Erwin & Ribeiro 1996, Grünwald et al. 2008, Jung et al. 2013b). As a consequence of the root and bark damage caused by soilborne spp., the crowns of affected trees develop non-specific symptoms of drought and malnutrition, including increased crown transparency, sparse ramification and stunted growth of lateral shoots leading to whip-like branch structures and clustering of leaves at the end of branches, small-sized, often chlorotic foliage, wilting, dieback of branches, crown-dieback and eventually mortality (Erwin & Ribeiro 1996, Jung et al. 1996, 2000, 2013b, Jung 2009). However, in mature trees it can take decades of inoculum build-up and progressive destruction of the fine root system before the crowns begin to show visible symptoms (Ribeiro 1978, Tsao 1990, Erwin & Ribeiro 1996, Jung et al. 1996, 2000). Predisposing factors, such as waterlogging or planting of trees on sites not suitable for the species, as well as contributing factors which either reduce the vitality of the tree (e.g., extreme droughts or defoliations) or favour the pathogen (e.g., excess soil moisture following heavy rain, flooding or irrigation), can accelerate the disease process or make it possible in the first place (Davison 1988, Brasier et al. 1993, Marçais et al. 1993, Erwin & Ribeiro 1996, Jung et al. 1996, 2013b, Jung 2009). For European oak decline, a conceptual model was presented by Jönsson (2006) which included the complex interactions between soilborne *Phytophthora* species and various biotic and abiotic factors. Airborne *Phytophthora* diseases usually progress gradually upwards and in severe cases cause complete defoliation within a few months (Buddenhagen & Young 1957, Erwin & Ribeiro 1996, Durán et al. 2008). Extent and progress of soil- and airborne *Phytophthora* diseases are strongly depending on both long-term climatic and short-term weather conditions as demonstrated by the current declines and diebacks of oak and beech stands in Europe (Brasier et al. 1993, Brasier & Scott 1994, Jung et al. 1996, 2000, 2013b, Jung 2009, this review), the leaf fall disease of rubber in India and Malaysia (Agnihothrudu 1975, Erwin & Ribeiro 1996), the epidemics of *P. ramorum* on oaks and tanoaks in the western USA and on larch trees in the UK (Rizzo et al. 2002, 2005, Brasier et al. 2010, Grünwald et al. 2012a, Harris & Webber 2016, this review), and the needle cast and defoliation caused by *P. pinifolia* on *Pinus radiata* in Chile (Durán et al. 2008, this review).

In 1996, 50 *Phytophthora* species were known to science (Erwin & Ribeiro 1996). During the past two decades, more than 100 new *Phytophthora* species have been described or informally designated (Brasier 2009, Jung et al. 2011, 2017a, b, c, Hansen et al. 2012, Scanu et al. 2015, Yang et al. 2017, Burgess et al. 2018). This exponential increase has been caused by several factors:

1. more researchers have been studying *Phytophthora* diversity in natural ecosystems while research in the past had been focussed on agricultural crops. A conservative estimation predicted that there may be another 200–600 unknown *Phytophthora* species in natural ecosystems awaiting their detection (Brasier 2009);
2. phylogenetic analyses of nuclear and mitochondrial gene regions allow to discriminate complexes of morphologically similar but phylogenetically distinct species (Jung et

al. 2003b, 2011, 2017b, c, Jung & Burgess 2009, Hong et al. 2009, 2011, Bezuidenhout et al. 2010, Rea et al. 2010, Ginetti et al. 2014, Henricot et al. 2014, Burgess et al. 2018);
3. the exponential increase in imports of living plants from overseas in combination with an outdated list-based plant biosecurity approach and notoriously understaffed plant protection services enables the continuous accidental introduction of exotic *Phytophthora* species (and other pests and pathogens) to Europe and other continents (Brasier 2008, Liebhold et al. 2012, Hantula et al. 2013, Santini et al. 2013, Eschen et al. 2015a, b, 2017, Jung et al. 2016); and
4. hybridisations between phylogenetically close *Phytophthora* species which due to geographic separation did not build up reproductive barriers and accidentally met after the introduction of one or both parents. Well-known examples of the latter are *P.* ×*alni*, *P.* ×*cambivora*, *P.* ×*pelgrandis*, *P.* ×*serendipita* and *P.* ×*stagnum*, all infecting woody plants, and undescribed hybrids in the complex of vegetable infecting *Phytophthora* species from Clade 8b (Brasier et al. 2004, Man in 't Veld et al. 2012, Bertier et al. 2013, Yang et al. 2014, Husson et al. 2015, Jung et al. 2017c).

A Europe-wide survey, conducted in almost 2 000 nursery stands of 730 nurseries and in 2 500 young forest, horticultural and ornamental plantings, demonstrated widespread *Phytophthora* infestations. More than 80 % of the nursery stands in more than 90 % of the tested nurseries, and 2/3 of the tested young plantings were infested with in total 68 different *Phytophthora* species, of which 44 were unknown to science before 1990 (Jung et al. 2016). Based on these results, a calculation suggested that across Europe between 1990 and 2010 approximately 680 000 new afforestations with a total area of almost 5 million hectares had been established with *Phytophthora*-infested nursery stock. In the same period, the area of potentially *Phytophthora*-infested re-forestations may have exceeded 17 million hectares (Jung et al. 2016). Apart from only a few potentially native *Phytophthora* species from Clades 3 and 6, the vast majority of these 68 *Phytophthora* taxa are considered alien invasive pathogens in Europe, based on an accumulating body of indirect evidence. These include high aggressiveness towards native European tree, crop and ornamental plant species, occurrence in healthy, undisturbed natural ecosystems in other continents, presence of both mating types of heterothallic *Phytophthora* species in natural ecosystems of other continents, low genetic variability of European *Phytophthora* populations, and close phylogenetic relatedness to non-native *Phytophthora* species (Jung et al. 2016). Also in Australia and the USA, *Phytophthora* infestations of nursery stock are common (Hardy & Sivasithamparam 1988, MacDonald et al. 1994, Davison et al. 2006, Schwingle et al. 2007, Yakabe et al. 2009, Bienapfl & Balci 2014, Parke et al. 2014, Yang et al. 2014, Simamora et al. 2015).

The potentially high number of unknown *Phytophthora* species and the unknown origin of many known aggressive *Phytophthora* species in combination with the high *Phytophthora*-infestation rates of nursery stock and the increasing complexity, intensity and volume of the international plant trade (Brasier 2008, Dehnen-Schmutz et al. 2010, Drew et al. 2010, Liebhold et al. 2012, Jung et al. 2016, Chapman et al. 2017, Eschen et al. 2017) pose a serious threat to the health and sustainability of natural ecosystems, managed forests and crop production systems on a global scale.

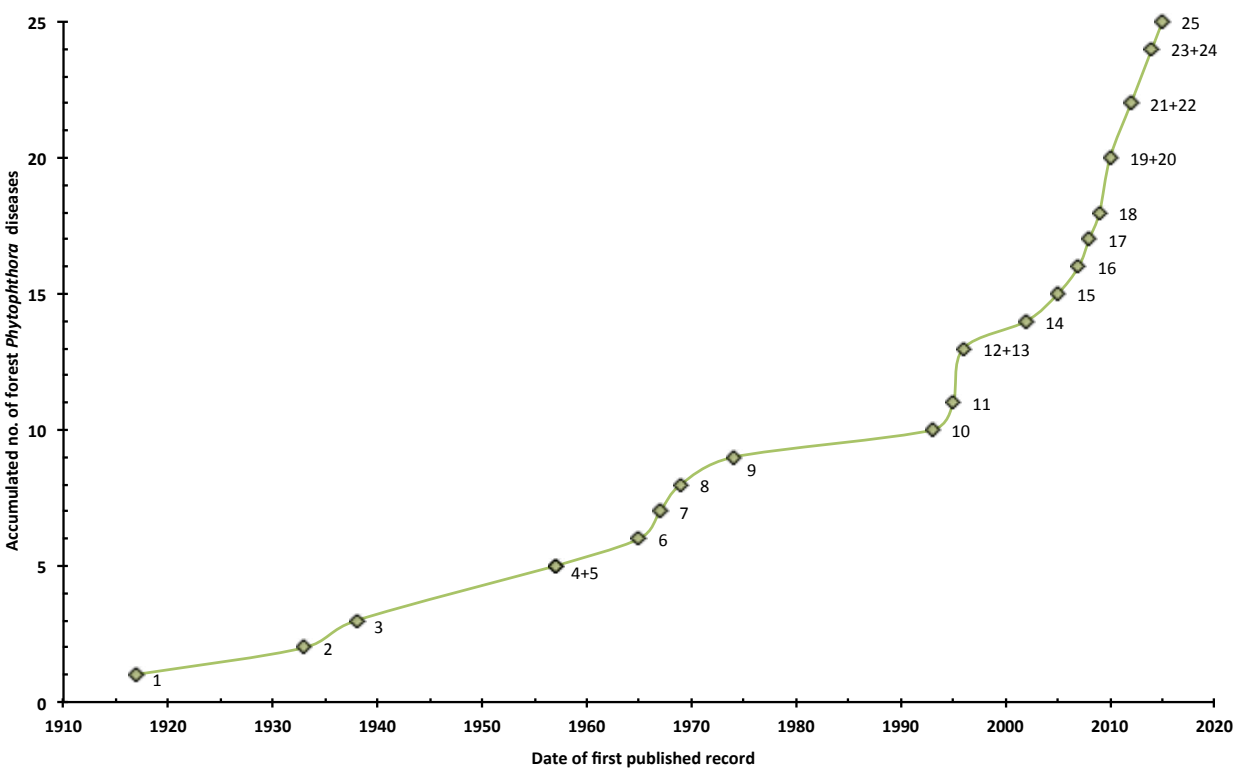

Fig. 1 Accumulated number of important *Phytophthora* declines and diebacks of forests and natural ecosystems over time; 1 = ink disease of *Castanea sativa* in Europe (observation of first typical symptoms in 1838); 2 = ink disease of *Castanea dentata* in the USA (observation of first typical symptoms in 1824); 3 = decline of *Fagus sylvatica* in the UK; 4 = littleleaf disease of pines in the USA; 5 = decline and mortality of *Chamaecyparis lawsoniana* in the Pacific Northwest; 6 = jarrah dieback in Western Australia (WA; observation of first typical symptoms in the 1920s); 7 = ink disease of *C. crenata* and chestnut hybrids in Korea; 8 = eucalypt dieback in Victoria (observation of first typical symptoms in 1935); 9 = kauri dieback in New Zealand; 10 = Mediterranean oak decline; 11 = *Alnus* mortality in Europe; 12 = temperate European oak decline; 13 = decline of *F. sylvatica* in mainland Europe; 14 = Sudden Oak Death in California and Oregon; 15 = mortality of *Austrocedrus chilensis* in Argentina (observation of first typical symptoms in 1948); 16 = oak decline in the Eastern USA; 17 = needle cast and defoliation of *Pinus radiata* in Chile; 18 = dieback of *Eucalyptus gomphocephala* in WA; 19 = dieback of *E. rudis* in WA; 20 = Sudden Larch Death in the UK; 21 = dieback of *Nothofagus* spp. in the UK; 22 = mortality of *Juniperus communis* in the UK; 23 = red needle cast of *P. radiata* in New Zealand; 24 = leaf and twig blight of *Ilex aqufolium* in Corsica and Sardinia; 25 = dieback of Mediterranean maquis vegetation.

Before 1950, the only known forest diseases caused by *Phytophthora* pathogens were ink disease of chestnuts in Europe and the USA and littleleaf disease of pines in the USA (Crandall et al. 1945, Zak 1957, Tainter & Baker 1996). During the past six decades, the number of previously unknown *Phytophthora* declines and diebacks of forests and natural ecosystems and the number of described species and informally designated taxa of *Phytophthora* have increased exponentially (Fig. 1; Brasier 2009). Most of these diseases are driven by exotic *Phytophthora* species which remain unnoticed in their native environment and often were unknown to science prior to their introduction to other continents. However, in their new environments they became invasive and threatened a non-adapted flora which due to a lack of co-evolution contains a high number of susceptible plant species (Shearer & Tippett 1989, Marks & Smith 1991, Erwin & Ribeiro 1996, Jung et al. 2000, 2013b, 2016, Rizzo et al. 2002, Shearer et al. 2004, Brasier 2008, Grünwald et al. 2012a). This review provides insights into the history, distribution, aetiology, symptomatology, dynamics and impact of the most important canker, decline and dieback diseases caused by soil- and airborne *Phytophthora* species in forests and natural ecosystems of Europe, Australia and the Americas.

SOILBORNE PHYTOPHTHORA DISEASES IN FORESTS AND WOODLANDS

Ink disease of chestnuts worldwide

Ink disease, caused by *Phytophthora* species, is one of the most destructive diseases affecting *Castanea sativa* worldwide. In Europe, typical symptoms were first reported in Portugal in 1838 (Crandall et al. 1945, Crandall 1950) and since then it has become widespread across the continent with an increase in incidences during the last decades (Vannini & Vettraino 2001, Vettraino et al. 2005, Černý et al. 2008, Jung et al. 2013b, Tziros & Diamandis 2014). In the United States, ink disease was the main problem of *C. dentata* before the chestnut blight epidemic (Crandall et al. 1945, Crandall 1950). Ink disease symptoms in the crown are best observed during the vegetative growing season. The trees initially show small-sized and chlorotic foliage followed by increasing transparency, defoliation and dieback of the whole crown, eventually leading to extensive dieback and mortality of trees (Fig. 2a–d). Dry leaves and fruits often persist on dead trees over the winter. These symptoms are caused by extensive root losses and dark-brown and flame shaped necrosis in the inner bark developing from the main roots into the collar (Fig 2e–g). Blue to black exudates, oozing from the necrotic tissues through cracks in the bark, are often visible on the stem, collar and roots. The disease was named after the black exudates from necrotic roots staining the surrounding soil (Fig. 2e). Infected chestnut stumps can lose their ability to resprout due to the destruction of the entire root system. Ink disease also occurs on chestnut seedlings in nurseries and new plantations which usually show a rapid or gradual wilting (Jung et al. 2016).

The hybrid species *P.* ×*cambivora* has been the main species associated with ink disease in central and south-eastern Europe, while *P. cinnamomi* seems to be more widespread in Atlantic regions such as England and France and in the USA (Day 1938, Crandall et al. 1945, Crandall 1950, Vettraino et al. 2001, 2005, Martins et al. 2007, Černý et al. 2008, Jung et al. 2016). In Portugal, *P. cinnamomi* and *P.* ×*cambivora* often co-occur resulting in particularly high disease incidences and mortality rates (Fig. 1a, b) (Lopes-Pimentel 1946, 1947, T. Jung & M. Horta Jung unpubl. data). Due to its intolerance to low temperatures, the current distribution of *P. cinnamomi* in European forests is limited to areas with an average minimum soil temperature above 1.4 °C (Marçais et al. 2004, Vettraino et al. 2005). In accordance with the intensification of *P. cinnamomi* activity in Europe predicted by the CLIMEX model for increasing average temperatures (Brasier & Scott 1994, Burgess et al. 2017), *P. cinnamomi* is currently spreading in Italy to chestnut areas characterised by mild winters (Vettraino et al. 2005). Also in Chile, *P. cinnamomi* is currently threatening planted forests of *C. sativa* (Fig. 1c) (Jung et al. 2018). Several other *Phytophthora* species of minor impact were found associated with declining chestnuts in Europe, including *P. cactorum*, *P. cryptogea*, *P. gonapodyides*, *P. megasperma*, *P. nicotianae*, *P. plurivora*, *P. pseudosyringae*, *P. sansomeana* and *P. syringae* (Vettraino et al. 2005, Černý et al. 2008, Perlerou et al. 2010, Scanu et al. 2010, Jung et al. 2013b). The recently described *P. castanetorum*, a close relative of *P. quercina*, was isolated alongside other *Phytophthora* spp. from diseased chestnut trees in Italy and Portugal (Jung et al. 2017b). In Oregon, *P.* ×*cambivora* causes root rot, girdling basal stem cankers and mortality of golden chinquapin (*Chrysolepis chrysophylla*) which is closely related to the genus *Castanea*. The concentration of affected trees along roads suggests recent introduction and spread of the pathogen (Saavedra et al. 2007). In Japan and South Korea, *P. castaneae* (previously *P. katsurae*) was repeatedly found causing bleeding bark lesions and mortality of *C. crenata* and the chestnut hybrid *C. crenata* × *C. mollissima* (Uchida 1967, Lee et al. 2009, Oh & Parke 2012). *Phytophthora castaneae*, together with *P. cinnamomi*, was also associated with bleeding bark lesions and mortality of *Castanopsis carlesii* in subtropical forests of Taiwan (Jung et al. 2017a).

Ink disease incidence is strictly related to climatic and site condition as well as human activities (Fonseca et al. 2004, Robin et al. 2006, Martins et al. 2007, Vannini et al. 2010). Heavy or continuous rain during the vegetative season, soil compaction and disturbance by tillage practices, physical restrictions to root expansion, poor soil fertility, vehicle movement along roads, and human recreational activities in forests are the main predisposing and contributing factors for disease development (Fonseca et al. 2004, Vannini et al. 2005, Martins et al. 2007). Planting of infested nursery stock and movement of contaminated substrates are the main pathways of short and long-distance inoculum dispersal (Jung et al. 2016). The existence of genetic variability in susceptibility to *P.* ×*cambivora* in *C. sativa* and resistance to *P. cinnamomi* in some clones of *C. sativa* and in many clones of the Euroasiatic chestnut hybrids *C. crenata* × *C. sativa* and *C. molissima* × *C. sativa* could provide the basis for a resistance screening programme necessary for a long-term management of ink disease in Europe (Robin et al. 2006, Miranda-Fontaíña et al. 2007, Costa et al. 2011, Santos et al. 2015, 2017a, b).

Oak declines and diebacks in Europe and North America

Episodically recurring declines of oak (*Quercus* spp.) stands have been reported since the early 1900s in both temperate and Mediterranean regions of Europe and in the USA (Staley 1965, Delatour 1983, Ragazzi et al. 1989, Schütt 1993, Gottschalk & Wargo 1996, Abrams 2003). In Europe, the current phase of oak decline started in the 1980s and is still ongoing (Delatour 1983, Brasier et al. 1993, Jung et al. 1996, 2000, 2013b, Vettraino et al. 2002, Balci & Halmschlager 2003a, b). A wide range of abiotic and biotic factors, including frost, drought, air pollutants, decreased groundwater levels, silvicultural mismanagement, insect defoliators, bark borers, fungal species like *Ophiostoma* and *Ceratocystis* spp., bacteria, mycoplasma-like organisms and viruses, were discussed as predisposing and inciting factors of this phenomenon (Manion 1981, Delatour 1983, Nihlgård 1985, Nienhaus 1987, Oleksyn & Przybyl 1987, Oosterbaan & Nabuurs 1991, Siwecki & Liese 1991, Ahrens & Seemüller 1994, Schlag 1995, Ragazzi et al. 1995, Thomas et al. 2002).

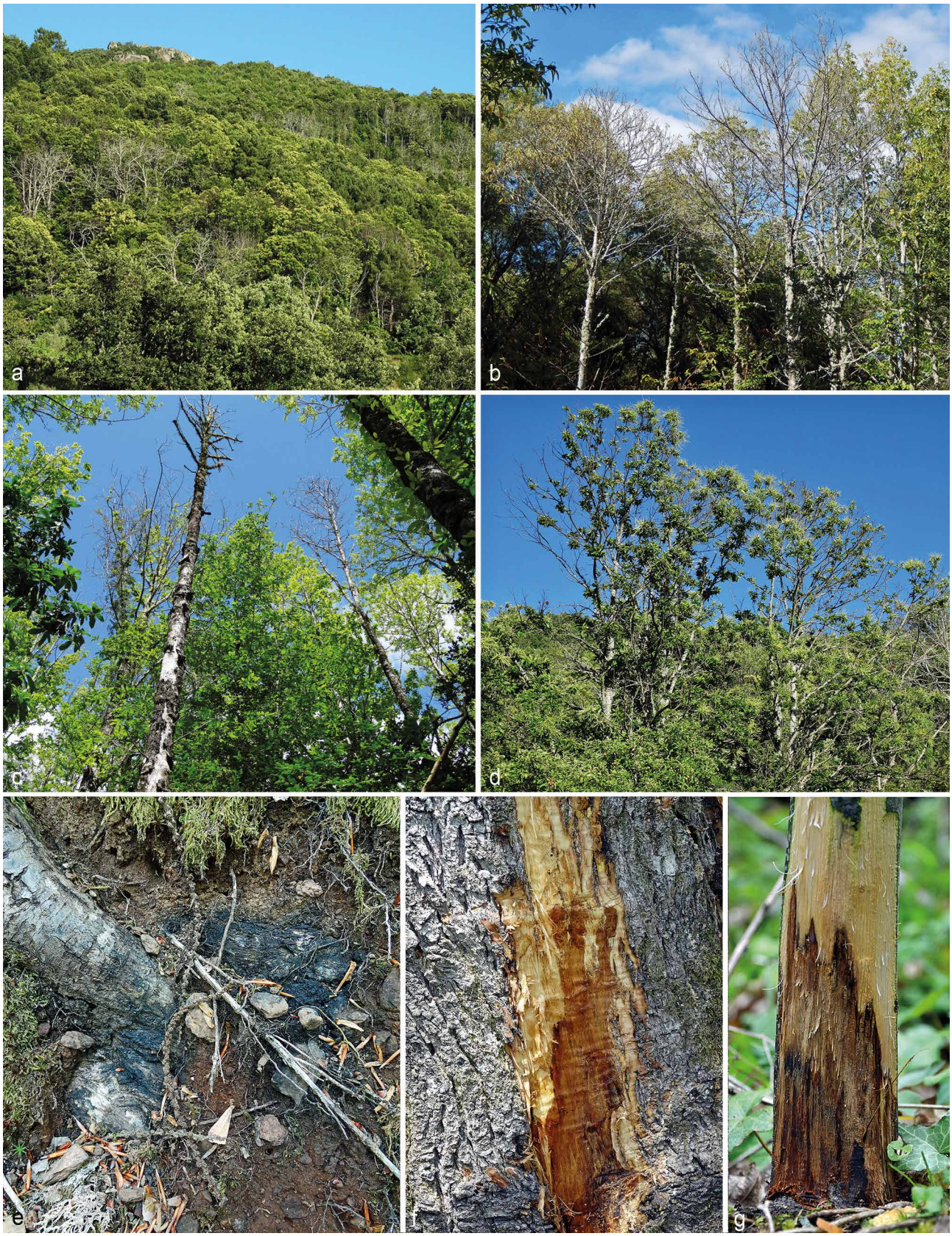

Fig. 2 Ink disease symptoms on *Castanea sativa* caused by *Phytophthora* spp. a. Extensive dieback and mortality caused by *P. cinnamomi*, *P.* ×*cambivora* and *P. castanetorum* in Portugal; b. patch dieback and mortality caused by *P.* ×*cambivora* in Portugal; c. chlorosis, microphylly, thinning, dieback and mortality of planted chestnut trees caused by *P. cinnamomi* in Chile; d. microphylly, severe thinning and dieback caused by *P. cinnamomi* and *P.* ×*cambivora* in Portugal; e. surface roots with bleeding lesions and black staining of surrounding soil caused by *P.* ×*cambivora* in Portugal; f, g. typical flame-shaped necrotic lesions of the inner bark caused by *P.* ×*cambivora* on the collar of a mature (f) and a young tree (g) in Italy. — Photos: a–e: T. Jung; f, g: B. Scanu.

However, none of these potential agents accounted for more than local or regional decline episodes. Due to similarities in aetiology between Mediterranean oak decline and eucalypt dieback in Western Australia (WA) caused by *P. cinnamomi* (Shearer & Tippett 1989, this review), and earlier records of *P. cinnamomi* from declining chestnuts and oaks in Portugal (Lopes-Pimentel 1946, 1947, this review), the possible involvement of *P. cinnamomi* in Iberian oak decline was suggested in 1991 and soon after confirmed (Brasier et al. 1993). Another study demonstrated the association of several known and previously unknown *Phytophthora* species with declining oak forests in central Europe and temperate and Mediterranean regions of Italy and Slovenia (Jung et al. 1996). Numerous surveys in oak stands throughout Europe uncovered a diverse assemblage of *Phytophthora* taxa including eight known species, *P. cactorum*, *P.* ×*cambivora*, *P. cinnamomi*, *P. cryptogea*, *P. drechsleri*, *P. gonapodyides*, *P. megasperma*, *P. syringae*; and 14 previously unknown *Phytophthora* taxa, *P. bilorbang*, *P. chlamydospora*, *P. europaea*, *P. gallica*, *P. multivora*, *P. plurivora*, *P. pseudosyringae*, *P. psychrophila*, *P. quercina*, *P. ramorum*, *P. tyrrhenica*, *P. uliginosa*, *P.* taxon forest soil and *P.* taxon river soil (Brasier et al. 1993, Brasier 1996, Jung et al. 1996, 1999, 2000, 2002, 2003b, 2013b, 2017b, Robin et al. 1998, Gallego et al. 1999, Hansen & Delatour 1999, Sanchez et al. 2002, Vettraino et al. 2002, Balci & Halmschlager 2003a, b, Jönsson et al. 2003b, 2005, Moreira & Martins 2005, Brown & Brasier 2007, Jung & Nechwatal 2008, Corcobado et al. 2010, Camilo-Alves et al. 2013, Scanu et al. 2013, T. Jung, M. Horta Jung & S.O. Cacciola unpubl. data). Several of these *Phytophthora* species, including *P.* ×*cambivora*, *P. cinnamomi*, *P. cryptogea*, *P. drechsleri*, *P. multivora*, *P. plurivora* and *P. ramorum*, are introduced invasive pathogens in Europe whereas most other species are considered to be native or of cryptic origin (Jung et al. 2016, 2017b). Recently, the presence of *P. cinnamomi* and other *Phytophthora* species has also been demonstrated in declining oak stands in Western Algeria (H. Smahi & B. Scanu unpubl. data). Against the background of almost ubiquitous infestations of oak stands in European nurseries with *P. cinnamomi*, *P. quercina*, *P. plurivora* and 13 other *Phytophthora* spp. (Jung et al. 2016), the massive afforestation during the previous three decades, stimulated by both national and EU subsidy programmes, may have contributed to the widespread *Phytophthora* infestations of oak woodlands across Europe.

In Mediterranean regions, the most affected species are *Quercus suber* and *Q. ilex* and, to a lesser extent, *Q. cerris*, *Q. faginea*, *Q. pubescens* and *Q. pyrenaica* (Brasier et al. 1993, Gallego et al. 1999, Sanchez et al. 2002, Vettraino et al. 2002, Pérez-Sierra et al. 2013, T. Jung & M. Horta Jung unpubl. data). In temperate regions, stands of *Q. petraea* and *Q. robur* usually show similar disease incidences. Generally, in oak decline a rapid death of previously healthy-looking trees and a slow chronic decline and dieback are distinguished although there are gradual transitions between both scenarios (Delatour 1983, Brasier et al. 1993, Jung et al. 1996, 2000, Gallego et al. 1999, Vettraino et al. 2002, Camilo-Alves et al. 2013). Rapid or acute death of oaks occurs mainly in Mediterranean regions and is usually caused by an interaction between excessive root losses and collar rot cankers by *P. cinnamomi* and severe summer droughts (Brasier et al. 1993, Gallego et al. 1999, Sanchez et al. 2002, Scanu et al. 2013, Jung et al. 2013b). Affected trees and whole stands of *Q. suber* and *Q. ilex* rapidly develop crown dieback and often collapse within the same year (Fig. 3a–c). Bark lesions at main roots and the collar may occur in severely infected oak trees, with black exudates oozing from the outer bark and necrotic lesions and staining of the underlying phloem and xylem tissues (Fig. 3e, f), often girdling the stem. Chronic decline and dieback are characterized by a progressive crown thinning, branch dieback, leaf chlorosis and abundant proliferation of epicormic shoots (Fig. 3d). These crown symptoms are caused by extensive losses of both fine roots and lateral small woody roots and callusing cankers on suberized roots (Fig. 3g–i). The ability of the root system to absorb and transport water and nutrients is increasingly hampered, eventually leading to a slow death of the trees (Jung et al. 1996, 2000, 2013b, Jönsson et al. 2005). However, the interaction with several abiotic stress factors, including prolonged droughts, waterlogging, fluctuating water tables, sandy or shallow soils and unseasonal heavy rain, and opportunistic pathogens and pests, can accelerate the disease progress and cause rapid wilting and mortality of trees (Brasier et al. 1993, Jung et al. 1996, 2000, 2003a, Vettraino et al. 2002, Balci & Halmschlager 2003a, b, Jönsson et al. 2003a, 2005, Jönsson 2006, Moreira & Martins 2005).

The distribution of *Phytophthora* species and their impact on oak trees depends on the site conditions, in particular soil drainage and pH. In a study of fine root systems in 35 forest stands in Germany, the oak-specific *P. quercina* and other *Phytophthora* spp. were frequently detected in the rhizosphere of mature *Q. robur* and *Q. petraea* on sites with a mean soil-pH higher than 3.5 and sandy-loamy to clayey soil texture (Jung et al. 2000). In infested forests, crown transparency and several fine root parameters were significantly correlated. Oak trees with *P. quercina* and other *Phytophthora* spp. in their rhizospheres had significantly higher losses of fine roots and of small woody roots, reduced crown density and a threefold higher decline risk than oaks without *Phytophthora*. In contrast, no *Phytophthora* species could be recovered from forests on well-drained sandy to sandy-loamy soils with a mean pH below 3.9, and root and crown conditions of oak trees were not correlated in these non-infested stands (Jung et al. 2000). Also in Austria, Italy, Turkey and Sweden, significant associations between presence of *P. quercina* and decline of oaks were demonstrated (Vettraino et al. 2002, Balci & Halmschlager 2003a, b, Jönsson et al. 2005).

In soil infestation tests, *P. cinnamomi*, *P. quercina*, *P.* ×*cambivora*, *P. plurivora* and *P. uliginosa* caused severe losses of fine roots and small woody roots, necrotic root lesions and mortality of *Q. ilex*, *Q. petraea*, *Q. robur* and *Q. suber* seedlings (Jung et al. 1996, 1999, 2002, 2003a, b, 2017c, Robin et al. 1998, Gallego et al. 1999, Sanchez et al. 2002, Jönsson et al. 2003a, Pérez-Sierra et al. 2013, Corcobado et al. 2017).

The primary role of *P. cinnamomi* in the decline of oak woodlands across Mediterranean countries, including Portugal, Spain and southern regions of France and Italy was demonstrated by numerous studies (Brasier et al. 1993, Brasier 1996, Robin et al. 1998, Gallego et al. 1999, Sanchez et al. 2002, Vettraino et al. 2002, Moreira & Martins 2005, Camilo-Alves et al. 2013, Jung et al. 2013b, Scanu et al. 2013). For many years, *P. cinnamomi* was considered the only *Phytophthora* species associated with Mediterranean oak decline (Camilo-Alves et al. 2013). However, the *Phytophthora* diversity in Mediterranean oak ecosystems is considerably higher than previously assumed. Several other *Phytophthora* species have recently been isolated from declining Mediterranean oaks, including *P. gonapodyides* from *Q. ilex* in Extremadura (Spain), *P. psychrophila*, *P. quercina* and *P. syringae* from *Q. ilex* and *Q. faginea* in two protected areas in Italy and eastern Spain, and the newly described species *P. tyrrhenica* from both *Q. ilex* and *Q. suber* in Sardinia and Sicily (Corcobado et al. 2010, Pérez-Sierra et al. 2013, Linaldeddu et al. 2014, Scanu et al. 2015, Jung et al. 2017b). Pathogenicity of all these *Phytophthora* species to the respective oak species was demonstrated, and their involvement in the declines suggested. In addition, in co-infection experiments *P. cinnamomi*, *P. gonapodyides* and *P. quercina* caused severe mortality of young *Q. ilex* seedlings which might explain the widespread

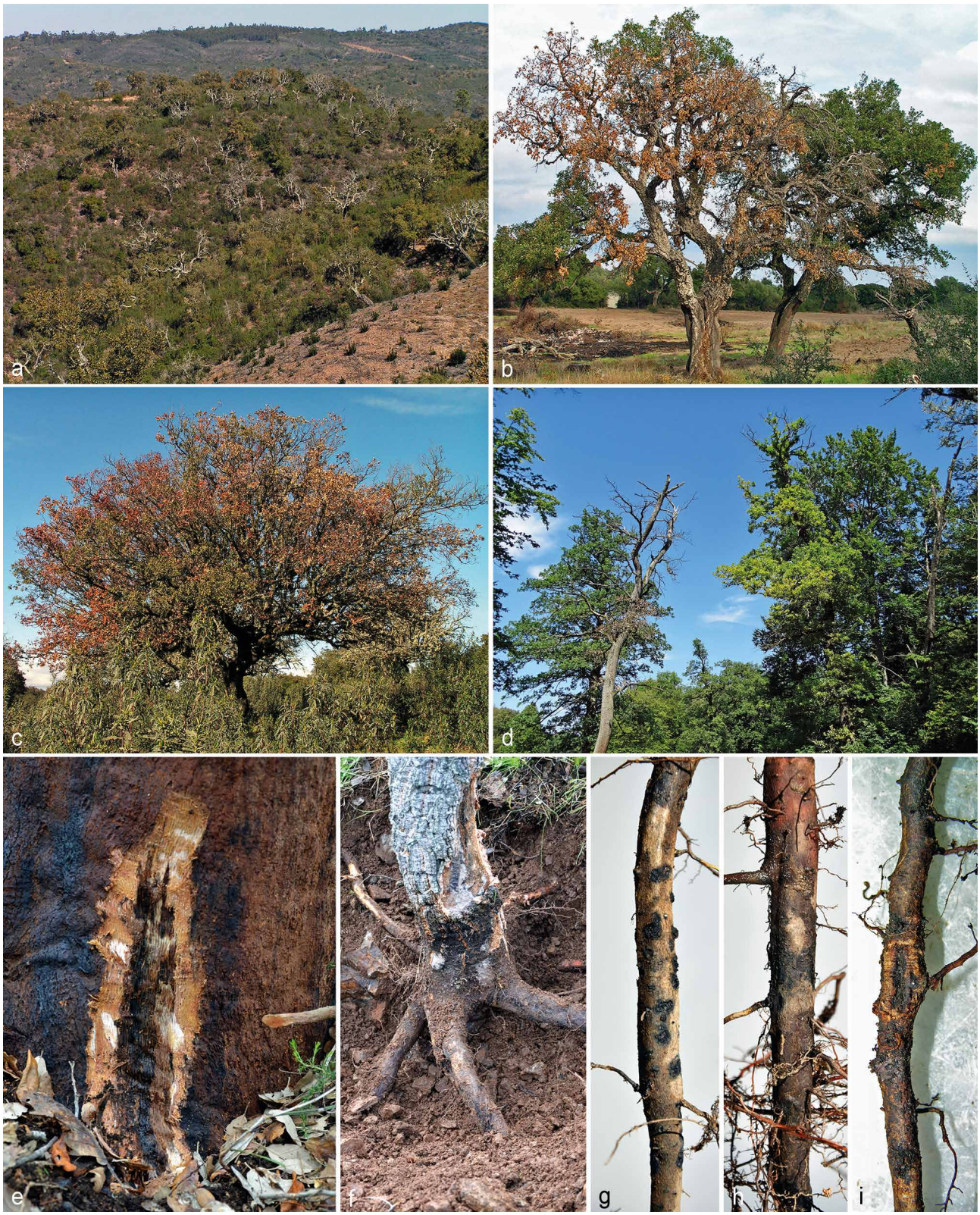

Fig. 3 Oak decline symptoms caused by *Phytophthora* spp. a. Extensive dieback and mortality of *Quercus suber* trees caused by *P. cinnamomi* in Portugal; b. progressive dieback and wilting of a mature *Q. suber* caused by *P. cinnamomi* and *P. quercina* in Sardinia (Italy); c. sudden death of a *Q. ilex* due to *P. cinnamomi* in a savannah-like ecosystem in Spain; d. *Q. robur* trees in Germany showing chlorosis, thinning and dieback of crowns, abundant proliferation of epicormic shoots, and mortality due to severe fine root destructions caused by *P. plurivora* and *P. quercina*; e. bleeding collar lesion with flame-shaped staining of the underlying xylem caused by *P. cinnamomi* on a mature *Q. suber* in Sardinia; f. root and collar rot caused by *P. cinnamomi* on a young *Q. suber* in a forest plantation in Sardinia; g, h. small woody roots of a mature *Q. suber* in Italy showing severe losses of fine roots and lateral roots and black necrotic lesions due to *P. cinnamomi* infections; i. small woody root of a mature *Q. robur* in Germany with severe losses of lateral roots and a callusing bark canker caused by *P. plurivora* and *P. quercina*. — Photos: a–d, i: T. Jung; e–h: B. Scanu.

lack of seedling recruitment in Mediterranean oak forests (Corcobado et al. 2017).

Several studies of Mediterranean oak decline have shown the interaction between *P. cinnamomi* and a combination of site factors, including the presence of shallow or compacted soils (Moreira & Martins 2005) and prolonged summer droughts (Brasier et al. 1993, Gallego et al. 1999). Mediterranean oak forests are delicate ecosystems, which are adversely impacted by human activities and global climate changes (Desprez-Loustau et al. 2010). The Mediterranean basin, which includes most of the natural range of *Q. suber* and *Q. ilex*, is considered one of the hot spots of future climate changes (Pachauri & Reisinger 2007). In this context, the predicted increase of *P. cinnamomi* activity with rising average temperatures (Brasier & Scott 1994, Brasier 1996, Burgess et al. 2017) could intensify root and collar rot incidences and further destabilise Mediterranean oak forests. Climate changes, in particular a rise of mean winter temperatures, a seasonal shift of precipitation from summer into wintertime, and a tendency towards heavy rain and prolonged droughts, have also been discussed as triggering factors for the current *Phytophthora*-related oak decline in temperate regions of Europe which, compared to previous oak decline episodes, is exceptional regarding its epidemic extent and long duration (Jung et al. 1996, 2000, 2003a, 2013b).

Also in the USA, episodes of oak decline with a symptomatology similar to European oak declines are occurring since decades (Staley 1965, Gottschalk & Wargo 1996, Abrams 2003). However, despite *P. cinnamomi* has been reported since the 1920s in North America, the involvement of *Phytophthora* species was not investigated conclusively (Balci et al. 2007). By the 1950s, *P. cinnamomi* was widespread in the north-eastern USA, but the significance of this species in tree health was only evaluated in connection with the demise of American chestnut (*C. dentata*). Today, in eastern US oak forests, *P. cinnamomi* is common, but due to its sensitivity to deep frost, its distribution is limited to below 40° northern latitude. In contrast, most other *Phytophthora* species are not restricted climatically but have a scattered distribution. The assemblage of *Phytophthora* species associated with oak stands includes *P. cryptogea*, *P. europaea*, *P. quercetorum*, *P. pini*, *P. plurivora*, *P.* ×*cambivora* and *P.* taxon ohioensis (Balci et al. 2007, 2008a, 2010, McConnell & Balci 2014b). *Phytophthora cactorum* and *P. heveae* occur only in southern oak forests (Meadows et al. 2011). All these species proved to be pathogenic to oaks in pathogenicity tests, but due to its widespread occurrence, *P. cinnamomi* has long been suspected as the main driver of oak decline in eastern US forests. Due to cold winter temperatures limiting canker development on stems, *P. cinnamomi* is mainly infecting the root system. Necrotic lesions on larger roots or bleeding stem cankers are only sporadically found on oaks in the southern USA, California and Mexico (Mircetich et al. 1977, Tainter et al. 2000, Wood & Tainter 2002, Balci et al. 2008b). Several recent studies investigated the role of *P. cinnamomi* in oak decline events in eastern US forests and provided insights into the epidemiology of the disease. Most significantly, both in the field and in pathogenicity tests *P. cinnamomi*-associated fine root losses were shown to be related to the propagule density of the pathogen. It was demonstrated that fine root losses are driving oak decline events in moist low-elevation stands where inoculum levels of the pathogen are higher and in areas like plant hardiness zones 6 and 7 where fine root regeneration is limited due to climatic constraints (McConnell & Balci 2014a, b).

Decline and mortality of Alnus species in Europe

In Europe, four *Alnus* species are indigenous. They are all characterised by having a symbiosis with the nitrogen-fixing actinomycete *Frankia alni* which is living in root nodules and enabling the trees to colonise extreme sites (Claessens 2003). The most widespread species is common alder (*Alnus glutinosa*) which, due to its ability to withstand permanently waterlogged conditions and prolonged flooding, typically colonises swamps and the banks and flood-plains along slow-flowing lowland streams. Grey alder (*A. incana*) is mainly distributed along fast-flowing white-water rivers and on dry rocky slopes, whereas the shrubby green alder (*A. viridis*) forms the subalpine tree line on heavy soils in the Alps and in the mountains of eastern Europe. The natural distribution of the Italian alder (*A. cordata*) is restricted to Corsica and southern Italy where it colonises moderately dry sites as a pioneer species. Alders have always been considered relatively non-problematic regarding their susceptibility to pests and pathogens. However, in 1993 a previously unknown lethal root and collar rot of *A. glutinosa* was recorded in southern Britain, which occurred mainly along riverbanks, but also in orchard shelterbelts and forest plantings (Brasier et al. 1995). In subsequent years, the disease was also found on *A. incana* and *A. cordata*, and in various countries including Austria, Belgium, Czech Republic, Croatia, Estonia, France, Germany, Hungary, Ireland, Italy, Lithuania, Norway, Poland, Portugal, Spain, Sweden, Switzerland and the Netherlands (Gibbs et al. 1999, 2003, Streito et al. 2002, Santini et al. 2003, Nagy et al. 2003, Jung & Blaschke 2004, Schumacher et al. 2006, Černý & Strnadová 2010, Solla et al. 2010, Jung et al. 2013b, 2016, Redondo et al. 2015, Trzewik et al. 2015, Kanoun-Boulé et al. 2016, M. Horta Jung & T. Jung unpubl. data). Affected trees show small-sized, sparse and often chlorotic foliage, a thinning and dieback of the crown, early and often excessive fructification, and eventually death (Fig. 4a, b). Young trees die within a few months while mature trees with large stem diameters take several years before they die (Gibbs et al. 1999, 2003, Streito et al. 2002, Jung & Blaschke 2004). Mortality rates can reach almost 100 % in permanently waterlogged swamps (Fig. 4a; Jung & Blaschke 2004), on sites with long retention of flood water such as oxbows and stretches behind bridges (Gibbs et al. 1999, Jung & Blaschke 2004) and in direct contact to slow-flowing stream water (Fig. 4b). A disease model, based on data from 35 rivers in north-eastern France, demonstrated that the disease incidence was increasing with decreasing distance of the stem base to the midwater line, with increasing river width, summer temperature of river water and clay content of the riverbank, as well as with slower water flow rates (Thoirain et al. 2007). Crown dieback and mortality are caused by root rot and collar rot lesions which can girdle the stem and extend up to 3 m from the stem base. Lesions are characterised by tarry or rusty exudate spots on the surface of the bark and flame-shaped orange-brown lesions of the inner bark (Fig. 4c–h). Along streams, waterborne inoculum, i.e. zoospores released from sporangia produced on infected alder tissues, is the main source of root and collar infections. Zoospores infect the collar region usually via the non-suberized adventitious roots and through the large lenticels (Gibbs et al. 2003, Jung & Blaschke 2004). During exceptionally high floods, infections can take place higher up the stem producing isolated aerial bark lesions (Fig. 4d). On non-flooded sites, above-ground bark lesions only develop when the pathogen progresses from infected main roots into the collar (Fig. 4f). If trees survive the first year after the infection occurred, lesions may progress in the following spring leaving the older parts sunken and surrounded by callusing tissues (Fig. 4f, i). Often the xylem underneath necrotic lesions shows a flame-shaped staining (Fig. 4j). Infected trees usually produce epicormic shoots in the vicinity of the bark lesions (Fig. 4g).

The causal organisms of the unknown lethal rot on *Alnus* species were identified as a previously unknown swarm of interspecific hybrids originally described as *Phytophthora alni*

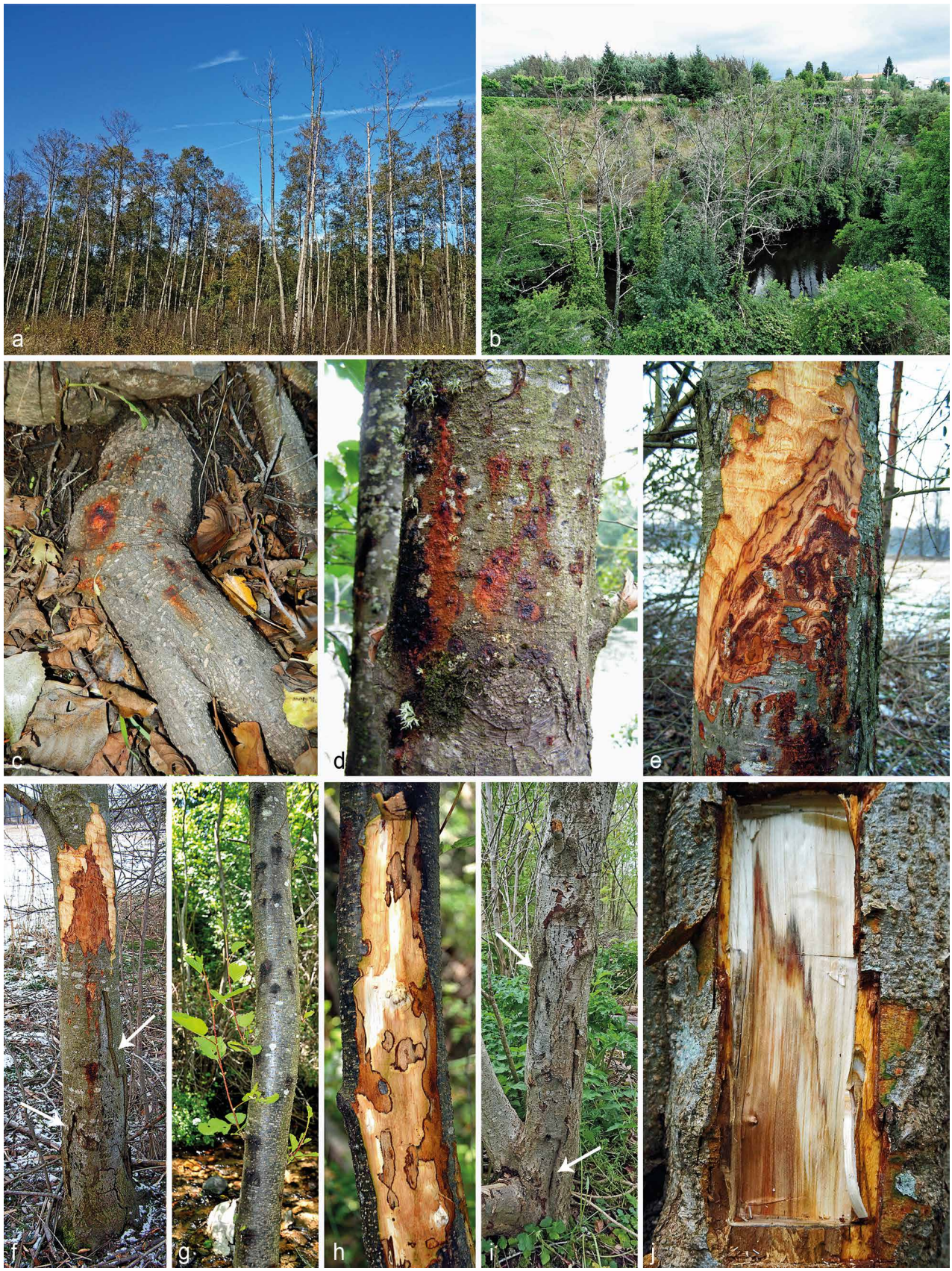

Fig. 4 Symptoms of *Phytophthora* root and collar rot of *Alnus* spp. in Europe. a. Severe dieback and mortality of a mature *A. glutinosa* stand caused by *P.* ×*alni* in a seasonally flooded swamp forest in Bavaria, Germany; b. extensive dieback and mortality caused by *P.* ×*alni* in a riparian stand of *A. glutinosa* at Rio Alva in Portugal; c. bleeding bark lesion caused by *P.* ×*alni* on a surface root of mature riparian *A. glutinosa* in Portugal; d. bleeding aerial bark lesion on a riparian *A. glutinosa* at Rio Miño in Galicia, Spain, caused by simultaneous infections of *P.* ×*alni* and *P.* ×*multiformis* in 2 m stem height during an extreme spring flood; e, f. collar rot lesions caused by *P.* ×*alni* on *A. glutinosa* (e) and *A. incana* (f) planted on a non-flooded site in Bavaria, Germany, with rusty and tarry spots on the outer bark and flame-shaped, orange-brown necrotic lesions of the inner bark; inactive older lesion areas from the previous year are sunken and surrounded by callussing tissues (arrows); g. black exudates oozing from multiple collar rot lesions caused by *P.* ×*multiformis* during a severe flood on a riparian young *A. glutinosa* in Sardinia, Italy, reacting with the production of epicormic shoots; h. same tree as in (g) with multiple, fresh necrotic lesions of the inner bark caused by *P.* ×*multiformis*; i. inactive sunken collar rot lesion with surrounding callusing tissues (arrows) caused by *P. siskiyouensis* on a young *A. cordata* in the UK; j. flame-shaped staining of the xylem tissue underneath an active collar rot lesion caused by *P. siskiyouensis* on a young *A. cordata* in the UK. — Photos: a–f: T. Jung; g, h: B. Scanu; i, j: A. Pérez-Sierra.

s.lat. comprising three subspecies: *P. alni* ssp. *alni* (PAA), *P. alni* ssp. *uniformis* (PAU) and *P. alni* ssp. *multiformis* (PAM) (Brasier et al. 2004). Originally, it was hypothesised that *P.* ×*cambivora* and an unknown species close to *P. fragariae* were the progenitors of the hybrids (Brasier et al. 1999, 2004). However, allele-specific analyses of four single-copy nuclear genes and mtDNA of a European collection of isolates of PAA, PAM, PAU, *P.* ×*cambivora* and *P. fragariae* shed more light on the parental origin of the hybrids:

1. PAU is most likely a distinct non-hybrid species;
2. PAA resulted from a hybridisation between PAU and PAM; and
3. PAM is an ancient hybrid.

Thus, multiple hybridization events must have occurred (Ioos et al. 2006). These results were further confirmed using allele-specific real-time PCR for the quantification of allele copy numbers of three single-copy nuclear genes to assess ploidy levels, and flow cytometry for the determination of genome sizes (Husson et al. 2015). Consequently, the allotriploid PAA, the allotetraploid PAM and the diploid PAU were renamed as *P.* ×*alni*, *P.* ×*multiformis* and *P. uniformis*, respectively (Husson et al. 2015). It is suggested that *P.* ×*multiformis* and *P. uniformis* are of exotic, but different, origin and that the hybridisation that created *P.* ×*alni* occurred in a European nursery where both accidentally introduced parental species met (Brasier et al. 2004, Jung et al. 2017c). Interestingly, *P. uniformis* is common in streams and alder stands in Alaska and Oregon without causing noticeable damage (Adams et al. 2010, Navarro et al. 2015). Using a microsatellite analysis, Aguayo et al. (2013) demonstrated higher genetic diversity of *P. uniformis* isolates from Alaska compared to their European counterparts indicating a North American origin of this species.

In pathogenicity tests, *P.* ×*alni* was significantly more aggressive to the bark of mature *A. glutinosa* logs than *P.* ×*multiformis* and *P. uniformis* (Brasier & Kirk 2001). *Phytophthora* ×*alni*, *P.* ×*multiformis* and *P. uniformis* were shown to be non-pathogenic to a range of other tree species indicating their host specificity to *Alnus* spp. (Brasier & Kirk 2001, Santini et al. 2003). In a comparative pathogenicity trial with different *Alnus* spp., the ranking of susceptibility to *P.* ×*alni* was *A. glutinosa* > *A. incana* and *A. cordata* > *A. viridis* > *A. rubra* (Jung & Blaschke 2006). Despite being only moderately aggressive to European alder species in pathogenicity trials, *P. uniformis* is primarily driving the root and collar rot epidemic of *A. glutinosa* and *A. incana* in Sweden and in the Bavarian Alps, Germany (Jung & Blaschke 2004, Redondo et al. 2015). This is most likely due to the significantly higher frost tolerance of *P. uniformis* compared to *P.* ×*alni* (Černý et al. 2012, J. Schumacher & T. Jung unpubl. data).

In Bavaria, root and collar rot and extensive mortality of *A. glutinosa* and *A. incana* was found in riparian forests along more than 20 000 km of rivers and streams. In the catchments of 58 of 60 river systems studied in detail, the primary source of inoculum could be traced back to numerous, young infested alder plantings established on the riverbanks or on forest sites draining into the rivers (Jung & Blaschke 2004). A similar association was found in 26 river systems in Austria (Jung et al. 2009, 2013b). The presence of the disease in more than 800 young forest and riparian alder plantings in Bavaria (Jung & Blaschke 2004), and the findings of *P.* ×*alni* and *P. uniformis* in 20 and 6.3 %, respectively, of the 64 nursery fields of *Alnus* spp. examined in seven European countries (Jung et al. 2016), clearly demonstrated that the rapid spread and the epidemic extent of this disease in Europe was primarily driven by the widespread planting of infested nursery stock. A range of other *Phytophthora* species, including *P. cactorum*, *P. gonapodyides*, *P. lacustris*, *P. plurivora* and *P. polonica*, are occasionally isolated from small root and collar lesions of *Alnus* spp. without playing a significant role in this epidemic (Gibbs et al. 2003, Jung & Blaschke 2004, Belbahri et al. 2006, Kanoun-Boulé et al. 2016). However, the recent finding of the introduced pathogen *P. siskiyouensis* causing collar rot and mortality of *A. cordata* in the UK (Fig. 4i, j) might pose an additional threat to alder stands in Europe. *Phytophthora siskiyouensis* was previously reported causing collar rot lesions on planted trees of *A. glutinosa* and *A. cordata* in Victoria, Australia, and in California, respectively, and from *A. rhombifolia* and *A. rubra* in riparian forests in Oregon (Smith et al. 2006, Rooney-Latham et al. 2009, Sims et al. 2015a). In underbark inoculation tests, *P. siskiyouensis* caused similar lesion lengths on *A. rubra* as *P. uniformis* (Navarro et al. 2015).

Variation in susceptibility of alder trees to *P.* ×*alni*, observed both in the field and in pathogenicity tests, could be the basis for a successful resistance screening programme which would allow sustainable long-term management of diseased alder stands. For example, in Bavaria, susceptibility of *A. glutinosa* trees, growing in permanent or seasonal contact to infested water on the banks of five rivers with long disease history, was tested and significantly higher tolerance to *P.* ×*alni* was found in many healthy-looking trees as compared to declining trees with root and collar rot symptoms (Jung & Blaschke 2006). Also in Belgium, considerable variation in susceptibility to *P.* ×*alni* was found among *A. glutinosa* trees randomly selected along 34 watercourses (Chandelier et al. 2016).

Decline and mortality of Chamaecyparis lawsoniana in Europe and North America

Chamaecyparis lawsoniana (Lawson's cypress or Port-Orford-cedar, POC) has a limited geographical distribution in humid regions of south-western Oregon and northern California. It grows on a wide range of soil types and sites as a major overstorey component in mixed forests with *Abies concolor*, *A. magnifica*, *Notholithocarpus densiflora*, *Pinus monticola*, *Pseudotsuga menziesii*, *Tsuga heterophylla* and *Sequoia sempervirens* at altitudes from sea level up to 2 000 m (Zobel et al. 1985, Jimerson et al. 2001). POC is also one of the most common ornamental trees worldwide used for amenity plantings, shelterbelts and in hedgerows. The first reports of diseased POC came in 1923 from an ornamental nursery in Seattle, USA, outside of the natural range of the species, and since the early 1950s decline and mortality have been observed in natural forest stands in Oregon. The association with a previously unknown *Phytophthora* species, *P. lateralis*, was established by Tucker & Milbradt in 1942. *Phytophthora lateralis* is a soil-borne pathogen which abundantly produces chlamydospores enabling its survival during dry and hot summers. Homothallic production of oogonia containing oospores was mentioned in the original description (Tucker & Milbradt 1942) but the sexual stage could not be confirmed in later studies (Hansen et al. 2000, Brasier et al. 2012). *Phytophthora lateralis* is a slow growing low-temperature species reproducing and infecting in the Pacific Northwest (PNW; California to British Columbia) mainly during cool and wet conditions in spring, autumn and summer (Hansen et al. 2000). Phylogenetically, *P. lateralis* is the closest known relative of *P. ramorum*, the causal agent of 'Sudden Oak Death' in California and Oregon and 'Sudden Larch Death' in the UK (Brasier et al. 2012, Yang et al. 2017). *Phytophthora lateralis* is highly aggressive to POC (Hansen et al. 2000, Robin et al. 2015). The only other tree species occasionally infected by *P. lateralis* in forests in Oregon is *Taxus brevifolia* but its susceptibility to the pathogen is much lower compared to POC (Murray & Hansen 1997, Hansen et al. 2000). Similar to *P. cinnamomi* in WA, the main pathway of *P. lateralis* in the USA is along roads and paths with infested soil particles

Fig. 5 Symptoms of Port-Orford-cedar (*Chamaecyparis lawsoniana*) decline and mortality caused by *Phytophthora lateralis* in the UK (a–e) and of kauri (*Agathis australis*) dieback caused by *P. agathidicida* in New Zealand (f–h). a, b. Shelterbelts of *C. lawsoniana* containing recently dead trees with sparse brown foliage (white arrows) and declining trees with chlorotic foliage and increased transparency (red arrows); c. *C. lawsoniana* stem with resinous exudates indicating necrotic lesion of the inner bark caused by *P. lateralis*; d. flame-shaped reddish brown inner bark lesion on a *C. lawsoniana* caused by *P. lateralis*, extending from the main roots to more than 1 m stem height; e. flame-shaped reddish brown lesion front caused by *P. lateralis* on a *C. lawsoniana* tree (detail from d); f. mature *A. australis* in a diverse kauri forest showing advanced thinning and dieback of the crown; g. massive stem of a mature *A. australis* with tongue-shaped resinous bark lesion caused by *P. agathidicida*, extending from the main roots up to 2 m stem height; h. resin exudations marking the front of the inner bark lesion caused by *P. agathidicida* on a mature kauri stem (detail from g). — Photos: a–e: A. Pérez-Sierra; f–h: T. Jung.

adhering to tyres of vehicles and boots (Hansen et al. 2000, Goheen et al. 2012). Once introduced to a forest, *P. lateralis* shows efficient and rapid spread downhill and along rivers and streams (Hansen et al. 2000, Jimerson et al. 2001, Jules et al. 2002). *Phytophthora lateralis* has spread throughout the total natural range of POC causing a devastating decline with high mortality rates, which can reach more than 90 % on riparian sites with devastating effects on stream ecology (Hansen et al. 2000, Jimerson et al. 2001, Jules et al. 2002).

Outside the PNW, *P. lateralis* was for the first time isolated from POC in a nursery in France and the infestation was considered eradicated (Hansen et al. 1999). However since 2005, *P. lateralis* has been causing severe decline and mortality of thousands of *C. lawsoniana* trees growing in shelterbelts in Brittany, France (Robin et al. 2011). Soon after, *P. lateralis* has also been recovered from declining POC and *C. pisifera* trees at numerous sites in forests, parks and shelterbelts in England, Scotland and Northern Ireland (Green et al. 2013, Schlenzig et al. 2014). In Europe and the Pacific Northwest, the symptoms of POC decline include chlorosis, wilting and bronzing of foliage, thinning and dieback of the crown and finally death of affected trees (Fig. 5a, b) (Hansen et al. 2000, Robin et al. 2011, Green et al. 2013). These crown symptoms and mortality are caused by root rot and basal stem lesions with resinous exudates on the outer bark and flame-shaped red-brown necrosis of the underlying phloem and cambium (Fig. 5c–e; Hansen et al. 2000, Robin et al. 2011, Green et al. 2013). While seedlings get killed within a few weeks, large trees usually die within one year after appearance of first crown symptoms (Hansen et al. 2000). In both Brittany and the UK, *P. lateralis* was also isolated from aerial bark lesions on stems and branches, and in Brittany also from necrotic foliage suggesting aerial infections (Robin et al. 2011, Green et al. 2013). Foliage infections and aerial bark cankers on stems and large branches, supposedly originating from rain and wind splash inoculum, were also observed earlier in the USA (Roth et al. 1957). In a Scottish nursery, *P. lateralis* was isolated from pale-green discoloured foliage of declining seedlings of *Thuja occidentalis* originally imported from a nursery in France (Schlenzig et al. 2011). In the Netherlands, *P. lateralis* was detected on POC nursery stock (Brasier et al. 2012).

Due to the high aggressiveness of *P. lateralis* to POC and the occurrence of several endemic *Chamaecyparis* species in eastern Asia with comparatively high resistance to *P. lateralis*, an Asian origin of this pathogen has long been suggested (Tucker & Milbradt 1942, Zobel et al. 1985, Hansen et al. 2000). Recently, *P. lateralis* was isolated at several sites in natural, high-altitude cloud forests of Taiwan from soil in wet seeps and from necrotic foliage of mature *C. obtusa* var. *formosana* with generally healthy crowns and non-damaged fine root systems (Brasier et al. 2010, 2012, Webber et al. 2012). A population genetic study using microsatellites demonstrated that isolates from France, the Netherlands and the majority of the UK isolates were identical to isolates from the PNW, whereas the Taiwanese isolates belonged to two distinct evolutionary lineages, designated as TWJ and TWK. Several isolates from Scotland constituted a separate UK lineage which might have resulted from a hybridisation between the PNW and the Taiwanese lineages (Vettraino et al. 2017). The lineages also show considerable phenotypic and morphometric differences (Brasier et al. 2012). In all lineages short preformed sporangial pedicels occur enabling aerial spread and infections of foliage and bark (Brasier et al. 2012). In comparative underbark shoot-dip and root-dip inoculation trials the PNW and TWJ lineages showed higher aggressiveness to POC than the TWK and the UK lineages (Robin et al. 2015). Based on the results of these studies, it was concluded that the European PNW isolates were recently introduced from the USA with infested nursery stock (Vettraino et al. 2017). The exact origin of the PNW lineage in Asia still remains to be found.

The detection of heritable genetic resistance to the PNW lineage of *P. lateralis* in the natural population of POC (Hansen et al. 1989) stimulated a successful long-term resistance screening programme (Hansen et al. 2000, 2011, Oh et al. 2006, Sniezko et al. 2006, 2011). Using stem-dip and root-dip infection trials resistance was found in 1600 of 12000 trees tested (Oh et al. 2006). In greenhouse trials survival rate of seedlings and rooted cuttings from resistant parent trees varied between 25 and 100 % compared to 0–10 % survival of seedlings from susceptible parents (Sniezko et al. 2006). In a long-term outplanting trial on a high disease impact site, seedlings and rooted cuttings from the five most resistant families showed after 16 years 20–80 % survival compared to less than 8 % in progenies from the three most susceptible families (Oh et al. 2006). Several POC families resistant to the PNW lineage of *P. lateralis* were also challenged with the UK, TWJ and TWK lineages and no breakdown of resistance was observed (Robin et al. 2015). These results are promising regarding the re-establishment of this important tree species on dieback sites. The successful POC resistance screening programme could serve as a role model for the management of other *Phytophthora* diseases of forest trees.

Kauri dieback in New Zealand

Kauri (*Agathis australis*), a particularly long-lived ancient conifer species in the *Araucariaceae*, can reach 5 m stem diameter and up to 50 m height. Naturally, it grows widely on the North Island of New Zealand as a keystone species in diverse, mixed lowland forests below 600 m altitude together with other conifers, including *Dacrycrydium cuppressinum*, *Phyllocladus trichomanoides*, *Podocarpus totara*, *P. laetus* and *Prumnopitys ferruginea* (Nicholls 1976, Wardle 1991, Steward & Beveridge 2010). Due to excessive logging in the past, the current distribution of kauri is largely fragmented and relatively pristine old growth stands occupy less than 5 % of the pre-European area (Ahmed & Ogden 1987).

In 1972, *Phytophthora* isolates were recovered from a dying kauri stand on the Great Barrier Island off the coast of the North Island and morphologically identified as *P. heveae* (Gadgil 1974). Apparently, not much attention had been paid to managing this local disease outbreak until dieback and mortality of kauri stands were observed 30 years later on the North Island (Beever et al. 2009, Scott & Williams 2014, Bellgard et al. 2016). Symptoms include thinning, chlorosis and dieback of crowns (Fig. 5f), fine root losses and tongue-shaped collar rot lesions with abundant resin exudations extending several meters up the trunk and also into the main roots (Fig. 5g, h) (Beever et al. 2009, Bellgard et al. 2016). A homothallic *Phytophthora* species, informally designated as *Phytophthora* taxon Agathis (PTA) and later described as *P. agathidicida*, was consistently isolated from necrotic bark lesions and from rhizosphere soil (Beever et al. 2009, Scott & Williams 2014, Weir et al. 2015). Although the smooth-walled oogonia of *P. agathidicida* resemble those of *P. heveae*, multigene phylogenetic analysis placed it closer to *P. castaneae* within *Phytophthora* Clade 5, together with *P. cocois* from Hawaii and *P. heveae* (Weir et al. 2015, Yang et al. 2017). Since its closest relatives *P. castaneae* and *P. heveae* are of Southeast Asian and Australasian origin (Arentz 1986, Brown 1999, Ko et al. 2006, Jung et al. 2017a) it is likely that *P. agathidicida* is also indigenous to this area (Beever et al. 2009, Weir et al. 2015, Bellgard et al. 2016). One original isolate, obtained in 1972 from a dying kauri tree on the Great Barrier Island, could be assigned to *P. agathidicida* and in 2006 this pathogen could again be isolated from kauri collar rot at

Fig. 6 Decline and dieback symptoms caused by *Phytophthora austrocedri* on *Austrocedrus chilensis* in Argentina (a–e) and on *Juniperus communis* in the UK (f, g). a. Extensive mortality of *A. chilensis* at landscape level; b. decline, dieback and mortality of *A. chilensis* at stand level; c. *A. chilensis* trees with thinning and dieback of crowns; d. healthy, declining chlorotic and dead *A. chilensis* (from right to left); e. mature *A. chilensis* stem with flame-shaped necrotic lesion of the inner bark; f. long-dead and recently killed *J. communis* with red-brown foliage; g. tongue-shaped orange-brown lesion extending from the main roots into the collar of a mature *J. communis*. — Photos: a–d: T. Jung; e–g: A. Pérez-Sierra.

this original outbreak site (Beever et al. 2009). It is assumed that *P. agathidicida* did not spread from the Great Barrier Island to kauri stands of the North Island but has rather been present there for decades and collar rot and dieback symptoms have simply been overseen (Beever et al. 2009). A reassessment of the dieback area on the Great Barrier Island revealed an annual rate of disease extension of approximately 3 m, comparable to *P. cinnamomi* in Western Australia (Strelein et al. 2006, Beever et al. 2009). Other *Phytophthora* species like *P. cryptogea*, *P. kernoviae*, *P. multivora* and *P. nicotianae* are occasionally recovered from kauri soils, while *P. cinnamomi* is widespread in kauri forests. Although in general *P. cinnamomi* is not associated with kauri dieback it can be infrequently associated with scattered mortality of individual kauri trees under particularly favouring conditions for the pathogen (Podger & Newhook 1971, Beever et al. 2009, Waipara et al. 2013, Horner & Hough 2014). In both underbark inoculation and soil infestation trials, *P. agathidicida* showed much higher aggressiveness to *A. australis* than *P. cinnamomi*, *P. cryptogea* and *P. multivora*, causing large girdling lesions and high mortality (Horner & Hough 2014). Recent surveys demonstrated a wide distribution of collar rot, dieback and mortality of kauri throughout much of the natural range in the North Island, and *P. agathidicida* has been isolated at many sites from collar rot lesions confirming this pathogen being the causal agent of this epidemic (Waipara et al. 2013, Scott & Williams 2014). *Phytophthora agathidicida* infects trees of all ages and, besides killing mature trees, poses a serious threat to kauri regeneration, hence, resulting in the long-term in an altered forest composition from kauri-dominated forests to forests dominated by *Podocarpus* spp., *D. dacrydioides* and *P. trichomanoides* (Beever et al. 2009, Bellgard et al. 2016).

Decline and mortality of Austrocedrus chilensis and Juniperus communis in Argentina and Europe

Austrocedrus chilensis (Cordilleran cypress or Chilean cedar) is a dioecious evergreen conifer, member of the *Cupressaceae*, and native to the mountains of South Chile and South Argentina. It grows in a wide range of soil types and under different environmental conditions between S36°30' and S43°35' latitude on the eastern slopes of the Andes in Argentina and between S32°39' and S44° latitude on the western slopes of the Andes in Chile (Veblen et al. 1995). Besides its ecological importance, *A. chilensis* is valued for its high timber quality and straight stems used for construction and woodworking (La Manna & Rajchenberg 2004). Decline and mortality of Cordilleran cypress, commonly known as 'mal del ciprés', was first detected in 1948 (Havrylenko et al. 1989, Greslebin et al. 2005) and has since spread throughout its whole range in Argentina. Chlorosis, progressive withering, defoliation, and finally the death of affected trees characterise the decline. Trees can die rapidly and in these cases the foliage turns from chlorotic to red. Such epidemics cause high mortality at landscape level (Fig. 6a–d). Earlier studies discussed several hypotheses regarding the causal agents of the decline including root infections by Pythiaceous pathogens, root decay by basidiomycete fungi, lack or dysfunction of mycorrhizae of Cordilleran cypress, global warming, droughts, and poor drainage and increased precipitation (Havrylenko et al. 1989, Rajchenberg et al. 1998, Filip & Rosso 1999). Although several studies of biotic and abiotic factors tried to elucidate the origin and causes of this decline, it was not before 2007 that a previously unknown *Phytophthora* species, *P. austrocedri* (synonym *P. austrocedrae*), was first described and associated with the mortality of *A. chilensis* in Argentina (Greslebin et al. 2007). *Phytophthora austrocedri* is a homothallic species with very slow growth and a low optimum growth temperature of 17.5 °C. It was placed in Clade 8 where the most closely related species are the soilborne *P. syringae* and *P. obscura*, both causing root rots, cankers and foliar and shoot blights on numerous trees and ornamentals (Grünwald et al. 2012b). *Phytophthora syringae* and *P.* ×*cambivora* were occasionally recovered from the rhizosphere of declining trees but are not involved in the decline (Greslebin et al. 2005, 2007). *Phytophthora austrocedri* was isolated from roots and from collar and stem lesions of symptomatic *A. chilensis* trees. Lesions extend from the roots up the trunk and are characterised by resin exudations at the surface of the bark and brown, flame-shaped lesions of the underlying phloem and cambium (Fig. 6e). Sometimes, a staining of the superficial sapwood underneath bark lesions can be observed. Pathogenicity tests on seedlings, saplings and adult trees demonstrated high aggressiveness of *P. austrocedri* to *A. chilensis* confirming the association of this pathogen with the decline in Argentina (Greslebin & Hansen 2010). The virtually clonal population structure of *P. austrocedri* and its aggressive behaviour on Cordilleran cypress, strongly suggested that it is an alien invasive pathogen in Argentina (Vélez et al. 2013).

Juniperus communis (common juniper) is another dioecious evergreen conifer from the *Cupressaceae*, with a wide holarctic, boreo-temperate distribution ranging from N30° in North Africa to northern Asia, North America and Europe (Preston et al. 2002, Thomas et al. 2007). In 2010, first reports of a serious decline of common juniper came from a National Nature Reserve in northern England (Britain). By 2013, the disease was detected at 19 sites in northern England and Scotland. Dead and dying juniper trees are scattered throughout the affected areas, mainly concentrated on wet, flat ground but also extending uphill across drier slopes. Affected trees and shrubs show fading green colour, reddening or browning foliage on individual branches or the whole crown, and eventually defoliation and tree death (Fig. 6f). Crown symptoms and mortality are caused by orange-brown tongue-shaped lesions in the phloem at the stem collar and along upper roots (Fig. 6g). In some cases, a distinct yellowing of healthy phloem in advance of the lesion margin can be observed on infected trees. *Phytophthora austrocedri* was confirmed as the cause of this dieback and mortality (Green et al. 2012). This pathogen was also identified on two other non-native conifer species in the UK, *Xanthocyparis nootkatensis* (Nootka cypress or Alaskan yellow cedar) (Green et al. 2016) and POC, both like *A. chilensis* and *J. communis* from the *Cupressaceae* family. British isolates from different geographical sites were analysed and also showed limited genetic diversity (Green et al 2015). In Germany, a single isolate of *P. austrocedri* was obtained from a young *J. horizontalis* 'Glauca' in a nursery (Werres et al. 2014).

Recently, Henricot et al. (2017) compared Argentinian, British and German isolates of *P. austrocedri* to clarify the epidemiological and evolutionary relationships between them. Morphological and physiological parameters did not differ significantly between the different populations and in cross-infection experiments both Argentinian and British isolates were pathogenic to the two main hosts *A. chilensis* and *J. communis*. However, phylogenetic analyses of sequences from two mitochondrial and five nuclear gene regions showed that all British isolates were near identical but phylogenetically distinct from the Argentinian and German isolates which share the same genotype. These results indicate that British isolates and Argentinian/German isolates of *P. austrocedri* constitute two evolutionarily distinct lineages originating from the same as-yet-unidentified source population (Henricot et al. 2017).

At the end of 2017, a new case of *P. austrocedri* was reported on a new host, *Cupressus sempervirens* (Italian cypress) in northern Iran (Mahdikhani et al. 2017). The symptoms in affected trees were consistent with those reported on other hosts and included bronzed foliage and an orange-brown lesion in

Fig. 7 Diebacks of natural ecosystems caused by *Phytophthora* spp. in the south-west of Western Australia (WA). a. Severe dieback and mortality of jarrah (*Eucalyptus marginata)* forest with extensive elimination of the previously diverse understorey caused by *P. cinnamomi*; b. high mortality of tuart (*E. gompho-cephala*) on calcaric sandy soils in the Swan Coastal Plain caused by extensive fine root losses due to *P. multivora* infections; c. severe dieback and mortality of riparian flooded gum (*E. rudis*) stand within the jarrah forest caused by *P. elongata* and *P. multivora*; d. extensive dieback and collapse of a *Banksia* woodland caused by *P. cinnamomi*; e. dieback and mortality of mature red tingle (*E. jacksonii*) caused by *P. cinnamomi* and *P. cryptogea* in a humid relic forest at the south coast of WA; f. girdling collar rot lesion caused by *P. cinnamomi* on a young *E. marginata* in the jarrah forest; g. bleeding collar rot lesion caused by *P. cinnamomi* on a mature *Banksia grandis* tree in an open *Banksia* woodland. — Photos: all T. Jung.

the phloem around the collar. To date, decline and canker diseases caused by *P. austrocedri* have been reported from three continents, South America, Europe and western Asia, and so far the host range has been confined to the *Cupressaceae* family.

Diebacks of natural ecosystems in Australia

Apart from tropical regions in north-eastern Queensland and cool-temperate Tasmania, the climate in Australia is characterised by hot and dry summers and relatively low annual precipitations. In the south-west of WA, soil temperatures during summer often exceed 30 °C, and soil water potentials and soil water contents often drop below -6 Mpa (-60 bar) and 1 %, respectively (Shearer & Tippett 1989, Lamont & Bergl 1991, Enright & Lamont 1992, Collins et al. 2011). Although such conditions are generally not believed to favour survival, spread and infection via zoospores of soilborne oomycetes, Australia hosts an impressive diversity of *Phytophthora* species. Since 2009, 19 previously unknown *Phytophthora* species were described from natural ecosystems in WA, including *P. amnicola*, *P. arenaria*, *P. bilorbang* (previously *P.* taxon oak soil), *P. balyanboodja*, *P. boodjera*, *P. condilina*, *P. constricta*, *P. cooljarloo*, *P. elongata*, *P. fluvialis*, *P. gibbosa*, *P. gregata*, *P. kwongonina*, *P. litoralis*, *P. mooyotj*, *P. multivora*, *P. pseudorosacearum*, *P. thermophila* and *P. versiformis* (Scott et al. 2009, Rea et al. 2010, 2011, Jung et al. 2011, Crous et al. 2011, 2012, 2014, Aghighi et al. 2012, Simamora et al. 2015, Paap et al. 2017, Burgess et al. 2018). A metagenomic survey of *Phytophthora* diversity at 640 natural sites across Australia demonstrated presence of 68 *Phytophthora* phylotypes, of which 21 were potentially new taxa and another 25 were previously not found in natural ecosystems or were new introductions to Australia (Burgess et al. 2017). With presence at 44.7 and 34.2 % of the sites, respectively, *P. multivora* and *P. cinnamomi* were by far the most common species. Interestingly, these two introduced wide-host range pathogens also have the most deleterious impact on Australian natural ecosystems.

During the long isolation from other continents, a diverse and largely endemic flora evolved in Australia which, due to a lack of co-evolution, contains a high proportion of plant species susceptible to non-native introduced *Phytophthora* species. Alone in the south-western Botanical Province of WA, 40 % of the 5710 endemic plant species from 39 families are susceptible to *P. cinnamomi* (Shearer et al. 2004). Recently, pathogenicity of *P. cinnamomi* and 21 other *Phytophthora* taxa from WA, most of them recently described or informally designated new species, to seven native plant species from WA, including *Eucalyptus marginata* (jarrah), *Banksia grandis*, *B. littoralis*, *B. occidentalis*, *Casuarina obesa*, *Corymbia calophylla* (marri) and *Lambertia inermis*, was demonstrated (Belhaj et al. 2018). *Eucalyptus marginata* and the three *Banksia* species were susceptible to the highest number of *Phytophthora* taxa whereas *C. calophylla* showed the highest resistance to all 21 *Phytophthora* taxa (Belhaj et al. 2018). Australia has a long history of diebacks of various eucalypt forests, *Banksia* woodlands and heathlands, which are particularly severe in the Mediterranean climates of WA (Shearer & Tippett 1989, Shearer & Dillon 1995, 1996, Shearer et al. 2004) and Victoria (Weste & Marks 1987, Marks & Smith 1991, Laidlaw & Wilson 2003, Weste 2003, Cahill et al. 2008). In WA, the most widespread forest type is the dry sclerophyll jarrah forest which is largely dominated in the overstorey by *E. marginata*, while the understorey contains a diverse flora mainly from the *Proteaceae*, *Dilleniaceae*, *Epacridaceae*, *Fabaceae* and *Xanthorrhoeaceae* (Shearer & Tippett 1989). First symptoms of dieback and mortality in the jarrah forest were reported during the 1920s but it took 40 years until the association with the presence of *P. cinnamomi* in the rhizosphere was established (Podger et al. 1965, Podger 1972, Shearer & Tippett 1989). Fifty years of intense research has produced a rich body of knowledge on the aetiology, site relations and dynamics of jarrah dieback and numerous other diebacks in WA and eastern Australia, the ecology, host range and pathways of *P. cinnamomi*, and the role that other *Phytophthora* spp. are playing. Although *P. elongata*, *P. gibbosa*, *P. gregata*, *P. litoralis*, *P. multivora*, *P. thermophila* and *P. versiformis* can cause local dieback and mortality of understorey species and sometimes also of *E. marginata* (*P. multivora*, *P. elongata*), the dieback of the jarrah forest is mainly driven by two clonal lineages of *P. cinnamomi* from the A2 mating type (Shea 1975, Shea et al. 1982, Shearer & Tippett 1989, Dobrowolski et al. 2003). The main pathway of *P. cinnamomi* in the jarrah forest and other natural ecosystems in WA is the accidental transport of infested soil adhering to vehicles and boots and the use of infested gravel material for building of forest roads (Shea et al. 1983, Shearer & Tippett 1989, Marks & Smith 1991). A few months after the introduction of *P. cinnamomi* to a site, first symptoms of chlorosis and wilting appear on highly susceptible understorey species, in particular *B. grandis*, *Xanthorrhoea preissii* and *Macrozamia riedlei*, hence, they are used as indicator species for presence of *P. cinnamomi* (Shea 1979, Shearer & Tippett 1989). The pathogen usually moves through the forest via zoospores and root-to-root contact with more or less sharply defined infection fronts (Weste & Marks 1987, Shearer & Tippett 1989). An extensive study at 55 sites across WA using aerial photography demonstrated mean disease progression rates of 1.4 m per year for uphill extension and 9.5 m per year across slopes (Strelein et al. 2006). Downhill progression is much faster reaching up to 400 m per year in Victoria (Weste & Law 1973). After several years, the majority of midstorey and understorey species are affected by dieback and mortality and highly susceptible species can be eliminated while the *E. marginata* overstorey shows severe thinning, chlorosis, wilting and dieback of the canopy and mortality of scattered trees or in groups up to several hectares in size (Fig. 7a) (Podger 1972, Shearer & Tippett 1989, Jung et al. 2013a). Although in jarrah the rate of symptom expression is varying depending on the site conditions, other tree species, in particular marri and bullich (*E. megacarpa*) can persist much longer. In *E. marginata*, *P. cinnamomi* causes excessive fine root losses, which interact with the extreme summer droughts leading to gradual decline and eventually dieback and mortality (Podger 1972, Shea 1975, Crombie et al. 1987, Shearer & Tippett 1989). In addition, *P. cinnamomi* can infect large woody roots and produce bark lesions, which on sites with impeded drainage due to a concrete lateritic hardpan close to the surface may girdle the vertical roots just above the lateritic layer resulting in acute wilting and mortality (Shea et al. 1982, Tippett et al. 1983, Shearer & Tippett 1989). Collar infections on jarrah are rare and mostly restricted to waterlogged sites or young trees (Fig. 7f; Shearer & Tippett 1989). Burgess et al. (1999) demonstrated increased susceptibility of jarrah stems to *P. cinnamomi* infection under conditions of root hypoxia. About 20 % of the 64 000 km^2 covered by the jarrah forest are already infested by *P. cinnamomi* and the structural and floristic changes it caused are unprecedented (Fig. 7a). Recently, it has been shown that *P. cinnamomi* survives the hot and dry summer conditions in WA within infected fine roots and small woody roots of woody host and non-host species and annual herbs. Particularly thick-walled oospores produced by selfing of the A2 mating type, stromata-like hyphal aggregations and intracellular hyphae encased by callose sheaths (lignitubers) serve as long-term survival structures, while the thin-walled chlamydospores enable short-term survival between consecutive rain events (Crone et al. 2013, Jung et al. 2013a).

Across its native range and in particular in the Swan coastal plane south of Perth, tuart (*E. gomphocephala*) forests, growing

on dry and sandy limestone sites, are suffering for almost 20 years from a severe decline characterised by thinning and dieback of the crowns and high mortality rates (Fig. 7b; Edwards 2004, Archibald 2006, Scott et al. 2009). Several environmental factors have initially been considered responsible for tuart decline until a firm association with the previously undescribed pathogen *P. multivora* was established (Scott et al. 2009). In pathogenicity tests, *P. multivora* demonstrated high aggressiveness to the fine root system of *E. gomphocephala* (Scott et al. 2012). In the tuart forest, *P. multivora* causes progressive fine root losses which exacerbate the severe drought stress during summer on the sandy soils predisposing affected trees to attacks by stem borers (Scott et al. 2009). Other tree species like *Agonis flexuosa* (peppermint) are also affected, but to a lesser extent (Scott et al. 2009). *Phytophthora multivora* has a wide host range in WA and in other continents whereas in South Africa it is widespread in natural ecosystems without causing visible disease symptoms suggesting long-term coevolution (Scott et al. 2009, Oh et al. 2013, Jung et al. 2016). The acidophilic *P. cinnamomi* is generally absent from these calcaric sites in WA and, hence, is not involved in tuart decline. Also in the south-west of WA, riparian gallery forests of flooded gum (*E. rudis*) along many rivers and streams are showing since the 1970s increasingly severe thinning and dieback of crowns with high levels of mortality (Fig. 7c). For a long time, attacks by psyllids and leaf miners and various environmental factors, including rising salinity levels of river water due to agricultural mismanagement, have been discussed as causal agents, but a satisfactory disease aetiology was not established (Curry 1981, Abbott 1999, Yeomans 1999, Clay & Majer 2001). However, recently *P. elongata* and *P. multivora* were regularly isolated along many rivers from the rhizosphere of dying riparian *E. rudis* trees with extensive fine root losses (Edwards et al. 2010). It seems likely that this widespread devastating decline is driven by root damage caused by these invasive *Phytophthora* species interacting with other biotic and abiotic factors including insect defoliations, droughts and salinity. *Phytophthora elongata* has a clonal population structure in WA and its origin is still unknown. It was most likely introduced to the jarrah forest during the 1970s with infested nursery stock used for rehabilitation plantings of mine sites, and subsequently spread into streams and river systems (Rea et al. 2010).

In WA, highly diverse *Banksia* woodlands and Kwongan heathlands constitute the climax vegetation in the northern and southern sandplains and on other sites too dry for supporting forest growth. In these ecosystems *P. cinnamomi* and many other *Phytophthora* spp. are causing fine root losses in susceptible plant species leading to dieback and bleeding collar lesions (Fig. 7g) which may girdle the plants resulting in acute wilting and mortality (Fig. 7b; Scott et al. 2009, Shearer et al. 2009, Rea et al. 2011, Jung et al. 2013a). Pathogenicity of 22 *Phytophthora* taxa to three *Banksia* species and *L. inermis* was recently demonstrated (Belhaj et al. 2018). While the invasive *P. cinnamomi* usually moves through these ecosystems in clearly visible infection fronts with high mortality rates, potentially indigenous species like *P. arenaria* from Clade 4, *P. constricta* from Clade 9, *P. balyanboodja*, *P. condilina*, *P. cooljarloo*, *P. kwongonina* and *P. pseudorosacearum* from Clade 6a and *P. gibbosa*, *P. gregata*, *P. litoralis* and *P. thermophila* from Clade 6b cause scattered or patchy dieback which is usually associated with episodic, unseasonal heavy rain events (Rea et al. 2011, Jung et al. 2011, Burgess et al. 2018).

In the humid south-western-most corner of WA, a tertiary relic forest dominated by particularly tall and long-lived tree species like karri (*E. diversicolor*), red tingle (*E. jacksonii*), yellow tingle (*E. guilfoylei*), Rates tingle (*E. brevistylis*) and marri is growing in a small area which is mostly protected within the Walpole-Nornalup National Park. For about a decade, numerous red tingle trees are suffering from a severe dieback and mortality which is associated with root infections by *P. cinnamomi* and *P. cryptogea* (Fig. 7e; T. Jung unpubl.). Due to the small natural habitat of red tingle this disease might threaten the survival of this endangered iconic tree species in nature.

In Victoria, severe diebacks occur in eucalypt forests and *Banksia* woodlands, in particular in the Grampians, in East and South Gippsland, the Wilsons Promontary and the Brisbane Ranges National Parks (Marks & Smith 1991). Symptomatologies, disease aetiologies, dynamics and site relations are largely similar to the situation in WA (Weste & Marks 1987, Marks & Smith 1991, Wilson et al. 2000, Weste 2001, Cahill et al. 2008). A 30-years study at 13 sites in representative eucalypt forests, woodlands and heathlands across Victoria demonstrated a gradual decline of *P. cinnamomi* inoculum to very low levels with progressing elimination of highly susceptible plant species from infested sites and their natural replacement by highly resistant species. After 20–30 years, substantial regeneration and survival of 30–40 previously eliminated susceptible plant species like *Xanthorrhoea australis* was observed, while the crowns of affected overstorey trees showed no recovery (Weste 2003). Future surveys are needed to assess whether the ecosystem recoveries will be sustainable or whether *P. cinnamomi* inoculum levels will increase again resulting in new dieback cycles.

In the tropical rainforests of northern Queensland, an extensive *Phytophthora* survey revealed presence of 10 *Phytophthora* species at 55 % of the 1 897 sites tested. More than 13 000 isolates were obtained of which 86 % and 9 % were *P. cinnamomi* and *P. heveae*, respectively. The remaining isolates belonged to *P. boehmeriae*, *P. castaneae*, *P. citricola* s.lat., *P. cryptogea*, *P. drechsleri*, *P. meadii*, *P. nicotianae* and *P. palmivora* (Brown 1999). *Phytophthora cinnamomi* was more frequently isolated from dead and dying than from healthy forests, and at nine sites this pathogen was associated with patch dieback of rainforest trees. All other *Phytophthora* spp. showed no association with disease (Brown 1999). The absence of large-scale dieback of forests might be explained by the constantly warm and humid climate which is favouring the trees more than *P. cinnamomi*, or indicate long-term coevolution between the flora and the pathogen, which could have spread from New Guinea to Queensland via a landbridge during the pleistocene. However, the presence of the A1 mating type at only four of the 646 sites infested by *P. cinnamomi* does not support the latter hypothesis, since in Papua New Guinea the A1 mating type is more widespread and considered an ancient introduction whereas the A2 mating type was most likely introduced in modern times (Arentz 1983, 2017, Arentz & Simpson 1986).

A recent survey in the diverse Gondwana rainforests of southern Queensland and northern New South Wales demonstrated the presence of eight *Phytophthora* species, including *P. cinnamomi*, *P. cryptogea*, *P. frigida*, *P. heveae*, *P. macrochlamydospora*, *P. multivora*, the previously unknown *P. gondwanensis* and another unknown *Phytophthora* taxon (Scarlett et al. 2015). Due to their wide host ranges and their high virulence, *P. cinnamomi* and *P. multivora* might pose a serious threat to the Gondwana rainforests which contain more than 200 rare and endangered plant species (Crous et al. 2015, Scarlett et al. 2015). Also in New South Wales, both *P. multivora* and *P. cinnamomi* were recovered from declining trees of the 'living fossil' *Wollemia nobilis* (Wollemi pine) in its only natural site and their high aggressiveness to this critically endangered tree species has been demonstrated (Puno et al. 2015).

Decline and dieback of the Mediterranean maquis vegetation

Mediterranean-type ecosystems, with their characteristic and unique climatic regimes of mild wet winters and warm and dry summers, occur just in five regions of the world: California, Central Chile, the Mediterranean Basin, South Africa and south-western and South Australia (Peel et al. 2007). These Mediterranean climate regions harbour a remarkable and globally significant level of plant diversity and endemism, accounting for almost 20 % of all plant species in the world (Myers et al. 2000, Cowling et al. 2006). In response to the climate, similar woody, shrubby plants, with evergreen sclerophyll leaves, have developed in communities of varying density. The names for the shrub vegetation vary by region because of language and plant structure, including 'maquis' and 'garrigue' in the Mediterranean Basin, 'chaparral' in California, 'matorral' in Chile, 'fynbos' or 'renosterveld' in South Africa and 'mallee' scrubs and shrublands and 'kwongan' heathlands in Australia.

The maquis vegetation in the Mediterranean Basin is characterised by scrub, sparse grass and scattered evergreen trees with a maximum size of 2–3 m, which differ in structure and species richness depending on local conditions (Spano et al. 2013). Since 2010, in the National Park of La Maddalena archipelago, off the northern coast of Sardinia, extensive dieback and mortality of a wide range of plant species, typical of the Mediterranean maquis in the archipelago as well as in other maquis sites in Sardinia, has been recorded across slopes downhill of roads and trekking paths (Fig. 8a, b) (Scanu et al. 2015). The main species affected are *Arbutus unedo*, *Asparagus albus*, *Cistus* sp., *Erica* spp., *Juniperus phoenicea*, *J. oxycedrus*, *Pistacia lentiscus* and *Rhamnus alaternus* (Scanu et al. 2015). Amongst the most susceptible species, *J. phoenicea*, *J. oxycedrus* and *A. unedo* show a wide range of symptoms including partial or complete dieback of the crown and abnormal production of epicormic shoots, and reddening or browning of drying foliage on dying and recently dead trees and shrubs (Fig. 8c, d). Crown symptoms are associated with extensive losses of both lateral small woody roots and fine roots, opening cankers and the presence of basal phloem lesions extending from the main roots up the stems (Fig. 8e). Root and collar rot on juniper trees and shrubs frequently occurs in low-laying areas with seasonal waterlogging. In wetlands, other tree/shrub species like *P. lentiscus* and *R. alaternus* also show severe crown thinning, dieback of single branches and mortality (Fig. 8b, f), which are caused by root and collar rot. Ground layer species such as *A. albus* (Fig. 8g) and *Cistus* spp. are also commonly affected showing chlorosis, wilting and dieback.

An unexpected high diversity of *Phytophthora* species has been found associated with the decline and dieback of this diverse ecosystem. In total 10 *Phytophthora* species were isolated from rhizosphere soil samples collected from declining Mediterranean maquis vegetation and stream catchments in the National Park of La Maddalena archipelago, including *P. asparagi*, *P. bilorbang*, *P. cinnamomi*, *P. crassamura*, *P. gonapodyides*, *P. melonis*, *P. ornamentata*, *P. parvispora*, *P. pseudocryptogea* and *P. syringae* (Scanu et al. 2015). The most common species detected were *P. asparagi* and *P. bilorbang*, both from Clade 6 (Jung et al. 2011, Aghighi et al. 2012). While *P. bilorbang* appears to be a common species in natural environments (Aghighi et al. 2012, Sims et al. 2015b), *P. asparagi* was previously only reported from horticultural and ornamental plants (Cunnington et al. 2005, Saude et al. 2008, Jung et al. 2016). Likewise, also *P. melonis* was previously only known from agriculture causing a severe disease of members of the *Cucurbitaceae* in Asia (Ho et al. 2007). This suggests the possible introduction of both *P. asparagi* and *P. melonis* with infested plant material (Moralejo et al. 2009, Jung et al. 2016). Two previously unknown species, *P. crassamura* and *P. ornamentata* have been described from *J. phoenicea* and *P. lentiscus*, respectively, in Sardinia (Scanu et al. 2015). While *P. crassamura* is widespread across continents with different climatic conditions (Brasier et al. 2003, Burgess et al. 2009), *P. ornamentata* seems to be restricted to the Mediterranean Basin (Scanu et al. 2015), although it has recently been detected from *C. calophylla* plants in Australia (G.E.St.J. Hardy pers. comm.). In pathogenicity trials, all *Phytophthora* species demonstrated pathogenicity to *J. phoenicea* and *P. lentiscus* (Scanu et al. 2015). On *J. phoenicea* the most aggressive pathogens were *P. asparagi* and *P. bilorbang*, killing 50 % and 37.5 % of inoculated plants, respectively, while *P. cinnamomi* caused 100 % mortality on *P. lentiscus*. *Phytophthora asparagi*, *P. crassamura*, *P. bilorbang*, *P. melonis* and *P. ornamentata* were also highly aggressive to *P. lentiscus* (Scanu et al. 2015).

Phytophthora cinnamomi was isolated from *R. alaternus* showing severe dieback and wilting (Scanu et al. 2015) and together with its closest relative *P. parvispora*, this pathogen was also isolated from *A. unedo*, another common species in Mediterranean maquis ecosystems (Scanu et al. 2014a). Also in Portugal, *P. cinnamomi* has been isolated from declining and wilting *A. unedo* plants in Mediterranean maquis vegetation (T. Jung & M. Horta Jung unpubl. data). *Phytophthora cinnamomi* was also found causing a severe decline and mortality of *Erica umbellata*, a small heather species native to the western Iberian Peninsula and northern Morocco, in a protected natural area in Extremadura (Spain) (Acedo et al. 2013). Several understory *Ericaceae* and *Cistaceae* scrubs commonly occurring in declining Mediterranean oak stands in Spain and Portugal were also found to be infected by *P. cinnamomi* (Brasier et al. 1993, Moreira & Martins 2005). These plant species are suspected to act as a reservoir of pathogen inoculum, contributing to the spatial distribution of *P. cinnamomi* (Moreira & Martins 2005). The frequent detection of *P. cinnamomi* from Mediterranean plant species in recent years indicates that this pathogen is currently spreading into the maquis vegetation ecosystems. Against the background of a predicted increase of *P. cinnamomi* activity under current climate change projections (Brasier & Scott 1994, Burgess et al. 2017), the severe destructions of the root system caused by *P. cinnamomi* in pathogenicity tests on *A. unedo*, *E. umbellata*, *J. phoenicea* and *P. lentiscus* (Acedo et al. 2013, Scanu et al. 2014a, 2015) suggest that this polyphagous pathogen has the potential to threaten the native maquis vegetation in the Mediterranean basin on a large scale.

Decline and dieback of European beech in Europe and the USA

European beech (*Fagus sylvatica*) is characterised by high shade tolerance and growth capacity, a wide climatic and geological amplitude ranging from atlantic to continental climate and from moderately dry to periodically wet soils with pH ranging from < 3 to > 7. Due to its high competitiveness, European beech would naturally dominate more than 50 % of the forests at hilly to mountainous sites in Western and Central Europe and in mountain areas of Eastern and Southern Europe (Walentowski et al. 2004, Ellenberg & Leuschner 2010).

Since the mid 1990s, forests and amenity stands of European beech across the entire natural range in Europe are increasingly threatened by a severe decline and dieback (Motta et al. 2003, Jung et al. 2003b, 2005, 2009, 2013b, Cacciola et al. 2005, Hartmann et al. 2006, Orlikowski et al. 2006, Brown & Brasier 2007, Munda et al. 2007, Vettraino et al. 2008, Černý et. al. 2009, Jung 2009, Schmitz et al. 2009, Stępniewska & Dłuszyński 2010, Telfer et al. 2015). Also in the USA and in Chile, European beech plantations experience a similar decline and mortality (Jung et al. 2005, 2018, Nelson 2009, Weiland

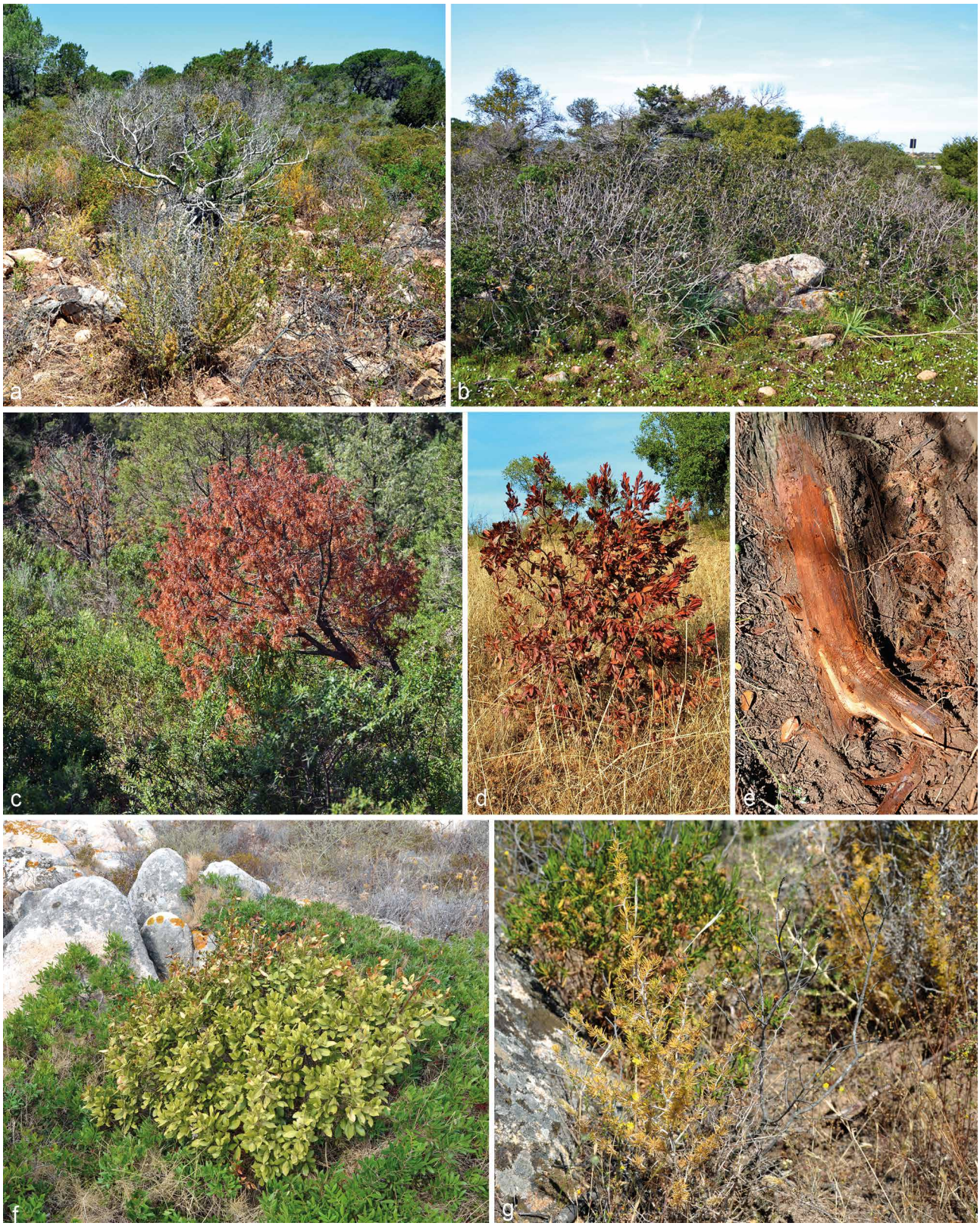

Fig. 8 Decline and dieback symptoms on maquis vegetation caused by *Phytophthora* spp. in the Mediterranean basin. a. Extensive dieback and mortality of shrub species due to *P. cinnamomi*; b. *Pistacia lentiscus* showing severe dieback and mortality caused by *P. ornamentata*; c. mature tree of *Juniperus oxycedrus* with severe wilting and red discoloration of the foliage; d. large orange-brown lesion extending from the main root into the collar of a mature *J. phoenicea*; e. young *Arbutus unedo* recently killed by *P. cinnamomi*; f. chlorosis, wilting and dieback of *Rhamnus alaternus* caused by *P. cinnamomi*; g. chlorosis, wilting and dieback of *Asparagus albus* caused by *P. asparagi*. — Photos: a–c, e–g: B. Scanu; d: T. Jung.

Fig. 9 Decline and dieback symptoms caused by *Phytophthora* spp. on *Fagus sylvatica*. a. Scattered mortality and patch dieback and chlorosis (arrow) due to root and collar rot and aerial bleeding cankers caused by *Phytophthora plurivora* and *P.* ×*cambivora* in a mountain forest in the Bavarian Alps. Germany; b. severe dieback and mortality due to root and collar rot caused by *P.* ×*cambivora* in Austria; c. chlorosis, microphylly, thinning and dieback due to root losses caused by *P. cactorum* and *P. plurivora* in Bavaria; d. small woody roots with extensive losses of lateral roots and fine roots caused by *P. cactorum* and *P. plurivora* in Bavaria; e. bleeding collar rot lesion and aerial bleeding canker caused by *P.* ×*cambivora* in a mountain forest in the Bavarian Alps; f. bleeding collar rot lesion caused by *P.* ×*cambivora* in a Swedish forest; g. collar rot lesion with orange- to dark-brown exudates on the outer bark and flame-shaped lesion front in the inner bark caused by *P. plurivora* in Sweden. — Photos: all T. Jung.

et al. 2010). The symptoms are typical for *Phytophthora* diseases and comprise small-sized and often yellowish foliage, thinning and dieback of crowns, extensive losses of fine roots and lateral roots, collar rot, and aerial bleeding cankers along the stem up to the canopy (Fig. 9). A local epidemic with similar symptoms was recorded in the 1930s in the UK and could be associated to *Phytophthora* infections (Day 1938). Severe destructions of the fine root system are common in affected forests and lead to a slow chronic decline, whereas bleeding collar rot and aerial bark cankers have a scattered or patchy distribution and usually cause rapid mortality (Jung et al. 2005, 2009, 2013b).

Several independent surveys in 17 European countries showed that more than 80 % of the almost 300 beech stands growing on a wide range of geological substrates with soil pH ranging from 3.3 to 7.8 were infested with in total 15 *Phytophthora* taxa. Most widespread were *P.* ×*cambivora*, *P. plurivora* and, in urban situations, also *P. cactorum*. *Phytophthora* ×*cambivora* was frequently encountered on acidic heavy soils, while *P. plurivora* was more common in calcaric soils. Other species had a scattered distribution like *P. chlamydospora*, *P. europaea*, *P. gonapodyides*, *P. pseudosyringae*, *P. psychrophila*, *P. quercina*, *P. syringae*, *P. tubulina* and *P. uliginosa*, or were restricted to areas with mild winters like *P. cinnamomi* in Portugal and the UK (Jung & Blaschke 1996, Balci & Halmschlager 2003a, Jung et al. 2003b, 2005, 2013b, 2017b, Motta et al. 2003, Cacciola et al. 2005, Hartmann et al. 2006, Orlikowski et al. 2006, Brown & Brasier 2007, Munda et al. 2007, Vettraino et al. 2008, Černý et. al. 2009, Jung 2009, Schmitz et al. 2009, Stępniewska & Dłuszyński 2010, Telfer et al. 2015). The airborne pathogens *P. kernoviae* and *P. ramorum* exclusively occurred in the UK where the humid climate allows the continuous production of caducous sporangia on infected foliage of adjacent *Rhododendron ponticum* plants and their aerial dispersal onto neighbouring beech stems where they cause extensive bleeding lesions (Brasier et al. 2005, Brown & Brasier 2007, Jung et al. 2013b). In the eastern USA, *P. cactorum*, *P. plurivora* and its close relative *P. pini* (previously *P. citricola* I) are causing root and collar rot and mortality on mature *F. sylvatica* in amenity plantings (Jung et al. 2005, Weiland et al. 2010). *Phytophthora cactorum*, *P.* ×*cambivora*, *P. kernoviae*, *P. plurivora* and *P. ramorum* proved to be highly aggressive to the bark and root systems of both mature and young beech trees (Fleischmann et al. 2002, Brasier & Jung 2003, Jung et. al. 2003b, 2017b, Brown & Brasier 2007, Vettraino et al. 2008, Weiland et al. 2010). In Europe, all five species are considered as exotic invasive pathogens (Jung 2009, Jung & Burgess 2009, Jung et al. 2013b, 2016).

Detailed investigations in Germany, Austria and the UK showed that aerial bleeding cankers on stems of mature beech trees are in most cases not following the classical pattern of the phenomenon 'Beech Bark Disease'. The latter is considered as complex interaction of predisposing drought stress, colonisation of the bark by the scale insect *Cryptococcus fagisuga*, infection of the colonised bark by the secondary parasite *Neonectria coccinea* and, eventually, invasion of the stem by wood decay fungi (Parker 1974, Lonsdale & Wainhouse 1987). However, this aetiology is apparently restricted to young pole-stage stands (Lonsdale & Wainhouse 1987) while in mature stands *Phytophthora* pathogens are the primary disease agents (Jung et al. 2005, 2013b, Brown & Brasier 2007, Jung 2009). Both soilborne *Phytophthora* species, in particular *P. plurivora*, *P.* ×*cambivora* and *P. gonapodyides*, and the airborne *P. cactorum*, *P. kernoviae*, *P. pseudosyringae* and *P. ramorum* can infect stem bark and shoots of beech via rain- or wind-splash dispersal of sporangia or progression from root lesions into the collar (Jung et al. 2005, 2013b, Brown & Brasier 2007, Jung 2009, Nechwatal et al. 2011). After establishment in the bark, *Phytophthora* pathogens can spread non-symptomatically in the xylem causing multiple aerial cankers along the stem (Brown & Brasier 2007).

The onset of the *Phytophthora* epidemics in Austria, Germany and Sweden was triggered by the succession of continuously wet conditions during the growing season followed by extreme drought in the same or the following year (Jung 2009, Jung et al. 2013b, T. Jung, T. Corcobado & T. Cech unpubl., T. Jung & J. Witzell unpubl. data).

A large-scale survey of nursery stands and young plantings of *F. sylvatica* in Europe revealed that 80 % of the nursery stands and almost 100 % of the plantings tested were infested with 9 and 11 *Phytophthora* species, respectively (Jung et al. 2016). Most common were *P.* ×*cambivora*, *P. plurivora* and *P. cactorum* demonstrating the importance of the nursery pathway for the spread of this devastating disease into beech stands.

AIRBORNE PHYTOPHTHORA DISEASES OF FORESTS AND WOODLANDS

Dieback and mortality of Nothofagus species in the UK and Chile

The genus *Nothofagus* is native to the Southern Hemisphere, where it constitutes an important component of temperate rainforests and mountainous forests (Kirkpatrick & DellaSala 2011, Tecklin et al. 2011) and represents an important economic and silvicultural resource (Heenan & Smissen 2013). Since the early 1900s, several *Nothofagus* species have been planted in the UK due to their fast growth and high wood quality (Danby 1991, Webber et al. 2011). Apart from very occasional infections by *P. ramorum* (Webber 2008), no other pests and pathogens have been recorded on *Nothofagus*. However, in 2009 severe dieback and mortality of *Nothofagus* trees, associated with bleeding cankers on stems and main branches, were reported in the UK (Scanu et al. 2012). Due to the similarity to aerial cankers caused by *P. ramorum* on *F. sylvatica* trees in the UK (Brown & Brasier 2007) this pathogen was initially thought to be the causal agent of this new disease outbreak. However, isolations from necrotic bark tissues revealed the presence of a homothallic *Phytophthora* species that was subsequently identified as *P. pseudosyringae* (Scanu & Webber 2016).

Nothofagus obliqua and *N. alpina* are the main species affected in the UK. Both species are affected by severe dieback and mortality of mature and young trees (Fig. 10a–c). At early stages, trees show symptoms of crown thinning, wilting and branch dieback (Fig. 10b, c), which are often associated with abundant proliferation of epicormic shoots on stems and branches. These symptoms are caused by up to 2 m long bleeding bark lesions on the main stem (Fig. 10d). Necrotic lesions often progress deep into the xylem (Fig. 10e) resulting in multiple bleeding aerial lesions along the stem and in branches, which may girdle the trunk and branches entirely, causing branch and crown dieback and wilting foliage. Up to 60 discrete lesions can be observed on a single tree and these can reach 16 m above ground level (Scanu & Webber 2016). In the canopy, shoot blight and necrosis on small branches, twigs and leaves are common (Fig. 10f–i). During dry conditions, lesion growth on small branches and twigs ceases, resulting in sunken cankers with cracking bark (Fig. 10f) and necrotic underlying xylem tissues. Although the pathogen was detected in rhizosphere soil samples, symptoms of root infection are largely absent (Scanu & Webber 2016).

Field observations and the high sporulation rate of *P. pseudosyringae* on *Nothofagus* foliage strongly indicate that aerial bark infections occur directly through penetration of intact

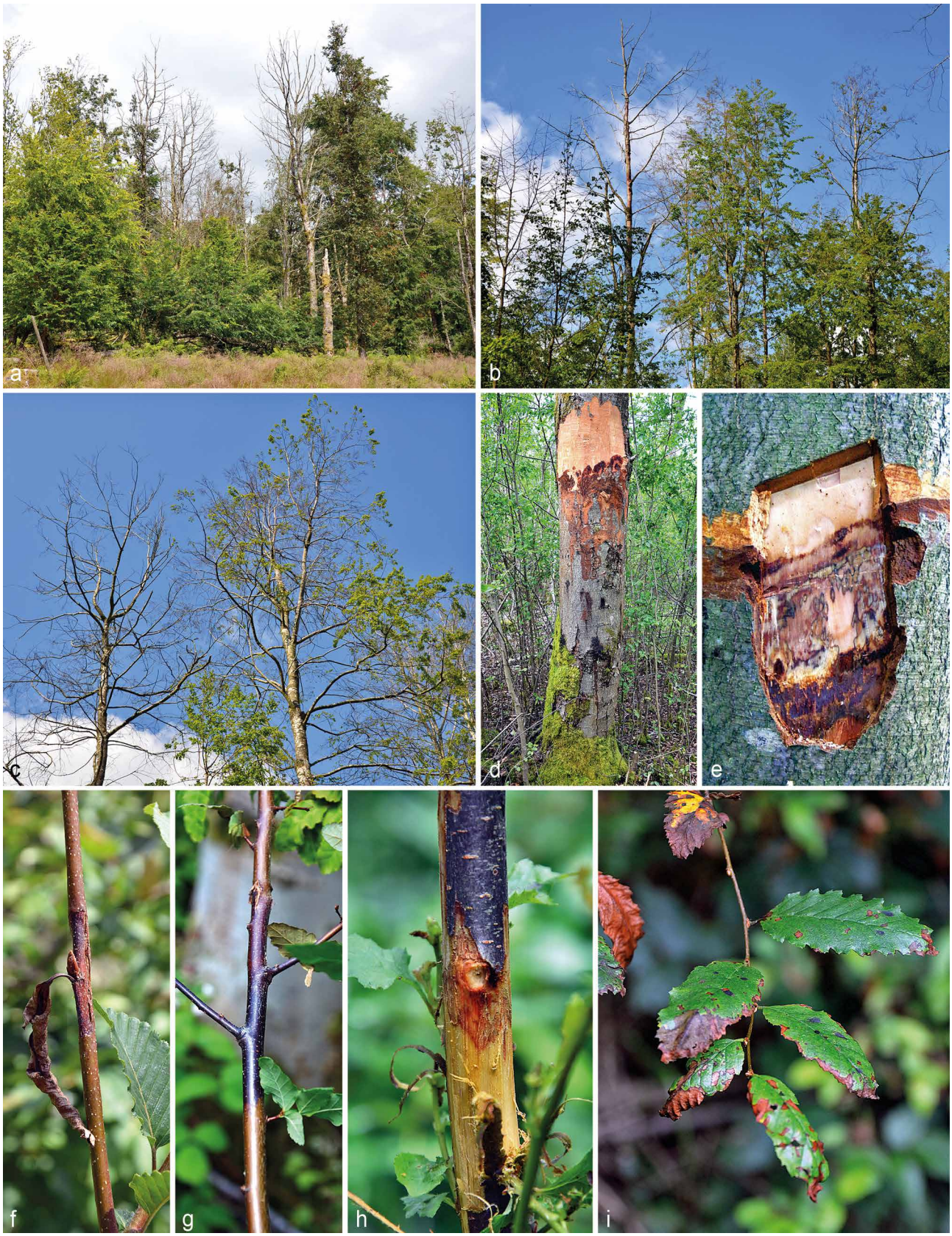

Fig. 10 Disease symptoms caused by *Phytophthora pseudosyringae* on *Nothofagus* spp. in the UK. a, b. Severe dieback and mortality of *N. obliqua* and *N. alpina* in mixed plantations in the UK; c. *N. obliqua* trees showing severe thinning, wilting and dieback of the crown and recent mortality; d. large, aerial bleeding canker with red-brown flame-shaped phloem lesion on the main stem of *N. obliqua*; e. deep xylem lesion on *N. obliqua*; f. sunken canker with cracking bark on a young *N. alpina* twig; g, h. *N. obliqua* twigs with dark necrotic bark lesions and orange-brown discoloration of the underlying necrotic phloem tissue (h); i. *N. obliqua* leaves with necrotic lesions resulting from aerial infections. — Photos: all B. Scanu.

bark with caducous sporangia produced on infected foliage and dispersed by wind and rain (Scanu & Webber 2016). At infected sites, aerial lesions are often observed on other host plants, including *F. sylvatica* and *Vaccinium myrtillus* (Beales et al. 2009, Denman et al. 2009b), most probably due to massive airborne inoculum coming from infected overstorey *Nothofagus* trees (Scanu & Webber 2016). *Phytophthora pseudosyringae* is characterised by having partial caducity of sporangia ranging from 10 to 90 % (Jung et al. 2003b). This enables the pathogen to be aerially dispersed and cause foliar and bark infections, as well as being a soil, water and root inhabitant. Previously in Europe, *P. pseudosyringae* was shown to be mainly involved in root and collar infections of alder, beech, chestnut and oak trees (Jung et al. 2003b, Cacciola et al. 2005, Denman et al. 2009b, Jung 2009, Scanu et al. 2010). However, in the humid forests of northern California and south-western Oregon, *P. pseudosyringae* has a similar lifestyle as *P. ramorum*, causing foliar infections on *Umbellularia californica* and aerial bark cankers on *Notholithocarpus densiflorus* and *Quercus agrifolia* (Wickland et al. 2008).

More recently, *P. pseudosyringae* has been detected associated with dieback and partial defoliation of *N. obliqua* in its natural range in central southern Chile (Fajardo et al. 2017). Main symptoms were similar to those reported from *Nothofagus* in Britain, with typical bleeding stem lesions and crown dieback, but twig and foliage infection were absent (Fajardo et al. 2017). Disease incidence seems to be lower in Chile compared to the UK (10 % vs up to 70 %). In inoculation trials under the bark of mature logs, and in zoospore inoculation trials of detached leaves, *N. obliqua* was highly susceptible to *P. pseudosyringae* isolates from the UK, while *N. alpina* was less susceptible (Scanu & Webber 2016). In both stem inoculation and soil infestation trials, isolates of *P. pseudosyringae* from Chile were highly aggressive to stems and roots of both *N. obliqua* and *N. alpina* (Fajardo et al. 2017). *Nothofagus dombeyi* was also shown to be susceptible to *P. pseudosyringae* in artificial inoculation experiments, although no natural infection has been reported so far (Fajardo et al. 2017).

The severity of *P. pseudosyringae* outbreaks on *Nothofagus* in the UK and its recent detection in native *Nothofagus* in South America, and the presence of *P. pseudosyringae* in the international nursery trade (Jung et al. 2016) highlight the risk of further spread of the pathogen into the Gondwanan *Nothofagus* forests in Chile, New Zealand and Tasmania. Previous records of *Phytophthora* spp. infecting *Nothofagus* spp. in their countries of origin include *P. cinnamomi*, associated with severe dieback and mortality of *Nothofagus* trees in the southern highlands of Papua New Guinea (Arentz 1983), and *P. citrophthora* and *P. nicotianae*, causing dieback of *N. macrocarpa* seedlings in a nursery in Chile (Valencia et al. 2011). Many natural forest ecosystems in the temperate regions of the southern Hemisphere, in particular in Chile, Argentina, New Guinea, Tasmania and New Zealand, are climatically favourable for inoculum production, spread and infections by airborne *Phytophthora* species (Scanu & Webber 2016), which may pose a serious risk to these native forests.

'Sudden Oak Death' and 'Sudden Larch Death' in the USA and the UK

'Sudden Oak Death' (SOD) is one of the most destructive epidemics of forest trees worldwide. This disease was first recorded in 1995 in Marine County, California, and subsequently spread across the relatively narrow coastal strip from Monterey county in central California to south-western Oregon (Goheen et al. 2002, Rizzo et al. 2002). In this area grow diverse temperate rainforests with an overstorey dominated by conifers like *Sequoia sempervirens*, *Abies grandis*, *Pseudotsuga menziesii* and *Tsuga heterophylla* and a mid- and understory layer of various broadleaved tree species including *Notholithocarpus densiflorus* (tanoak), *Quercus agrifolia* (coast live oak), *Umbellularia californica* (California bay laurel), *Acer macrophyllum*, *Alnus rubra* and *Arbutus menziesii* (Abrams & Ferris 1960, Knapp 1965, Lanner 1999).

The disease rapidly reached epidemic proportions in forests and in urban-forest interfaces in California, with a large number of *N. densiflorus*, *Q. agrifolia*, and to a lesser extent *Q. kellogii* and *Q. parvula* trees showing severe wilting and high mortality (Fig. 11a). First reports from forests in Oregon came in 2002 (Goheen et al. 2002). The causal organism was the heterothallic airborne pathogen *P. ramorum* which was first described from ornamental *Rhododendron* spp. and *Viburnum tinus* in Europe (Werres et al. 2001). The scale of devastation in California prompted national and international regulatory actions and local eradication efforts (Rizzo et al. 2005, Grünwald et al. 2012a, Hansen et al. 2012). Within a decade, extensive research had produced a rich body of knowledge on disease aetiology, infection biology, host range, pathways and population structure of *P. ramorum*. Over the last 20 years, the host list of *P. ramorum* has grown continuously exceeding to date 150 host species ranging from herbaceous plants and ferns to woody shrubs and trees (Grünwald et. al. 2012a). Extensive pathogenicity trials in Oregon demonstrated that 80 % of 49 native tree and shrub species tested were susceptible to *P. ramorum* (Hansen et al. 2005). Host lists, distribution of the pathogen in California and any new research findings are periodically updated on the web page www.suddenoakdeath.org.

While the host list is extensive, the susceptibility and role of different hosts in disease development varies considerably and relatively few plant species are actually killed by *P. ramorum*. One important distinction is that between the 'leaf or sporulation hosts' and 'canker hosts' which are dead-end hosts due to the inability of *P. ramorum* to produce sporangia on infected bark (Garbelotto et al. 2003, Davidson et al. 2005, Rizzo et al. 2005, Grünwald et al. 2008). Several plant species like *N. densiflorus* fall into both categories. In the humid coastal forests of northern California and south-western Oregon, *P. ramorum* demonstrates the typical multicyclic infection pattern of *Phytophthora* diseases. The pathogen is infecting with its caducous sporangia and zoospores the leaves and young shoots of a wide range of herbaceous and woody species, causing necrotic lesions and shoot blight (Fig. 11b, c). On infected necrotic and symptomless tissues prolific numbers of sporangia are produced and dispersed via rain and wind splash onto other leaves, branches and stems (Rizzo et al. 2002, 2005, Grünwald et al. 2008, Denman et al. 2009a). Infectious propagules are produced readily on petioles and leaves in the crowns and are spread aerially as demonstrated by the isolations of *P. ramorum* from canopy drip baited in rainwater traps (Davidson et al. 2005, Hansen et al. 2012). Efficient aerial spread and infections occur within 10 m distance of an infected host (Davidson et al. 2005, Hansen et al. 2008), although the sporadic appearance of isolated disease outbreaks suggests that long-distance dispersal of sporangia with strong blowing winds and fog may sometimes incite new disease foci in distances of more than 4 km from infected trees (Peterson et al. 2015).

Foliar infections, in contrast to bark cankers, do not usually result in plant death, thus plant species that have susceptible foliage only are not eliminated, enabling continuous inoculum production (Garbelotto et al. 2017, Lione et al. 2017). Due to its common occurrence in coastal forests and the high sporulation capacity of *P. ramorum* on its leaves, California bay laurel is the main driver of inoculum build-up and, hence, the epidemic in California, whereas infected tanoak leaves are the most important source of inoculum in south-western Oregon (Davidson et

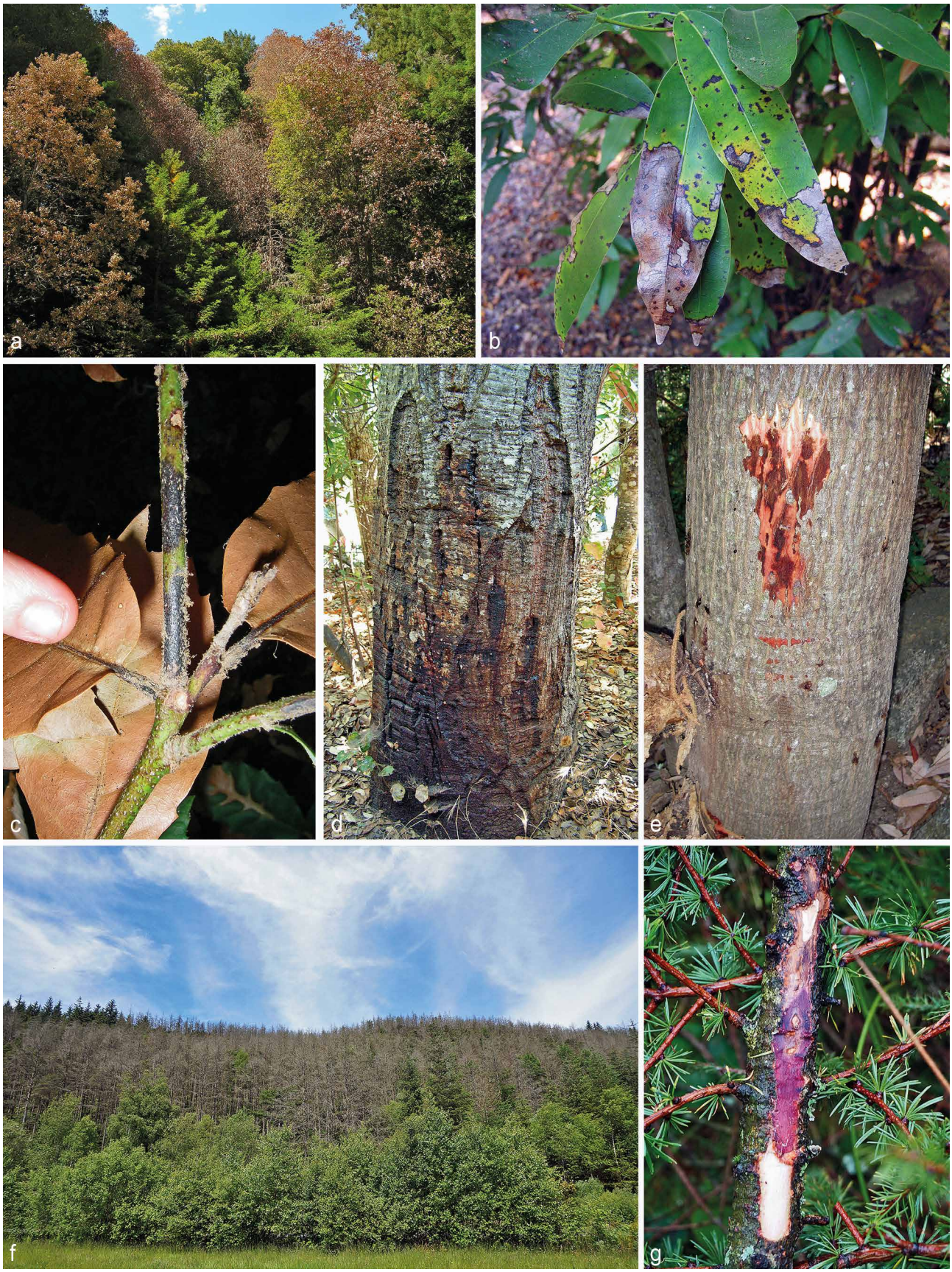

Fig. 11 Sudden Oak Death and Sudden Larch Death symptoms caused by *Phytophthora ramorum* in the USA (a–e) and in the UK (f, g), respectively. a. Severe wilting and mortality of tanoaks (*Notholithocarpus densiflorus*) in a mixed coastal forest with *Sequoia sempervirens* in California; b. necrotic lesions on leaves of California bay laurel (*Umbellularia californica*) in California; c. necrotic lesions on tanoak shoot in California; d. bleeding bark lesions on a tanoak stem in California; e. tanoak stem in California with multiple red-brown necrotic lesions of the inner bark; f. severe defoliation and mortality of a Japanese larch (*Larix kaempferi*) plantation in the UK; g. phloem lesion on a branch of a Japanese larch in the UK showing brown discoloration of older necrotic parts and a maroon-red advancing margin. — Photos: a, b, d: T. Jung; c, e: Y. Balci; f, g: A. Pérez-Sierra.

al. 2005, Rizzo et al. 2002, 2005, Hansen et al. 2008, Peterson et al. 2015). The existence of genetically identical *P. ramorum* populations in forests separated by distances exceeding 50 km, strongly indicates long-distance spread of the pathogen via infested nursery stock and infested substrate adhering to boots and tyres (Davidson et al 2005, Mascheretti et al. 2008, Filipe et al. 2012, Grünwald et al. 2012a). Long-term survival and long-distance spread in infested soil particles is achieved with thick-walled chlamydospores produced abundantly in infected plant tissues (Werres et al. 2001, Rizzo et al. 2002, Shishkoff 2007, Grünwald et al. 2008). *Phytophthora ramorum* can also be isolated from soil and streams, but propagules in such ecological niches are not contributing to disease development (Peterson et al. 2014). On stems and branches of *N. densiflorus*, *Q. agrifolia* and other oak species, *P. ramorum* sporangia cause infections leading to bleeding cankers with red-brown lesions of the underlying phloem and cambium (Fig. 11d, e). Due to multiple cankers girdling large branches and the stem, affected trees die within a short time, often in considerable numbers (Fig. 11a).

Using data on host susceptibility, host species distribution and reproduction, dispersal, and climate suitability of *P. ramorum*, Meentemeyer et al. (2004) developed a rule-based model of *P. ramorum* establishment and spread risk in California plant communities. This model demonstrated high accuracy when spread risk predictions were compared to data about presence, absence and severity of the disease from field surveys. In 2017, citizen science – based SOD Blitz surveys documented in California a three-fold increase in infection rates following the ending of a long period of drought in 2015 (Meentemeyer et al. 2015, Anonymous 2017). This increase in disease incidence during the wet years 2016 and 2017 and the recovery of susceptible tree species during the preceding long-lasting drought are correlated with the El Niño-Southern Oscillation (ENSO) cycle, an ocean-atmosphere phenomenon which originates in the tropical Pacific (Ropelewski & Halpert 1987, 1989). This is highlighting the importance of climatic parameters in the development of disease epidemics by airborne *Phytophthora* pathogens like *P. ramorum*.

Two other airborne *Phytophthora* species, *P. nemorosa* and *P. pseudosyringae*, are widely distributed in forests in California and Oregon. Both species cause leaf necrosis on foliar hosts like California bay laurel and cankers on oaks and tanoaks indistinguishable from the symptoms caused by *P. ramorum*. However, cankers associated with *P. nemorosa* and *P. pseudosyringae* are less common, have a scattered distribution and much lower mortality levels compared to *P. ramorum*, possibly suggesting host-pathogen coevolution (Wickland et al. 2008, Hansen et al. 2012, Kozanitas et al. 2017). Results from a population genetic AFLP analysis supported this hypothesis for *P. nemorosa* while *P. pseudosyringae* appears to be of European origin (Linzer et al. 2009).

Although *P. ramorum* was detected in Europe in the early 1990s, its impact has long been restricted to *Rhododendron* spp., *Viburnum* spp. and other ornamentals in nurseries and amenity plantings (Werres et al. 2001, Ivors et al. 2006, Vercauteren et al. 2010, Jung et al. 2016). Using a climate matching model (CLIMEX) revised according to the Californian risk ranking model of Meentemeyer et al. (2004) to Europe, a high risk of *P. ramorum* infections has been predicted for the Atlantic regions along the western parts of the British islands, Portugal, Spain and North-western France (Anonymous 2007). In accordance with this prediction, *P. ramorum* has since 2003 been causing aerial bleeding lesions on individual trees of *F. sylvatica*, *Aesculus hippocastanum* and *Q. rubra* growing in close proximity to heavily infected *R. ponticum* foliage in the UK (Brasier et al. 2004, Denman et al. 2006, Brown & Brasier 2007, Webber 2008). In August 2009, in south-western England, extensive mortality on mature and juvenile Japanese larch (*Larix kaempferi*) was detected for the first time and found being associated with *P. ramorum* but not with infected *Rhododendron* foliage (Brasier & Webber 2010, Webber et al. 2010). The disease rapidly reached epidemic proportions and was soon named 'Sudden Larch Death' (SLD) (Brasier & Webber 2010). Larch trees (*Larix* spp.) are fast growing deciduous conifers from the family *Pinaceae* which are distributed in all cold-temperate and boreal zones of the northern hemisphere. In the UK, Japanese larch, European larch (*L. decidua*) and their hybrid (*L.* × *leptolepis*) are all grown commercially for their durable timber with high mechanical strength and decay resistance. In 2010, the disease was also detected on Japanese larch in Scotland, Wales and Northern Ireland, and in 2011 *P. ramorum* was confirmed on European larch and on hybrid larch across the UK. This was the first record of *P. ramorum* causing lethal infection on a commercially important conifer species anywhere in the world. By the end of 2013, more than 3 million trees growing on over 10 000 hectares had been felled or were under notice to fell (Forest Research, unpubl. data). In Northern Ireland, it was recently demonstrated that the felling and removal of larch trees significantly reduces the inoculum of *P. ramorum* in the soil and successfully eradicates the pathogen from infested sites (O'Hanlon et al. 2017). SLD has a serious impact on the UK larch industry. Landowners suffer from significant losses through the destruction of immature crops, implementation of biosecurity measures and decreased timber values as the market is flooded with surplus supplies of felled larch (Harris 2014).

Initial symptoms of SLD on affected trees include purple or pale needle discolouration, wilting of short shoots, aborted buds, defoliation, dieback of branches or death of the entire crown (Fig. 11f). Crown symptoms are most pronounced in autumn. Resin bleeding can be observed on bark lesions of lateral shoots, branches and trunks of affected trees with brown discoloration and a deep pink to maroon-red margin of the underlying necrotic phloem (Fig. 11g) (Brasier & Webber 2010, Webber et al. 2010). On infected needles of all three larch species, *P. ramorum* produces very high numbers of 800 to almost 1 800 sporangia per cm^2 making larch the best of all sporulation hosts known to date (Harris & Webber 2016). This massive sporulation on the infected needles is driving multicyclic infections of needles and bark resulting in rapid and large-scale mortality of larch (Fig. 11f). In addition, sporangia from infected larch needles cause massive infection pressure on other tree species growing underneath or adjacent to infected larch trees (Brasier & Webber 2010, Harris & Webber 2016). Underneath infected larch trees, *P. ramorum* infections have been detected on broadleaved woody species such as *Betula pendula*, *C. sativa*, *F. sylvatica*, *Nothofagus* spp. and *R. ponticum*, and on other conifers such as *Abies procera*, *A. grandis*, *Picea sitchensis*, *P. menziesii* and *T. heterophylla* (Brasier & Webber 2010, Webber et al. 2010, Harris & Webber 2016). Climate and weather play a key role in the distribution and intensity of the disease on larch.

Initially, apart from the UK, the Republic of Ireland had been the only other country where larch trees were suffering from *P. ramorum* infections. However, in 2017, *P. ramorum* was for the first time detected affecting Japanese larch in mainland Europe in Brittany (France) (http://ephytia.inra.fr/fr/C/24935/Forets-Phytophthora-ramorum).

Phytophthora ramorum resides, together with *P. lateralis*, *P. foliorum* and *P. hibernalis*, within phylogenetic Clade 8c (Werres et al. 2001, Grünwald et al. 2008, Yang et al. 2017). There are four known clonal lineages named after the continent of their first appearance as EU1 and EU2 from Europe and NA1 and NA2 from North America (Ivors et al. 2006, Grünwald et al. 2008,

Van Poucke et al. 2012). All European isolates of the EU1 and EU2 lineages belong to the A1 mating type while the North American NA1 and NA2 lineages contain exclusively A2 isolates (Brasier & Kirk 2004, Werres & Kaminski 2005, Vercauteren et al. 2011). In interlineage pairing tests of A1 and A2 isolates the mating frequency is extremely low and the resulting oospores have aberrant genome sizes and unusually high abortion rates, suggesting the presence of reproductive barriers (Brasier & Kirk 2004, Boutet et al. 2010, Vercauteren et al. 2011, Franceschini et al. 2014). This is supported by the results of a coalescence analysis indicating that the EU1, NA1 and NA2 lineages are separated since 165 000–500 000 yr (Goss et al. 2009). The apparent lack of sexual reproduction explains the clonal structure of the *P. ramorum* populations in Europe and the USA. In North America, three of the four known clonal lineages of *P. ramorum* are currently recognised: NA1, NA2 and EU1. The NA1 lineage most likely arrived in California in the 1990s via infected nursery stock imported from an unknown exotic origin, and has since spread throughout California and southwest Oregon as primary driver of the SOD epidemic (Grünwald et al. 2012a). Interestingly, the Oregon outbreak could genetically not be linked to the Californian NA1 population and, apparently, originates from a separate introduction of infested nursery plants (Prospero et al. 2007, Mascheretti et al. 2008, Grünwald et al. 2012a). The NA2 lineage was most likely introduced to nurseries in the PNW and occurs only infrequently in Californian forests (Ivors et al. 2006, Goss et al. 2011, Grünwald et al. 2012a). The EU1 lineage was introduced from Europe to the PNW where it is thriving in ornamental nurseries and eventually spread to California (Goss et al. 2011, Grünwald et al. 2012a). In the UK, the majority of the isolates from larch belong to the widespread EU1 lineage, while the EU2 lineage occurs currently only in Northern Ireland and in a small area in south-western Scotland (Van Poucke et al. 2012). The EU2 lineage shows faster growth and tolerates higher temperatures than the EU1 lineage (Franceschini et al. 2014). In addition, the EU2 lineage is also significantly more aggressive to larch bark tissue than the EU1 lineage (Harris et al. 2015) and, therefore, is likely to kill affected trees more rapidly (King et al. 2015). In 2016, *P. ramorum* was detected in several streams running through diverse mountain forests in northern Vietnam. Extensive mating tests demonstrated that the Vietnamese population contained both mating types, which together with the absence of apparent leaf symptoms and bleeding stem lesions in these forests suggest potential endemism of *P. ramorum* to Southeast Asia (T. Jung, M. Horta Jung, C.M. Brasier unpubl. data).

Fig. 12 Disease symptoms caused by *Phytophthora ilicis* on *Ilex aquifolium* in mountain forests of the Mediterranean islands Corsica and Sardinia. a. Mature trees showing complete defoliation and severe dieback; b. bleeding stem canker; c. fresh twig lesion around an axillary node and dead infected leaves; d. inactive twig canker with cracking surface due to the production of brown suberised tissue during the dry season; e. necrotic twig lesion originating from the progression of *P. ilicis* from the infected leaf through the petiole into the twig; f. leaf necrosis and progression of *P. ilicis* through the petiole into the twig; g. black necrotic leaf spots indicating multiple fresh infections. — Photos: all B. Scanu.

Leaf and twig blight of Ilex aquifolium in Europe and North America

Native to Atlantic regions of Europe and to southern Europe and western Asia, English holly (*Ilex aquifolium*) is an important component of the understorey vegetation in temperate *Fagaceae* forests and in cool and humid mountain ecosystems in Mediterranean regions (Pignatti 1982). This species is also widely planted as an ornamental plant and in hedgerows across Europe, and it is grown extensively in the PNW, USA for the production of holly cuttings and young trees.

In 1954, a previously unknown *Phytophthora* species was consistently isolated from black leaf spots, twig blight, berry infections and limb and trunk cankers of English holly orchards in Oregon and later also Washington, which were previously attributed to ascomycete fungi like *Diaporthe crustosa*, *Vialaea insculpta* and *Fusarium* spp. (Buddenhagen & Young 1957). This new species was described as *P. ilicis*, and a detailed description of the disease aetiology was given by Buddenhagen & Young (1957). In severe cases, the whole crown became completely defoliated with leaf shedding starting from the lower branches. No root or collar infection of *P. ilicis* were reported and the pathogen has never been detected from river water (Hansen et al. 2017).

Thirty years later, *P. ilicis* was recovered from *Ilex* spp. in parks and gardens of the UK, where it caused infections along hedges, and also from symptomatic nursery stock (Strouts et al. 1989). The pathogen was considered introduced to the country, probably during the 20th century (Tubby & Webber 2010). More recently, the pathogen has been reported causing twig blight on ornamental *I. aquifolium* in Galicia, Spain (Pintos et al. 2012) and in Germany from nursery plants (https://gd.eppo.int/reporting/article-5866).

Based on these reports, the geographic distribution of *P. ilicis* seemed to be restricted to cool-temperate regions (Buddenhagen & Young 1957, Strouts et al. 1989, Pintos et al. 2012). However, a recent study demonstrated the widespread occurrence of *P. ilicis* on *I. aquifolium* in natural forests of the Tyrrhenian islands Corsica and Sardinia (Scanu et al. 2014b). The symptomatology matches previous descriptions in the USA and Europe, including severe defoliation of the whole crown (Fig. 12a) and bleeding cankers on the main stem and branches (Fig. 12b). Necrotic twig lesions are often observed around the axillary node (Fig. 12c, e) and where the petiole is inserted (Fig. 12f), most likely due to the accumulation of zoospores produced by sporangia that emerge through stomata on the leaf surface and by growth of the pathogen through the petiole (Scanu et al. 2014b). Black leaf spots occur in the early decline stages (Fig. 12g), starting from branches close to the ground. Necrotic twig lesions develop as cankers with brownish orange, suberized and cracking epidermal tissues during the dry season (Fig. 12d, e). In these tissues, *P. ilicis* forms oospores, which allow the pathogen to survive dry summer conditions and germinate in the following wet season. Having caducous sporangia and a low optimum temperature for growth of 20 °C, *P. ilicis* infections occur mainly during the cool rainy period, from October to May, while the disease is completely inactive during summer (Buddenhagen & Young 1957, Scanu et al. 2014b).

Since all previous records of the species worldwide came from horticultural, parks, gardens and nurseries, the widespread distribution of *P. ilicis* in natural ecosystems of Corsica and Sardinia strongly indicates endemism of this pathogen in the Mediterranean basin (Scanu et al. 2014b, Hansen et al. 2017). This hypothesis is supported by the fact that two close relatives of *P. ilicis*, *P. pseudosyringae* and *P. psychrophila*, are most likely also native in Europe (Jung et al. 2002, 2003b, 2016, Linzer et al. 2009, Pérez-Sierra et al. 2013, Hansen et al. 2017).

Needle cast and defoliation of Pinus radiata in Chile

Pinus radiata (Monterey pine) has a scattered limited natural distribution along the Pacific coast of central California and Baja Californica in Mexico (Rogers 2004, Rogers et al. 2006). On a global scale, it is one of the most common plantation trees in Mediterranean regions, in particular in Australia, Chile, New Zealand, South Africa and Spain (Rogers et al. 2006, Richardson et al. 2007). In 2004, a needle cast and defoliation disease, named 'Daño foliar del pino', was reported for the first time from 70 ha coastal *P. radiata* plantations in the Arauco province of central Chile (Durán et al. 2008). By 2006, the affected area had increased to almost 60 000 ha, with varying levels of damage. In 2007 the disease area decreased to less than 2 000 ha and has remained at that level (Durán et al. 2008, 2010, Ahumada et al. 2013). Currently, the disease occurs between Constitución and Valdivia, exclusively in areas with high humidity during most of the year due to their proximity to the Pacific coast (Ahumada et al. 2013). 'Daño foliar del pino', one of the most important foliar diseases affecting Monterrey pine, is caused by the previously unknown *P. pinifolia* which was the first *Phytophthora* species reported to cause needle infections on *Pinus* spp. (Durán et al. 2008). The pathogen has only been found in Chile where it exclusively affects *P. radiata* (Durán et al. 2008, 2010, Ahumada et al. 2012, 2013). The considerable variation of disease incidences and affected areas over the years is related to the El Niño cycle and has been explained using favourable days calculated with the Hyre model. With 141 d, the peak of favourable conditions was recorded in 2006, which correlated with the highest disease incidence recorded until now, while each of the following years had less than 60 favourable days coinciding with low incidence of the disease (Ahumada et al. 2013). Due to the lack of preformed sporangial pedicels, *P. pinifolia* is not a true airborne species. However, as in *P. constricta* in Western Australia, sporangia break off with relative ease enabling an aerial lifestyle (Durán et al. 2008, Rea et al. 2011). *Phytophthora pinifolia* survives non-favourable dry conditions in infected needles on the ground. After onset of humid conditions in spring or early winter, the pathogen infects needles on lower branches via sporangia formed on infected needles on the ground and spreading via rain and wind splash onto healthy needles. If humid conditions persist, *P. pinifolia* produces new sporangia on infected needles causing multicyclic infections and moving gradually up in the canopy (Fig. 13a–f) (Durán et al. 2008). In seedlings and trees younger than three years old, total defoliation occurs frequently resulting in plant death. In addition, the pathogen can progress in the shoots and the stem causing girdling lesions, leading to quick death of the trees. In trees older than three years the infection progress and the symptoms are similar but bark lesions are not girdling the stem and mortality is rare (Ahumada et al. 2012). On trees older than six years, the disease can affect all needles except those less than one year old (Fig. 13c–f). Needle infection is usually characterized by the presence of a black band (translucent areas) and a pale-green to greyish discolouration (Fig. 13g) turning brown at the end of spring (Ahumada et al. 2013). Long-dead needles have a pale greyish colour (Fig. 13e). The symptoms are generally best observed during the rainy season, from May to November, but heavy rain at the end of summer can induce an early appearance of symptoms.

Since 2008, another needle disease, named Red Needle Cast, with similar symptoms and disease aetiology has been reported in *P. radiata* plantations in New Zealand. However, in this case the airborne *Phytophthora* species *P. pluvialis* from Clade 3 was shown to be the causal agent (Dick et al. 2014).

Phytophthora pinifolia belongs to Clade 6, which contains mostly aquatic species and opportunistic soil- and waterborne pathogens of woody plants (Jung et al. 2011, Burgess et al. 2018).

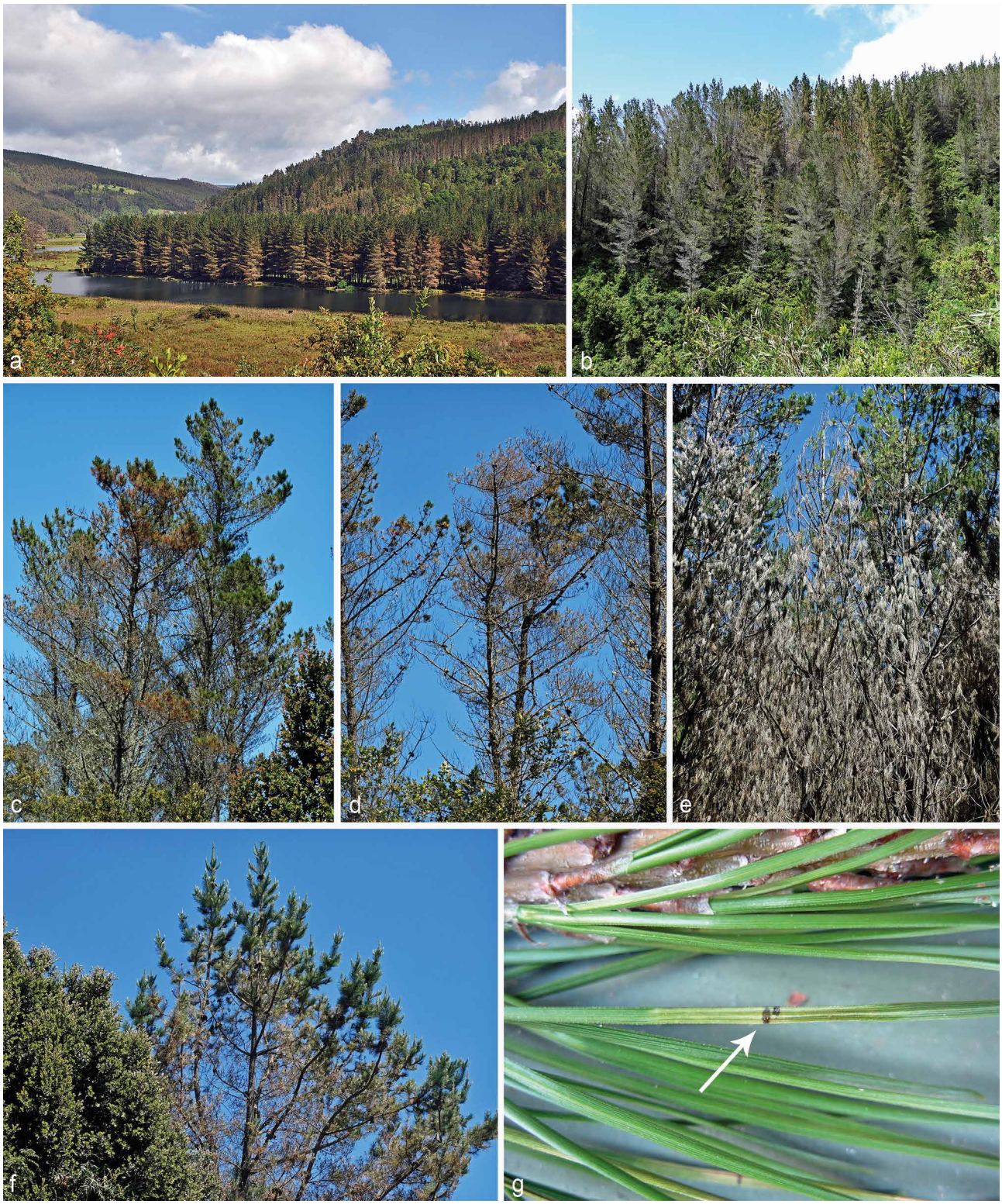

Fig. 13 Needle cast and defoliation caused by *Phytophthora pinifolia* on *Pinus radiata* in the Valdivian region of Chile. a. Mature plantation with extensive red-brown discolouration of infected foliage; b. mature plantation with largely defoliated crowns due to massive needle infections by *P. pinifolia*; c. extensive defoliation of mature crowns resulting from the progressive browning and shedding of infected needles which started at the lowest branches and spared only the youngest needles; d. recently killed tree with severe defoliation; e. dying young tree in a dense stand with greyish discolouration of infected needles; f. upper crown typically showing transparency due to shedding of older infected needles, pale-green discolouration of freshly infected needles and uninfected, dark-green young needles; g. needle showing typical black bands (translucent areas; arrow) within the pale-green to greyish area of infected tissue. — Photos: a–f: T. Jung; g: A. Durán.

Currently, the only known foliage-infecting member of Clade 6 is *P. pinifolia* (Durán et al. 2008, Burgess et al. 2018). An amplified fragment length polymorphism (AFLP) analysis demonstrated a clonal population structure of *P. pinifolia* in Chile indicating a single clonal introduction of the pathogen (Durán et al. 2010). A character shared between *P. pinifolia* and many other Clade 6 species is the sterile breeding system (Durán et al. 2008) which is responsible for maintaining the clonal population structure of *P. pinifolia* in Chile and reduces the ability of the pathogen to adapt to changes in *P. radiata* resistance.

An integrated management approach for the needle disease caused by *P. pinifolia* includes the selection of tolerant *P. radiata* clones, species replacement in high-risk areas and fungicide application (Ahumada et al. 2013). Using a disease model, areas with high, medium and low risk of disease development have been identified. In high-risk areas, *P. radiata* plantations are being replaced by alternative species, in particular *Eucalyptus* spp., while in medium-risk areas tolerant *P. radiata* clones are planted. Alternatively, fungicides like mefenoxam and metalaxyl (phenylamides) and the fungi-static potassium phophite have proven suitable for disease control, reducing symptoms by up to 90 % and plant mortality to less than 5 % (Ahumada et al. 2013). Fungicides are mainly applied in one- or two-years old plantations to reduce the heavy infection pressure.

Despite the significantly reduced disease incidence since the peak in 2006 and the development of successful management strategies (Ahumada et al. 2013), *P. pinifolia* continues to be an important threat to the *P. radiata* industry worldwide. In Chile, the introduction of new genotypes or the appearance of mutations in the existing clonal population (Durán et al. 2010) could produce new pathotypes able to overcome the current genetic resistance or pesticide effectiveness, as has been reported for other *Phytophthora* spp. (Gisi & Cohen 1996, Goodwin et al. 1996). Since the origin of *P. pinifolia* is still unknown, the pathogen also poses a serious threat to natural populations of *P. radiata* and other pine species in their native ranges (Widmer & Dodge 2015).

CONCLUDING REMARKS

Concern about *Phytophthora* pathogens in natural and forest ecosystems began in the 1960s in Australia, where the invasive *P. cinnamomi* has been spreading for more than one century threatening some of the world's richest plant communities in Western Australia and Victoria (Podger et al. 1965, Podger & Newhook 1971, Shearer & Tippett 1989, Marks & Smith 1991, Shearer et al. 2004, Hardham 2005). During the past 60 years, the detection of previously unknown *Phytophthora* diseases in natural and semi-natural ecosystems has increased exponentially (Fig. 1). Since the 1990s, the finding of *P. cinnamomi* and many other soilborne *Phytophthora* spp. involved in the oak declines in southern and central Europe (Brasier et al. 1993, Jung et al. 1996, 2000), the discovery of the airborne *P. ramorum* causing 'Sudden Oak Death' in the western USA (Rizzo et al. 2002), the resurgence of chestnut ink disease and the widespread alder mortality across Europe due to the *P.* ×*alni* hybrid complex (Vettraino et al. 2001, Brasier et al. 2004, Jung & Blaschke 2004, Husson et al. 2015), have raised the interest by many plant pathologists for this important diseases causing genus. Consequently, in 1999 the International Union of Forestry Research Organizations (IUFRO) Working Party 7.02.09 'Phytophthora Diseases of Forest Trees' (https://www.iufro.org/science/divisions/division-7/70000/70200/70209/) was established which has subsequently become the main platform for researchers studying all aspects of *Phytophthora* pathogens and the diseases they are causing in forests and other natural woody ecosystems. This review provides the first comprehensive overview of the history, distribution, aetiology, symptomatology, dynamics and impact for the main diseases caused by *Phytophthora* species on woody host plants in natural ecosystems on a global scale, hence giving a baseline from which to identify and compare similar disorders in the future.

Most of the diseases presented in this review are caused by exotic invasive *Phytophthora* pathogens with a clear link between the 'plants-for-planting' pathway and subsequent impacts in natural ecosystems (Jules et al. 2002, Jung & Blaschke 2004, Brasier 2008, Chadfield & Pautasso 2012, Jung et al. 2016). There is an accumulating body of indirect and partly also direct evidences that *P. cinnamomi*, *P. lateralis*, *P. plurivora* and *P. ramorum* originate from Southeast and eastern Asia (Shearer & Tippett 1989, Brasier et al. 1993, 2010, 2012, Chang et al. 1996, Hansen et al. 2000, 2012, Jung et al. 2000, 2016, 2017a, b, c, Rizzo et al. 2002, Shearer et al. 2004, Goss et al. 2009, Hardham 2005, Jung 2009, Jung & Burgess 2009, Brasier & Webber 2010, Webber et al. 2010, 2012, Franceschini et al. 2014, Arentz 2017). Also for *P. agathidicida* in New Zealand *P. austrocedri* in Argentina and the UK, *P. acerina* and *P. cactorum* in Europe, *P. elongata* in Australia, *P. kernoviae* in the UK, *P. multivora* in Australia and Europe, *P. pinifolia* in Chile, *P.* ×*cambivora* in Europe and North America, and for the parents of *P.* ×*alni*, i.e., *P.* ×*multiformis* and *P. uniformis*, the high aggressiveness to native woody species, low genetic variability of pathogen populations and co-existence with healthy native vegetation in other continents, respectively, indicate exotic origin (Crandall et al. 1945, Jung et al. 2000, 2002, 2003b, 2016, 2017b, c, Brasier & Kirk 2001, Vettraino et al. 2001, 2005, Jung & Blaschke 2004, 2006, Brasier et al. 2005, Greslebin et al. 2007, 2010, Saavedra et al. 2007, Beever et al. 2009, Jung 2009, Scott et al. 2009, Durán et al. 2010, Rea et al. 2010, Green et al. 2013, Vélez et al. 2013, Ginetti et al. 2014, Henricot et al. 2014, Scott & Williams 2014, Weir et al. 2015). Recently, several studies identified the international plant trade as the main pathway for the introduction of invasive forest diseases into North America and Europe (Liebhold et al. 2012, Santini et al. 2013, Chapman et al. 2017). Accordingly, Jung et al. (2016) demonstrated almost ubiquitous infestations of nurseries and young plantings across Europe with a wide range of *Phytophthora* species. This study and similar results from Australia and the USA (Hardy & Sivasithamparam 1988, MacDonald et al. 1994, Davison et al. 2006, Schwingle et al. 2007, Yakabe et al. 2009, Bienapfl & Balci 2014, Parke et al. 2014, Yang et al. 2014, Simamora et al. 2015) leave no doubt that plant production facilities are the major source of *Phytophthora* spread into the wider environment. Due to the application of fungicides or fungistatic chemicals, nursery plants infected by *Phytophthora* spp. often appear visually symptomless and, hence, pass unnoticed through the phytosanitary controls acting as 'inoculum reservoirs' in the nurseries and resulting in accidental *Phytophthora* spread (Pérez-Sierra & Jung 2013, Bienapfl & Balci 2014, Migliorini et al. 2015, Jung et al. 2016). Another danger, arising from the intensified international nursery trade, is the accidental encounter of closely related allopatric *Phytophthora* species, which due to geographic separation have not build up reproductive barriers and readily hybridise. Such interspecific hybrids may differ in host range and virulence from the parental species as demonstrated by *P.* ×*alni* (Brasier & Kirk 2001, Brasier et al. 2004, Husson et al. 2015), thus making predictions about the potential effects of an ongoing invasion even more difficult.

Once introduced to a new suitable environment, a *Phytophthora* pathogen will inevitably spread, actively via root-to-root infection and with motile zoospores in soil and surface water and passively through the movement of infested soil or infested water. Therefore, the control and management of *Phytophthora*

pathogens and diseases are mainly focused on the prevention of their introduction, and on slowing down their spread once they are introduced. Prevention of primary *Phytophthora* introductions can be achieved by testing nursery stock using classical isolation methods, sensitive high-throughput molecular detection methods and temporary outplanting in quarantine-facilities. When managing natural ecosystems, the only cost-effective and ethically acceptable approach is the use of plants from certified pathogen-free production facilities (Parke & Grünwald 2012). Preventative system approaches for the production of *Phytophthora*-free nursery stock in nurseries have been repeatedly suggested by the scientific community to the regulators (Brasier 2008, Parke & Grünwald 2012, Jung et al. 2016). Parke & Grünwald (2012) highlighted the importance to define the hazards for *Phytophthora* contamination within a nursery and employ best management practices to reduce the risk of infestation for all pathogens and pests. This systems approach demonstrated to improve the control of pathogens and pests in nursery production and prevent the movement of exotic pathogens or pests in the nursery trade. Training nursery growers to identify early symptoms minimizes risks. Thermosterilisation of potting media and the filtering or disinfestation of irrigation water also helps to reduce the risk of *Phytophthora* introductions (Stewart-Wade 2011, Pérez-Sierra & Jung 2013).

The current approach to prevent the movement of pests and pathogens via the nursery trade is mainly based on international plant biosecurity protocols, including certification, endpoint inspections for list-based pests and pathogens, and quarantine measures. However, these methods have largely failed to prevent the arrival of invasive and exotic pathogens (Brasier 2008, Liebhold et al. 2012, Santini et al. 2013, Jung et al. 2016, Eschen et al. 2017). For plants moving through the nursery trade, a phytosanitary certificate, indicating the production facility is free of regulated *Phytophthora* spp., is mandatory. In the absence of a certificate, a visual inspection of plants has to be performed (Brasier 2008). However, visual inspections of symptoms are costly and often ineffective, especially since they are based on lists of known harmful species despite the fact that the majority of introduced aggressive *Phytophthora* species were unknown to science before they caused serious damages in their new environments. In addition, the regular use of fungicides and fungistatic chemicals in nurseries is decreasing disease incidences without eliminating the *Phytophthora* pathogens. This practice is masking the presence of the pathogens, which are surviving with enduring resting structures, resulting in plantings of visually healthy nursery stock that develop disease symptoms in subsequent years (Jung & Blaschke 2004, Brasier 2008, Pérez-Sierra & Jung 2013, Migliorini et al. 2015, Jung et al. 2016). In a critical review, Brasier (2008) underlined the need for a scientific revision of the international plant biosecurity protocols. The scientific community recommended replacing the list-based species-by-species regulation approach by a more efficient pathway regulation approach based on pathway risk analyses and combined with a strict quarantine system (Keller et al. 2007, Jung et al. 2016).

In natural ecosystems, there are a number of possible strategies to mitigate the impact of *Phytophthora* species. Phosphite (phosphoric acid) applications are the most common and most successful method for controling dieback of trees and forests resulting from extensive fine root losses caused by *Phytophthora* infections and slowing down disease spread (Shearer & Tippett 1989, Fernandez-Escobar et al. 1999, Pilbeam et al. 2000, Hardy et al. 2001, Tynan et al. 2001, Smith 2003, Shearer & Fairman 2007, Barrett & Rathbone 2018). Several early studies demonstrated that treatments with phosphite indirectly control *Phytophthora* diseases by acting on host physiology and on host-pathogen interactions (Fenn & Coffey 1984, Guest & Grant 1991), but the exact mode of action had long been a mystery. Recent studies have elucidated that phosphite induces systemic activity against *Phytophthora* spp. in plants mainly by priming plants for a rapid and intense response to infection via up-regulation of several defence-related genes in the jasmonate, salicylic acid, ethylene and auxin signalling pathways (Eshragi et al. 2011, 2014a, Dalio et al. 2014). However, it was suggested that phosphite-induced resistance to *P. cinnamomi* in susceptible *Arabidopsis thaliana* ecotypes and natural resistance of tolerant ecotypes may be triggered through different signaling pathways (Eshragi et al. 2014b). Generally, phosphite appears to be more effective for soilborne than for airborne *Phytophthora* diseases, although phosphite applications can significantly reduce canker size in tanoaks infected by *P. ramorum* (Garbelotto et al. 2009). The efficacy of the treatments is higher when plants are physiologically active (Pilbeam et al. 2000). Phosphite can be applied by injecting a water dilution directly into the trees (Fernandez-Escobar et al. 1999) or through foliar applications, with or without surfactants, although not all evergreen plants with waxy leaves absorb phosphite (Shearer & Fairman 2007). Application rates vary depending on the application mode, the host plants and the region (Hardy et al. 2001, Smith 2003, Shearer et al. 2006, Garbelotto et al. 2009). Overdosing commonly leads to phytotoxicity (Thao & Yamakawa 2009). Although phosphite applications can slow down disease spread and protect individual plants (Hardy et al. 2001), they cannot completely halt *Phytophthora* spread and disease progression in native vegetation (Shearer et al. 2004). Silvicultural and site-specific management measures, like soil amendments and reducing host plant density, have a proven effect in reducing disease incidence and/or slowing down further disease spread (Erwin & Ribeiro 1996, Pérez-Ramos et al. 2008). Thinning should be more intensive in those areas predicted to be more easily infested, such as areas downhill or downstream from outbreaks. If multiple plant species are present at an infested site, selective thinning of the best sporulation host will have the most significant impact on disease spread (Serrano et al. 2010, Fichtner et al. 2011). Containment and eradication of soilborne *Phytophthora* species from spot infestations in natural ecosystems can be achieved by a combination of robust treatments including host plant destruction with herbicides, application of selective fungicides, fumigation with strong biocides like metham-sodium, and mechanical root barriers (Dunstan et al. 2009). However, due to the lack of legal authorisations and for environmental reasons, such treatments are not realistic options for the control of *Phytophthora* diseases in natural areas of most countries.

On the long-term, increasing the genetic resistance of susceptible tree species against *Phytophthora* spp. seems to be the most promising sustainable management approach for stabilising declining natural ecosystems and reintroducing susceptible tree species at sites with high disease impact. The successful long-term resistance screening programme of *C. lawsoniana* concerning *P. lateralis*, can serve as a role model for other *Phytophthora* diseases (Hansen et al. 2000, 2011, Oh et al. 2006, Sniezko et al. 2006, 2011). Also for the pathosystems *Castanea* / *P. cinnamomi* and *P.* ×*cambivora*, *A. glutinosa* / *P.* ×*alni*, and *E. marginata* / *P. cinnamomi* the natural occurrence of genetic resistance has been demonstrated (Jung & Blaschke 2006, Robin et al. 2006, Miranda-Fontaíña et al. 2007, Costa et al. 2011, Santos et al. 2015, 2017a, b, Shearer et al. 2014, Chandelier et al. 2016).

Recently, the potential origin of several invasive *Phytophthora* species, including *P. cinnamomi*, *P. lateralis*, *P. plurivora* and *P. ramorum*, in Southeast Asia has been unravelled (Brasier et al. 2010, 2012, Huai et al. 2013, Jung et al. 2017a, c, T. Jung, C.M. Brasier, M. Horta Jung unpubl. data). Comparative pheno-

typic and molecular studies of the invasive and native populations of these pathogens will advance our understanding of their adaptation and evolution after introduction to new environments. In addition, future studies of the behaviour and ecological role of these pathogens and their interaction with the native vegetation in their centres of origin will help to elucidate the factors that shaped their outstanding invasiveness. This knowledge will help improving management concepts for the diseases these pathogens are causing. Moreover, tolerant or resistant species from tree genera affected by these pathogens in other continents may be found in the centres of origin which could be used in resistance screening programmes. Finally, natural antagonists which may be used as biological control agents in invasive situations, may be detected in the future.

Whether and how the projected future changes in temperature and precipitation patterns will affect the spread and activity of *Phytophthora* pathogens on the global and the regional scales remains unknown. However, using the CLIMEX model a significant increase in the activity of *P. cinnamomi* and the area suitable for this pathogen has been proposed (Brasier & Scott 1994, Brasier 1996, Burgess et al. 2017). Due to the known interaction between *Phytophthora*-caused fine root losses and droughts, and the multicyclic spread of *Phytophthora* zoospores and sporangia during persisting humid conditions, rising temperatures, in particular during winter, and increased summer droughts alternating with periods of unseasonal heavy rain, predicted by several models and extrapolations of climatic trends of the 20th century (Schönwiese et al. 1994, Rapp & Schönwiese 1995, Houghton et al. 1996, Watson et al. 1996, 1998, Pachauri & Reisinger 2007, Battles et al. 2008, Giannakopoulos et al. 2009, Ozturk et al. 2015), will most likely intensify root and collar rot incidences. This will further destabilise *Phytophthora*-infested natural ecosystems. Therefore, effective plant biosecurity protocols to prevent further introductions and spread of *Phytophthora* pathogens, management concepts and control measures to mitigate the impact of invasive *Phytophthora* diseases in natural ecosystems, and resistance screening programmes are urgently required.

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