An overview of ash (*Fraxinus* spp.) and the ash dieback disease in Europe

Rasmus Enderle[1]*, Jan Stenlid[2] and Rimvydas Vasaitis[2]

Address: [1] Institute for Plant Protection in Horticulture and Forests, Julius Kuehn Institute, 38104 Braunschweig, Germany.
[2] Department of Forest Mycology and Plant Pathology, Swedish University of Agricultural Sciences, 75007 Uppsala, Sweden.

*Correspondence: Rasmus Enderle. Email: rasmus.enderle@julius-kuehn.de

The electronic version of this article is the definitive one. It is located here: http://www.cabi.org/cabreviews

Abstract

Ash dieback, caused by the invasive fungal pathogen *Hymenoscyphus fraxineus*, has become a serious threat to ash trees (*Fraxinus* spp.) and ash-related ecosystems in Europe. The fast emergence and expansion of this new disease has called for an intensive investigation, which resulted in a large number of research projects in Europe. Recently, as a result of the European cooperation and exchange programme FRAXBACK, numerous reports containing detailed information about the situation of ash dieback in different countries and related research have been published. For this review, we performed a systematic analysis of these country reports. We focussed on differences and similarities between European regions and countries regarding the importance, genetics and resistance of ash, the spread, monitoring, impact and management of the disease and on factors that have an influence on its severity. By including most recent scientific literature, this review provides a concise yet substantial overview about ash and ash dieback in Europe.

Introduction

Ash dieback is a lethal disease of ash trees (*Fraxinus* spp.) in Europe. It is caused by an invasive ascomycete, *Hymenoscyphus fraxineus* Baral et al. (syn. *H. pseudoalbidus* Queloz et al., anamorph: *Chalara fraxinea* T. Kowalski), which is native to Far East Asia [3–5]. First symptoms of ash dieback have been observed in the early 1990s in north-eastern Poland [6, 7 and references therein, 8], but a genetic study suggests that the introduction of the pathogen to Europe had occurred one to three decades earlier [3]. The disease spreads fast and is today present in most part of the ash distribution range in Europe, causing a dramatic decline of native trees that belong to the genus *Fraxinus*. Leaves and petioles are infected by airborne ascospores that can be transported over long distances [5, 7]. The fungus can spread to woody tissues through the petiole–shoot junction [9], where it causes bark necroses and locally kills shoots and twigs. The successive crown decline caused by every year occurring infections often is lethal to the trees. Another important yet little understood factor of mortality is the symptom of root collar necrosis, which is most likely caused primarily by *H. fraxineus* and often is followed by root and but rot. In this regard, wood-decay fungi, particularly fungi of the genus *Armillaria*, play a presumably secondary yet decisively aggravating role [10–15].

The European dimension of the ash dieback epidemic did soon demand for an international approach to address challenging research questions. Within the European Cooperation in Science and Technology (COST) programme, an action called FP1103 FRAXBACK was conducted from 2012 to 2016, providing a platform for more than 200 scientists from 38 countries and different disciplines to exchange and interact. As a main outcome of this action, a comprehensive book (299 pages) [1] containing detailed country reports about the situation and research regarding ash dieback was published in 2017,

along with a special issue of the scientific journal Baltic Forestry [2]. This review article can be understood as a short summary of the book with a focus on differences and similarities between European countries and regions. It deals with the importance, genetics and resistance of ash, the spread, monitoring, impact and management of the disease and with factors that have an influence on its severity. While the book is the main source of information for this review, it is placed in the wider context of other literature including most recent publications on the topic.

The exchange that was triggered by the FRAXBACK action was also a basis for a review on silvicultural strategies [16]. There are other reviews that cover evolutionary aspects regarding ash dieback [17], the possible pathways of the pathogen's introduction to Europe [18], the morphology and ecology of *H. fraxineus* (and some related species) [4], the quantitative genetics of disease resistance [19], general knowledge about *H. fraxineus* [5] and possible ecologic consequences of the disease [20].

Importance of Ash in Europe

There are four species of ash native to Europe. Common ash (*Fraxinus excelsior* L.) is the most widely distributed species and present in all European countries except Island, Malta and Portugal, but rather scarce in the Mediterranean. Here, narrow-leafed ash (*Fraxinus angustifolia* Vahl) and manna ash (*Fraxinus ornus* L.), which are restricted to more southern parts of Europe, are of higher importance. *F. ornus* is not very susceptible to ash dieback [21] and a rather small tree, although of economic importance in southern Europe [22]. The fourth species, *Fraxinus pallisiae* Wilmott ex Pallis, grows rarely in steppe and steppe-forest zones in south-eastern Europe, as does a subspecies of common ash, *F. excelsior* ssp. *coriariaefolia* [23]. The susceptibility of the latter two taxa to ash dieback is unknown. This review focuses on *F. excelsior* and *F. angustifolia*.

In most European countries, ash trees account for 1–2% of the forest area or standing volume stock [8, 23–28]. At the northern distribution range, the share of ash trees is even smaller [29–31], while ash trees hold a slightly higher proportion in forests of central Europe [21, 32–34]. Of particular importance is ash in England and Wales, where the proportion of this tree species in woodland ranges from 4.5 to 12.7% [35], and on the Island of Ireland, where ash is an omnipresent landscape component as a major part of hedgerows and woodland patches [36].

The economical importance of ash in Europe was much greater in ancient times and before the onset of the industrialization, when its wood was indispensable because of its toughness and bending properties and was used widely, e.g. for tools, arms and transport [37]. As a pollarding tree, ash has also been a source of fodder for livestock [29, 37]. In the last century, the use of ash timber has often been replaced by steel or other modern materials. However, ash timber is still a favourite for furniture, certain sports equipment, tool handles and some other goods (e.g. [25, 33]). An example in this respect is the hurl (used in traditional Gaelic sports hurley and camogie), which is crafted from ash wood [36]. Overall, the economic contribution of ash to forestry is considered significant in many regions of Europe, as is indicated in almost all country reports of the book [1]. Moreover, ash is very important from an ecological point of view. It provides critical ecological services, such as wildlife habitat and niche, stabilization of water balance and stream banks, production of high quality litter and as a component in slowing zones for forest fires [24, 25, 35, 38]. A large number of organisms are associated with or even entirely dependent on ash [21, 29, 30, 35, 39]. In forestry, ash was popular also because of its robustness, its drought tolerance, fast growth (on rich sites) and prolific regeneration. In the decades before the outbreak of the epidemic, forestry deliberately increased the proportion of ash in many regions [25, 26, 33].

The importance of ash is also demonstrated by a British study, which identified a wide range of national stakeholders for ash from government, the private sector and civil society that either have an influence on or a special interest in the ash dieback epidemic [40]. However, stakeholders in Romania tend to underestimate the importance of ash and the implications of ash dieback, as was concluded from a survey by Drăgoi et al. [41].

Gene Flow, Genetic Structure and Diversity of *H. fraxineus*

Several population studies demonstrated that in Europe there is low allelic diversity and little to no genetic differentiation between *H. fraxineus* populations [26, 29, 31, 34, 35, 42, 43]. Genetic differentiation was even small between long-established populations (from Lithuania) and populations from the epidemic front (in Switzerland), and there was no reduced genetic diversity in the front populations [26, 34]. Also between populations in the UK and Continental Europe there was only little genetic differentiation [43]. The uniform genetic structure throughout Europe indicates high-gene flow and a recent bottleneck due to a very small founder population. In fact, analyses of the coding regions of core eukaryotic genes in Asian and European isolates suggest that the European population was founded by two divergent haploid individuals [44]. This finding is supported by comparisons of European and eastern Asian populations, where much higher genetic diversity and similarly high-gene flow was observed [3, 34, 35]. An ancestry analysis indicates that the Russian Far East is the source of the European populations [3].

It can be assumed from expansion rates and gene flow analyses that viable ascospores are transmitted by wind

over long distances. However, on a small scale, along a tree species mixture gradient in Germany, some slight population differentiation was observed, which may have reflected recent colonization history [45]. Moreover, an increase of genetic diversity of *H. fraxineus* was observed along an altitude gradient in Poland [8, 46]. Population studies also indicate that the fungus is outcrossing and high-genotypic diversity was detected within the European populations [26, 29, 34, 35, 42]. These results may explain part of the variations in virulence, growth rates, temperature optima for growth and exoenzyme profiles, which have been observed among isolates [47–49].

Gene Flow, Genetic Structure and Diversity of Ash in Europe

The genetic structure of European *F. excelsior* populations is still shaped by the ice ages, despite the predominantly outcrossing nature of the species. After the last ice age, the present range was recolonized from glacial refugia in Iberia, Italy, the eastern Alps and the Balkan Peninsula, as was clearly shown by analyses of chloroplast DNA (cpDNA) variation of 62 populations throughout Europe [50]. In western and central Europe, admixture of populations from different refugia and high-gene flow during the recolonization process led to relatively high homogeneity, allelic richness and genetic diversity [36, 51]. For example, 96% of the variation was detected within Irish *F. excelsior* populations with little evidence of population structuring [36]. Results of a recent reciprocal transplanting experiment along a transect from Scotland to the Pyrenees led to an assumption that there is little local adaption in western Europe, as some populations grew well at all sites while local populations never performed best [35]. On the other hand, restricted postglacial gene flow caused by a rapid population expansion in the mountainous topography has likely caused rather strong genetic differentiation over short distances and less genetic diversity within populations in south-eastern Europe, as was suggested by a microsatellite study [51]. There is evidence that the present northern range of common ash was colonized from south-eastern Europe and similarly is characterized by rather limited gene flow and low-allelic richness [29, 31, 50, 52]. It is feared that these more fragmented populations are particularly vulnerable to presumable genetic effects of ash dieback such as loss of allelic structure and inbreeding [31]. In Lithuania, for example, it is very likely that the density of flowering trees and thus the effective population size has already decreased to a degree that compromises the remaining level of genetic diversity and possibilities for successful regeneration of ash populations [26].

When compared with other central European populations, higher genetic variation was detected in south-eastern Germany, where phylogenetic lineages from different refugia meet [33]. The transgression zone between the central and eastern European lineage along Hungary, Slovakia, Czech Republic and Poland could be detected in a corridor of 36 km width when analysing maternally inherited cpDNA, whereas the analysis of bi-parentally inherited nuclear gene markers detected an average width of the transgression zone of 275 km [27]. Another study in Germany confirmed that, while seed dispersal is rather restricted, there is considerable pollen flow at the landscape level [53]. In former times it was believed that *F. excelsior* trees from floodplain forest and from dry, calcareous sites form two ecotypes, but such distinction could never be proved by reciprocal transplants or progeny trials [21, 30]. There are inconsistent results concerning differences in observed heterozygosity between groups of differently susceptible *F. excelsior* trees in Germany and Poland [54, 55].

Similarly to *F. excelsior*, cpDNA analyses of *F. angustifolia* and *F. ornus* populations indicate distinct glacial refugia in southern Europe, with the south-eastern populations being relatively polymorphic [56]. Overall cpDNA diversity in these two species was lower than in *F. excelsior*. Generally, just like *F. excelsior*, *F. angustifolia* is a species with high intrapopulation genetic variation and low differentiation among populations [22]. Only little is known about local inbreeding or outcrossing rates and their impact on the genetic structure of *F. angustifolia* and *F. ornus* [38].

Genetic Resistance of Ash to *H. fraxineus*

Throughout Europe, there is a remarkable consistency in reports of genotypic differences in susceptibility of *F. excelsior* and *F. angustifolia* to ash dieback. Typically, about 1% of trees show no or only insignificant symptoms (Figure 1) and are considered resistant (or tolerant, depending on definition). Remarkably consistent is also the high degree of genetic control of this quantitative resistance, as was indicated by heritability values in a number of studies from several countries (Table 1). Heritability is defined as the proportion of the total variance due to genetic effects (e.g. [57]). Further evidence for genetic control of the resistance is provided from clonal seed orchards in Austria, the Netherlands, Croatia and Slovakia for both *F. excelsior* [21, 58] and *F. angustifolia* [21, 58–60]. There is also indication that development and severity of ash collar necroses are under considerable genetic control (h^2 (narrow-sense heritability) $= 0.49$ for prevalence and $h^2 = 0.42$ for necrosis width) [61]. Based on these studies it can be assumed with very high certainty that throughout the European populations there is a small proportion of ash trees with genetically controlled resistance. However, there was no significant correlation of damage data between parent ash trees and their offspring in natural regeneration in two heterogenic Austrian ash stands, indicating that environmental conditions and the interaction of genotypes with these play an important role *in situ* [61, 62].

Figure 1 This picture (taken in August 2018) shows neighbouring crowns of two *F. excelsior* trees in Gotland (Sweden), where the ash dieback pathogen arrived probably 2001 or 2002 [73]. Only minor parts of the left crown are still alive, because epicormic shoots, which were produced to compensate for dead twigs, gradually became diseased as well. In contrast, despite similar infection pressure for much more than a decade, the right tree is barely affected by ash dieback. It can be assumed that it possesses a relatively high degree of genetic resistance to ash dieback.

Although some part of the variation in the health status of different genotypes is probably due to disease escape or phenological mismatch (see section 'Influencing factors'), inoculation experiments clearly indicate that an active defence is involved [66]. The mechanisms that play a role in this defence are still virtually unknown. Different gene expression markers and a coding single-nucleotide polymorphism were associated with resistance to ash dieback and indicate a role of MADS box transcription factors (named after genes MCM1, AGAMOUS, DEFICIENS, and SRF) and iridoid glycoside [74, 75]. Fourier-transform infrared spectroscopy of phenolic extracts from bark tissue proved to be a relatively effective method to identify resistant individuals of ash, although on its own is not indicative regarding underlying resistance mechanisms [76]. However, such technologies can significantly advance efforts in selection and breeding for resistance.

Spread and Monitoring of Ash Dieback

According to Hietala et al. [77], the strong saprobic competence of *H. fraxineus* in defending the sporulation niche against abiotic stress and microbial competition, which enables production of a large amount of sexual spores, may be a main reason for the successful and invasive spread of this pathogen in Europe. The authors further assume that the ability to produce conidia may be connected to its invasion success, although the biological role of these asexual spores needs further investigation.

Our present knowledge of the spatiotemporal spread of the epidemic throughout Europe is based on dates of observations of symptoms and pathogen records, which is, however, biased by the spatiotemporally uneven presence of

forest pathology experts and their individual awareness of the disease. For example, *H. fraxineus* was first detected in England in 2012 on imported nursery stock, but mortality dating of ash trees indicated that ash dieback was already active in England in 2004 or 2005 [78]. A rough overview of the historical spread of ash dieback throughout Europe was given by McKinney et al. [19], showing a more or less concentric expansion from the epicentre of the disease, which is believed to be located in north-eastern Poland [8]. It is quite likely that the disease in its early stage can be overlooked and that the first introduction remained localized for a prolonged period. The subsequent concentric range expansion is documented in some countries on a regional scale, e.g. in Norway, Switzerland, France and Italy, typically with continuous steps of about 30–70 km per year [31, 34, 79, 80]. It is believed that this pattern of disease spread is mainly due to wind dispersal of airborne ascospores. In contrast, irregular or even inverse disease spread occurred in numerous other countries (e.g. [21, 27, 30, 33]), and there is broad agreement that this is an effect of anthropogenic movement of plant material, i.e. the trade of ash plants. It is probable that even to the UK, despite its isolated geographic situation, *H. fraxineus* was introduced by both wind dispersal and imports of infected plant material [43].

Large parts of central and eastern Europe have been infested before the first description of the causal agent in 2006 [6] and even larger areas before the correct identification of its teleomorph in 2011 [81]. This is why in mainland Europe there were no official attempts to limit the spread of the disease. *H. fraxineus* had been listed in the EPPO Altert List from 2007 to 2014, but has never been listed as a regulated organism (A1 or A2 List) in the Plant Health Directive of the European Union [82]. Only on the British Isles, legislations were changed in order to limit or prevent further introductions from mainland Europe by control of importation of plant material. Moreover, ash plants on infested sites were removed and destroyed [35, 36]. Although soon it became clear that there is no hope that these measures can lead to eradication of *H. fraxineus*, they were continued in Ireland in order to slow spread and build-up of the disease within the island [36]. Measures to prevent possible outbreaks of ash dieback were also taken in the northern Apennines, Italy, by the Regional Phytosanitary Services of Tuscany and Emilia Romagna [79]. There are concerns that introduction of further pathogen genetic diversity to Europe may cause increased disease severity. It is thus recommended to regulate imports of live ash plants and material that may contain ash leave debris from eastern Asia [44].

Ash dieback and its effects have been monitored in many local trials and forests throughout Europe. An extensive list of studies and sites was provided by Coker et al. [83]. These studies generated much data, which are, however, difficult to compare, because different scoring systems were used and the scoring of ash dieback and its severity is intricate and, in visual assessments, to some degree subjective. Kopinga and de Vries [84] compared different methods

Table 1 Heritability values for traits related to ash dieback severity (crown symptoms) as a measure for genetic control of resistance to ash dieback

Country	Trial type	Number of sites	Year of establishment	Time of data collection	Species	Heritability value(s)	Reference(s)
Denmark	Progeny	1	2003	2009–2014	*F. excelsior*	h^2: 0.42–0.53	[63]
Denmark	Progeny	2	2004	2008–2010	*F. excelsior*	h^2: 0.37–0.52	[64]
Denmark	Clonal	2	1998	2007–2009	*F. excelsior*	H^2: 0.25–0.54	[65, 66]
France	Progeny	1	1995	2010–2014	*F. excelsior*	h^2: 0.42	[61]
Germany	Clonal	4	1991–1995	2012–2013	*F. excelsior*	H^2: 0.48–0.59	[67]
Lithuania	Progeny	2	2005	2010	*F. excelsior*	h^2: 0.40–0.49	[26, 68]
Lithuania	Clonal	7	2012	2012–2013	*F. excelsior*	H^2: 0.21–0.65	[26, 69]
Slovenia	Clonal	1	1989	2009–2012	*F. angustifolia*	H^2: 0.23–0.50	[70]
Sweden	Clonal	2	1992–1995	2006–2011	*F. excelsior*	H^2: 0.10–0.42	[71, 72]

H^2, broad-sense heritability; h^2, narrow-sense heritability.
The values are based on studies with different settings and plant materials.

of ash dieback assessment in a large-scale survey. Examples for the assessed data are the degree of dieback, defoliation and the presence of epicormic shoots in percentage or ordinal scales. All evaluated specific aspects of ash dieback increased the accuracy of the health status assessments of trees and were significantly correlated, but a certain combination of aspects that is the most distinctive in monitoring of ash dieback has not yet been identified. Mortality is unambiguous and was recorded in many studies. It can serve as a measure of ash dieback severity, although it can have other causes. Mortality rates were reviewed and modelled including many different data sources in two recent papers, one using data from studies throughout Europe [83], another using data from Belgium and France [80]. Both studies suggest that the increase of annual mortality rates tend to stagnate at a level that depends on the forest stand after about 6–8 years of strong infection pressure. According to theory, future decrease of mortality rates due to a decrease of susceptibility in the remaining population and due to a dilution effect is likely, but extrapolation of mortality data with regards to ash dieback is still a matter of great uncertainty [14, 80, 83]. Also, detailed studies on the expansion of the pathogen within diseased trees indicated that a significant proportion of individual twig and branch infections stop expanding and eventually die out [85, 86]. This suggests that the dynamics of new and old infections within a tree are crucial for ash survival.

Influencing Factors

Severity of ash dieback in a stand depends on the time of pathogen arrival, the age of ash trees in the stand and several other factors. In numerous monitoring studies a number of factors that were connected to the severity of ash dieback symptoms and the extent of disease-induced mortality have been identified. An overview of factors that were associated with ash dieback is given in Table 2.

As shown there, most of these factors were features of individual trees. Characteristics that are related to age and size of trees are often negatively correlated with the severity of symptoms (i.e. the disease often progresses more slowly on larger or older trees). However, this result depends on the set of investigated trees, which is why such relationship had not been detected in every study (see Table 2 for reference). Moreover, the severity of symptoms is connected to the host tree species, since *F. angustifolia* seems to be slightly less susceptible than *F. excelsior* (and on *F. ornus* only minor symptoms occur on leaves). In a Dutch study, predominantly male ash trees performed better than trees with higher proportions of female flowers. Relatively strong differences in susceptibility were also reported for certain cultivars. Differences in susceptibility between central and western European provenances are only small, but Slovakian provenances apparently differ more, which possibly is connected to higher population differentiation (see the section 'Gene flow, genetic structure and diversity of ash in Europe'). In Lithuania, autochthonous provenances performed better than foreign ones, which may be due to already commenced natural selection in this long-time infested country, or due to genotype − environment interactions, which were found significant in some trials. Processes of natural selection may also be the reason for young seedlings performing better than regeneration that emerged by sprouting. Trees that are infected by *Armillaria* at the root collar are more likely to be severely damaged in the crown.

The severity of ash dieback symptoms also depends on environmental factors. There is a consensus that moisture and humidity is conducive for the disease. These factors seem to be especially important for the symptom of collar lesion [13]. It was speculated that the formation of apothecia on shoots and roots, which generally occurs only very rarely, may be of higher importance in the wet, maritime climate of Ireland [36]. Although it was not confirmed in a study on veteran trees in Sweden [39], it is a general observation that trees in forests are more severely affected than trees in the open landscape [21], which may be due to higher humidity and infection pressure. On the other hand, there is evidence that drought and high temperature are unfavourable for ash dieback [89].

Table 2 Factors that are associated with symptom severity or mortality due to ash dieback according to references in the FRAXBACK book [1]

Factor	Symptoms are more severe on/at ...	Reference(s)
Characteristics of individual trees		
Host species	... *F. excelsior* compared with *F. angustifolia* (depending on site)	[27, 84]
Gender	... male trees	[58]
Provenance	Not applicable	[25–27, 33, 66]
Cultivar	Not applicable	[25, 58, 84]
Leaf yellowing/leaf shed	... later shedding trees	[66]
Spring phenology	... later flushing trees	[25, 26, 29, 87]
Diameter at breast height (DBH)	... (inconsistently) trees of smaller DBH	[26, 29, 39, 80]
Crown surface projection	... trees with smaller crowns	[8]
Age	... (inconsistently) younger trees	[25, 33, 88]
Seedling versus sprouts	... sprouts	[26]
Armillaria root rot	... *Armillaria*-infected trees	[26]
Site conditions		
Altitude	... lower altitudes	[25]
Shade, woodland, open, grazed	... (inconsistently) shaded, forested sites	[21, 39, 84]
Temperature	... low to moderate temperature	[28, 79]
Drought, arid climate	... less dry sites	[21, 23, 28, 79]
Moisture, humidity	... more humid sites	[21, 79]
Soil type	... not specified	[27]
Management		
Pollarding, pruning	Inconsistent results	[39, 58]
Thinning	Not significant	[29]

The disease risk is considered to be low in warm areas of southern Europe and global warming is expected to alleviate the disease to some extent in the future [89, 90]. Thinning can change the microclimate and possibly alter conditions of infection and reduce disease severity. However, in young even-aged ash stands in Denmark, disease severity was the worst in unthinned plots, but otherwise unrelated to stand density [91]. Pruning of diseased branches reduces the probability of stem infections, although such control tactic may not be economical [92]. Interestingly, old trees that had been pollarded some decades ago are less affected than other more recently or not at all pollarded veteran trees [39]. Although some fungal ash endophytes with high *in vitro* antagonistic activity against *H. fraxineus* have been identified [93–96], an influence of endophytic mycobiomes on the health status of ash trees (i.e. the susceptibility to ash dieback) or on the establishment of the pathogen has not yet been demonstrated [77, 94, 96, 97].

Impact of Ash Dieback on Timber Logging

An obvious reaction to ash dieback is increased timber logging due to salvage cuttings. Published data on this increase, however, are relatively rare and often of rather indirect nature. Swedish national forest inventory data reveal a notable decline of the standing timber stock since 2009, which is probably due to increased cuttings in stands damaged by ash dieback [29]. In Lithuania, where symptoms of ash dieback were first observed in 1996, heavy salvage cutting (selective felling and clearcutting of more than

1500 ha of ash stands) commenced in 2001 [26]. Annual salvage cutting reached its maximum in 2006 (about 3500 ha) and smoothly decreased down to about 1000 ha in 2015. At this time, 18 years after the first observation of symptoms, the former area of ash stands was reduced by more than 50% [26]. Clearcutting was also carried out in other countries. For example, as an ultimate option, immature but heavily devastated ash stands often have been clearcut in Austria [21, 98]. In Grafenegg (Austria), where ash dieback arrived about 2005, logging of ash more than doubled by 2011, quadrupled by 2014 and sextupled by 2015 when compared with 2008. A similar exponential increase in timber logging is reported from state owned forests in Baden-Württemberg, Germany [98], and from Denmark [19, 98]. In Wallonia (Belgium), a very sharp increase in the number of ash timber lots greater than 50 m^3 was recorded in 2015 [32]. In summary, it can be said that sharp increases of ash timber logging commenced 5–8 years after ash dieback symptoms were detected in a region or country for the first time.

Management of Ash Dieback

Silvicultural strategies in response to ash dieback were reviewed comprehensively by Skovsgaard et al. [16], who concluded with the main advice to retain tolerant-appearing ash trees wherever reasonable within the context of management objectives and overall forest structure in order to preserve these genetic resources (i.e. heritable resistance). However, there are reports from numerous countries that forest owners do not always

follow this advice, which may be due to the lack of information [39], economic reasons [26] or general loss of interest and expectation regarding forestry with ash [21, 25, 26, 33]. The latter often is connected to the experience of observing declining trees that appeared resistant in the early stages of the disease.

Generally, official recommendations for disease management in the ash dieback-affected countries are in line with the strategies that are proposed in the silviculture review [16], but there are regional adaptations and emphases of certain aspects. For example, measures to prevent disease spread and inoculum build-up (i.e. destruction of infected plants, plant material and leaf litter) were carried out extensively in the British Isles, although with low impact [35, 36]. In contrast, in continental Europe there were only a few local attempts to slow down the spread of the disease, e.g. in order to prevent outbreaks in the Apennines in Italy [79]. Awareness of the risks rose too late for efficient quarantine measures in large parts of Europe, where the disease spread before fundamental knowledge was available.

In most infested countries nursing and planting of new ash trees is not recommended and has decreased sharply since the onset of the disease [24, 25, 29, 32, 33, 58, 66]. In Poland, the production of ash plant material even was banned completely [8]. On the other hand, large numbers of ash plants were still produced in Slovakia (up to 873 000 plants in 2015), where 150 000 to 175 000 trees were annually planted in the forests [27]. In 2017, planting of ash was still subsidized in Flanders, Ireland and Greece [22, 24, 36]. At the northern limit of the range of *F. excelsior*, planting of healthy seedlings from local sources is considered to be the best option to prevent loss of genetic diversity on the one hand and loss of local adaption on the other [31], which both is crucial in these areas (see the section 'Gene flow, genetic structure and diversity of ash in Europe'). A similar strategy is also proposed in so far not infected areas, i.e. in Portugal, with the aim to increase among- and within-population genetic diversity and hence prepare ash populations for invasive pathogens such as *H. fraxineus* [38].

Tending and early thinning is recommended in Poland in order to increase tree vigour, which helps ash trees to survive the disease, by fostering the growth of crowns and root systems [8]. In Germany, such early promotion is recommended only for resistant appearing ash trees [33], whereas in Lithuania pre-commercial thinning is recommended only in heavily damaged stands with low proportions of ash [26].

For the management of middle-aged stands there are different concepts that depend on the severity of the disease and the proportion of ash. Generally, stands should be thinned as usual to promote canopy development while focusing on retaining resistant appearing trees and species other than ash. Foresters in Sweden are advised to mark the healthiest ash trees in order to avoid cutting these in error and to enable somewhat objective observations of the development of the disease over time [29]. In Norway it was recommended to preferably remove susceptible ash trees in order to lower the local overall infection pressure and to reduce their genetic influence on the next tree generation [31]. However, new research demonstrated that trees with higher levels of crown damage produce much less amounts of propagules than healthier trees [99]. This relationship is stronger for seeds than for pollen, so it seems recommendable to focus particularly on diseased male trees when thinning. Valuable timber should be harvested before wood quality deteriorates. If, however, it can be expected that the damage will become too large to allow for silvicultural control of stand development, salvage felling of larger parts of the stand or even clearcutting may be the only options.

Replacement of ash trees that have died or have been felled due to the disease can be achieved by either natural forest regeneration or planting. Natural regeneration of ash can be used and even fostered, e.g. by retaining some relatively healthy ash individuals as seed trees, as is recommended in Lithuania and Estonia [18, 26]. Since planting of new ash trees is, if at all, recommended only exceptionally (see above), the choice of alternative tree species needs to be considered carefully. Especially in monocultural ash woodlands, which often is the case in Ireland, it is suggested to aim for a transition to more mixed species forests, in order to build a greater degree of resilience [100]. There is a consensus that replacement in forests by non-native *Fraxinus* species is not a preferable option, even if these species are more resistant to ash dieback [16, 18, 21, 23, 25, 30, 33]. Instead, recommendations for alternative tree species are mainly based on specific site adaption and thus differ between regions and countries. In Great Britain, the ability of tree species and species mixtures to substitute the ecosystem functions of ash is considered as an additional important criterion [35, 101].

Conclusion

Ash species native to Europe provide important ecosystem services and are considered of high value both as ornamental trees and as a forest component. In many places, particularly in central Europe and the British Isles, wood production with *F. excelsior* or *F. angustifolia* contributes significantly to local economy. There is little genetic differentiation of ash populations in western and central Europe, but some population differentiation is characteristic to its eastern and, due to local adaptation, particularly to its very northern range. The European population of *H. fraxineus* is characterized by extremely low genetic variation and local differentiation, which is a result of its recent introduction, its subsequent rapid spread and effective sexual outcrossing. The disease spread both by wind dispersal of ascospores and by human-mediated transportation of infected plant material. Large parts of

the continent were infested before the identification of *H. fraxineus* as the causal agent of ash dieback, and sharp increases of ash timber logging typically commenced 5–8 years after ash dieback symptoms were detected for the first time in a region.

Severity of ash dieback is influenced by individual tree characteristics (e.g. tree age) and site conditions (e.g. moisture or humidity), but there is rather little and inconsistent evidence for an influence of forest management practices. Strategies for disease management, which differ slightly between countries, thus aim mainly for the retention and propagation of resistant or tolerant trees. Heritability values for ash crown damage and other disease-related traits from numerous clonal trials and progeny trials were consistently high, regardless of their location in Europe. This is an indication for strong genetic control of these traits, although it was not yet possible to demonstrate a connection of tree health between parent trees and natural regeneration under heterogeneous conditions.

Acknowledgements

References

1. Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. 299 p. Available from: URL: http://www.slu.se/globalassets/ew/org/inst/mykopat/forskning/stenlid/dieback-of-european-ash.pdf.

2. Enderle R, Pliura A, Vasaitis R, editors. Advances in ash dieback research – and some other invasive diseases of trees. Baltic Forestry 2017;23(1):1–333. Available from: URL: https://www.balticforestry.mi.lt/bf/index.php?option=com_content&view=article&catid=14&id=482.

3. Sønstebø JH, Vivian-Smith A, Adamson K, Drenkhan R, Solheim H, Hietala A. Genome-wide population diversity in *Hymenoscyphus fraxineus* points to an eastern Russian origin of European ash dieback. bioRxiv 2017:154492. doi: 10.1101/154492.

4. Baral H-O, Bemmann M. *Hymenoscyphus fraxineus* vs. *Hymenoscyphus albidus* – A comparative light microscopic study on the causal agent of European ash dieback and related foliicolous, stroma-forming species. Mycology 2014;5(4):228–90. doi: 10.1080/21501203.2014.963720. PubMed PMID: 25544935.

5. Gross A, Holdenrieder O, Pautasso M, Queloz V, Sieber TN. *Hymenoscyphus pseudoalbidus*, the causal agent of European ash dieback. Molecular Plant Pathology 2014;15(1):5–21. doi: 10.1111/mpp.12073. PubMed PMID: 24118686.

6. Kowalski T. *Chalara fraxinea* sp. nov. associated with dieback of ash (*Fraxinus excelsior*) in Poland. Forest Pathology 2006;36(4):264–70. doi: 10.1111/j.1439-0329.2006.00453.x.

7. Timmermann V, Børja I, Hietala AM, Kirisits T, Solheim H. Ash dieback: pathogen spread and diurnal patterns of ascospore dispersal, with special emphasis on Norway. EPPO Bulletin 2011;41(1):14–20. doi: 10.1111/j.1365-2338.2010.02429.x.

8. Gil W, Kowalski T, Kraj W, Zachara T, Łukaszewicz J, Paluch R, *et al.* Ash dieback in Poland – History of the phenomenon and possibilities of its limitation. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 176–84.

9. Haňáčková Z, Koukol O, Čmoková A, Zahradník D, Havrdová L. Direct evidence of *Hymenoscyphus fraxineus* infection pathway through the petiole-shoot junction. Forest Pathology 2017;47(6):e12370. doi: 10.1111/efp.12370.

10. Husson C, Caël O, Grandjean JP, Nageleisen LM, Marçais B. Occurrence of *Hymenoscyphus pseudoalbidus* on infected ash logs. Plant Pathology 2012;61(5):889–95. doi: 10.1111/j.1365-3059.2011.02578.x.

11. Chandelier A, Gerarts F, San Martin G, Herman M, Delahaye L. Temporal evolution of collar lesions associated with ash dieback and the occurrence of Armillaria in Belgian forests. Forest Pathology 2016;46(4):289–97. doi: 10.1111/efp.12258.

12. Enderle R, Peters F, Nakou A, Metzler B. Temporal development of ash dieback symptoms and spatial distribution of collar rots in a provenance trial of *Fraxinus excelsior*. European Journal of Forest Research 2013;132(5–6):865–76. doi: 10.1007/s10342-013-0717-y.

13. Marçais B, Husson C, Godart L, Caël O. Influence of site and stand factors on *Hymenoscyphus fraxineus*-induced basal lesions. Plant Pathology 2016;65(9):1452–61. doi: 10.1111/ppa.12542.

14. Enderle R, Sander F, Metzler B. Temporal development of collar necroses and butt rot in association with ash dieback. iForest 2017;10(3):529–36. doi: 10.3832/ifor2407-010.

15. Langer G. Collar rots in forests of Northwest Germany affected by ash dieback. Baltic Forestry 2017;23(1):4–19.

16. Skovsgaard JP, Wilhelm GJ, Thomsen IM, Metzler B, Kirisits T, Havrdová L, *et al.* Silvicultural strategies for *Fraxinus excelsior* in response to dieback caused by *Hymenoscyphus fraxineus*. Forestry: An International Journal of Forest Research 2017;90(4):455–72. doi: 10.1093/forestry/cpx012.

17. Landolt J, Gross A, Holdenrieder O, Pautasso M. Ash dieback due to *Hymenoscyphus fraxineus*: what can be learnt from evolutionary ecology? Plant Pathology 2016;65(7):1056–70. doi: 10.1111/ppa.12539.

18. Drenkhan R, Sander H, Hanso M. Introduction of Mandshurian ash (*Fraxinus mandshurica* Rupr.) to Estonia: Is it related to the current epidemic on European ash (*F. excelsior* I.)? European Journal of Forest Research 2014;133(5):769–81. doi: 10.1007/s10342-014-0811-9.

19. McKinney LV, Nielsen LR, Collinge DB, Thomsen IM, Hansen JK, Kjaer ED. The ash dieback crisis: genetic variation in resistance can prove a long-term solution. Plant Pathology 2014;63(3):485–99. doi: 10.1111/ppa.12196.

20. Pautasso M, Aas G, Queloz V, Holdenrieder O. European ash (*Fraxinus excelsior*) dieback – A conservation biology challenge. Biological Conservation 2013;158:37–49. doi: 10.1016/j.biocon.2012.08.026.

21. Heinze B, Tiefenbacher H, Litschauer R, Kirisits T. Ash dieback in Austria – history, current situation and outlook. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 33–52.

22. Spanos K, Gaitanis D. An overview of ash species in Greece: ecology, biology and taxonomy, silviculture, genetics and health status. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 273–83.

23. Chira D, Chira F, Tăut I, Popovici O, Blada I, Doniță N, *et al.* Evolution of ash dieback in Romania. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 185–94.

24. Sioen G, Roskams P, de Cuyper B, Steenackers M. Ash dieback in Flanders (Belgium): research on disease development, resistance & management. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 61–67.

25. Rozsypálek J, Dvořák M, Longauer R, Botella L, Prouza M, Palovčíková D, *et al.* Ash and ash dieback in the Czech Republic. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 79–88.

26. Pliūra A, Bakys R, Suchockas V, Marčiulynienė D, Gustienė A, Verbyla V, *et al.* Ash dieback in Lithuania: disease history, research on impact and genetic variation in disease resistance, tree breeding and options for forest management. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 150–66.

27. Longauerová V, Kunca A, Longauer R, Maľová M, Leontovyč R. The ash and ash dieback in Slovakia. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 209–19.

28. Davydenko K, Meshkova V. The current situation concerning severity and causes of ash dieback in Ukraine caused by *Hymenoscyphus fraxineus*. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 220–27.

29. Cleary M, Nguyen D, Stener DG, Stenlid J, Skovsgaard JP. Ash and ash dieback in Sweden: A review of disease history, current status, pathogen and host dynamics, host tolerance and management options in forests and landscapes. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 195–208.

30. Drenkhan R, Agan A, Palm K, Rosenvald R, Jürisoo L, Maaten T, *et al.* Overview of ash and ash dieback in Estonia. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 115–24.

31. Børja I, Timmermann V, Hietala AM, Tollefsrud MM, Nagy NE, Vivian-Smith A, *et al.* Ash dieback in Norway – current situation. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 166–75.

32. Chandelier A, Delahaye L, Claessens H, Lassois L. Ash dieback in Wallonia, southern Belgium: research on disease development, resistance & management options. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 53–60.

33. Enderle R, Fussi B, Lenz HD, Langer G, Nagel R, Metzler B. Ash dieback in Germany: research on disease development, resistance and management options. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 89–105.

34. Queloz V, Hopf S, Schoebel CN, Rigling D, Gross A. Ash dieback in Switzerland: history and scientific achievements. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 68–78.

35. Clark J, Webber J. The ash resource and the response to ash dieback in Great Britain. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 228–37.

36. McCracken AR, Douglas GC, Ryan C, Destefanis M, Cooke LR. Ash dieback on the island of Ireland. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 125–39.

37. Pratt J. Management and use of ash in Britain from the prehistoric to the present: some implications for its preservation. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 1–14.

38. Bragança H, Varela M. C. *Fraxinus angustifolia* and forest health in Portugal – an overview. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 284–87.

39. Bengtsson V, Senström A. Ash dieback – a continuing threat to veteran ash trees? In: Vasaitis R, Enderle R, editors. Dieback

of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 262–72.

40. Dandy N, Marzano M, Porth EF, Urquhart J, Potter C. Who has a stake in ash dieback? A conceptual framework for the identification and categorisation of tree health stakeholders. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 15–26.

41. Drăgoi M, Chira D, Popovici O. Socio-economic outcomes of ash-dieback in Romania. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 27–32.

42. Bengtsson SBK, Vasaitis R, Kirisits T, Solheim H, Stenlid J. Population structure of *Hymenoscyphus pseudoalbidus* and its genetic relationship to Hymenoscyphus albidus. Fungal Ecology 2012;5(2):147–53. doi: 10.1016/j.funeco.2011.10.004.

43. Orton ES, Brasier CM, Bilham LJ, Bansal A, Webber JF, Brown JKM. Population structure of the ash dieback pathogen, *Hymenoscyphus fraxineus*, in relation to its mode of arrival in the UK. Plant Pathology 2018;67(2):255–64. doi: 10.1111/ppa.12762. PubMed PMID: 29527064.

44. McMullan M, Rafiqi M, Kaithakottil G, Clavijo BJ, Bilham L, Orton E, *et al.* The ash dieback invasion of Europe was founded by two genetically divergent individuals. Nature Ecology and Evolution 2018;2(6):1000–8. doi: 10.1038/s41559-018-0548-9. PubMed PMID: 29686237.

45. Nguyen DT, Cleary MR, Enderle R, Berlin A, Stenlid J. Analyses of the ash dieback pathogen, *Hymenoscyphus fraxineus*, suggest role of tree species diversity on colonization and population structure differentiation. Forest Pathology 2016;46(1):82–4. doi: 10.1111/efp.12236.

46. Kraj W, Zarek M, Kowalski T. Genetic variability of *Chalara fraxinea*, dieback cause of European ash (*Fraxinus excelsior* L.). Mycological Progress 2012;11(1):37–45. doi: 10.1007/s11557-010-0724-z.

47. Kowalski T, Bartnik C. Morphological variation in colonies of *Chalara fraxinea* isolated from ash (*Fraxinus excelsior* L.) stems with symptoms of dieback and effects of temperature on colony growth and structure. Acta Agrobotanica 2010;63(1):99–106.

48. Junker C, de Vries J, Eickhorst C, Schulz B. Each isolate of *Hymenoscyphus fraxineus* is an individualist as shown by exoenzyme and growth rate profiles. Baltic Forestry 2017;23(1):25–40.

49. Lygis V, Prospero S, Burokiene D, Schoebel CN, Marčiulynienė D, Norkute G, *et al.* Virulence of the invasive ash pathogen *Hymenoscyphus fraxineus* in old and recently established populations. Plant Pathology 2017;66(5):783–91. doi: 10.1111/ppa.12635.

50. Heuertz M, Fineschi S, Anzidei M, Pastorelli R, Salvini D, Paule L, *et al.* Chloroplast DNA variation and postglacial recolonization of common ash (*Fraxinus excelsior* L.) in Europe. Molecular Ecology 2004;13(11):3437–52. doi: 10.1111/j.1365-294X.2004.02333.x. PubMed PMID: 15488002.

51. Heuertz M, Hausman J-F, Hardy OJ, Vendramin GG, Frascaria-Lacoste N, Vekemans X. Nuclear microsatellites reveal contrasting patterns of genetic structure between western and southeastern European populations of the Common ash (*Fraxinus excelsior* L.). Evolution 2004;58(5):976–88. doi: 10.1111/j.0014-3820.2004.tb00432.x.

52. Tollefsrud MM, Myking T, Sønstebø JH, Lygis V, Hietala AM, Heuertz M. Genetic structure in the northern range margins of Common ash, *Fraxinus excelsior* L. PLoS One 2016;11(12):e0167104. doi: 10.1371/journal.pone.0167104. PubMed PMID: 27907032.

53. Semizer-Cuming D, Kjær ED, Finkeldey R. Gene flow of common ash (*Fraxinus excelsior* L.) in a fragmented landscape. PLoS One 2017;12(10):e0186757. doi: 10.1371/journal.pone.0186757. PubMed PMID: 29053740.

54. Fussi B, Konnert M. Genetic analysis of European common ash (*Fraxinus excelsior* L.) populations affected by ash dieback. Silvae Genetica 2014;63(5):198–212.

55. Pacia A, Nowakowska JA, Tkaczyk M, Sikora K, Tereba A, Borys M, *et al.* Common ash stand affected by ash dieback in the Wolica Nature Reserve in Poland. Baltic Forestry 2017;23(1):183–97.

56. Heuertz M, Carnevale S, Fineschi S, Sebastiani F, Hausman JF, Paule L, *et al.* Chloroplast DNA phylogeography of European ashes, *Fraxinus* sp. (Oleaceae): roles of hybridization and life history traits. Molecular Ecology 2006;15(8):2131–40. doi: 10.1111/j.1365-294X.2006.02897.x. PubMed PMID: 16780430.

57. Burton GW, Devane EH. Estimating heritability in tall fescue (*Festuca arundinacea*) from replicated clonal material. Agronomy Journal 1953;45(10):478–81.

58. de Vries SMG, Kopinga J. Differences in susceptibility to *Hymenoscyphus fraxineus* (twig dieback of ash) of selections of Common ash (*Fraxinus excelsior*) in The Netherlands – report of the observations and results of 2012 and 2015. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 238–48.

59. Diminić D, Kajba D, Milotić M, Andrić I, Kranjec J. Susceptibility of *Fraxinus angustifolia* clones to *Hymenoscyphus fraxineus* in lowland Croatia. Baltic Forestry 2017;23(1):233–43.

60. Adamčíková K, Pažitný J, Pastirčáková K. Individual resistance of *Fraxinus angustifolia* and *F. excelsior* clones to *Hymenoscyphus fraxineus*. Journal of Plant Protection Research 2018;58(3):227–33.

61. Muñoz F, Marçais B, Dufour J, Dowkiw A. Rising out of the ashes: additive genetic variation for crown and collar resistance to *Hymenoscyphus fraxineus* in *Fraxinus excelsior*. Phytopathology 2016;106(12):1535–43. doi: 10.1094/PHYTO-11-15-0284-R. PubMed PMID: 27349738.

62. Wohlmuth A, Essl F, Heinze B. Genetic analysis of inherited reduced susceptibility of *Fraxinus excelsior* L. seedlings in Austria to ash dieback. Forestry: An International Journal of Forest Research 2018;91(4):514–25. doi: 10.1093/forestry/cpy012.

63. Lobo A, Hansen JK, McKinney LV, Nielsen LR, Kjær ED. Genetic variation in dieback resistance: growth and survival of *Fraxinus excelsior* under the influence of *Hymenoscyphus pseudoalbidus*. Scandinavian Journal of Forest Research 2014;29(6):519–26. doi: 10.1080/02827581.2014.950603.

64. Kjær ED, McKinney LV, Nielsen LR, Hansen LN, Hansen JK. Adaptive potential of ash (*Fraxinus excelsior*) populations

against the novel emerging pathogen *Hymenoscyphus pseudoalbidus*. Evolutionary Applications 2012;5(3):219–28. doi: 10.1111/j.1752-4571.2011.00222.x. PubMed PMID: 25568043.

65. McKinney LV, Nielsen LR, Hansen JK, Kjær ED. Presence of natural genetic resistance in *Fraxinus excelsior* (Oleraceae) to *Chalara fraxinea* (Ascomycota): an emerging infectious disease. Heredity (Edinb) 2011;106(5):788–97. doi: 10.1038/hdy.2010.119. PubMed PMID: 20823903.

66. Kjær ED, McKinney LV, Hansen LN, Olrik DC, Lobo A, Thomsen IM, *et al.* Genetics of ash dieback resistance in a restoration context – experiences from Denmark. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 106–14.

67. Enderle R, Nakou A, Thomas K, Metzler B. Susceptibility of autochthonous German *Fraxinus excelsior* clones to *Hymenoscyphus pseudoalbidus* is genetically determined. Annals of Forest Science 2015;72(2):183–93. doi: 10.1007/s13595-014-0413-1.

68. Pliura A, Lygis V, Suchockas V, Bartkevièius E. Performance of twenty four European *Fraxinus excelsior* populations in three Lithuanian progeny trials with a special emphasis on resistance to *Chalara fraxinea*. Baltic Forestry 2011;17(1):17–34.

69. Pliura A, Marèiulynienè D, Bakys R, Suchockas V. Dynamics of genetic resistance to *Hymenoscyphus pseudoalbidus* in juvenile *Fraxinus excelsior* clones. Baltic Forestry 2014;20(1):10–27.

70. Hauptman T, Ogris N, de Groot M, Piškur B, Jurc D. Individual resistance of *Fraxinus angustifolia* clones to ash dieback. Forest Pathology 2016;46(4):269–80. doi: 10.1111/efp.12253.

71. Stener L-G. Clonal differences in susceptibility to the dieback of *Fraxinus excelsior* in southern Sweden. Scandinavian Journal of Forest Research 2013;28(3):205–16. doi: 10.1080/02827581.2012.735699.

72. Stener L-G. Genetic evaluation of damage caused by ash dieback with emphasis on selection stability over time. Forest Ecology and Management 2018;409:584–92. doi: 10.1016/j.foreco.2017.11.049.

73. Östbrant I-L, Vasaitis R, Stenlid J, Pliūra A, Menkis A. Natura 2000 habitats dominated by ash and elm, invaded by alien invasive fungi on the Gotland Island of Sweden: an overview. Baltic Forestry 2017;23(1):264–9.

74. Harper AL, McKinney LV, Nielsen LR, Havlickova L, Li Y, Trick M, *et al.* Molecular markers for tolerance of European ash (*Fraxinus excelsior*) to dieback disease identified using Associative Transcriptomics. Scientific Reports 2016;6:19335. doi: 10.1038/srep19335. PubMed PMID: 26757823.

75. Sollars ESA, Harper AL, Kelly LJ, Sambles CM, Ramirez-Gonzalez RH, Swarbreck D, *et al.* Genome sequence and genetic diversity of European ash trees. Nature 2017;541(7636):212–6. doi: 10.1038/nature20786. PubMed PMID: 28024298.

76. Villari C, Dowkiw A, Enderle R, Ghasemkhani M, Kirisits T, Kjær ED, *et al.* Advanced spectroscopy-based phenotyping offers a potential solution to the ash dieback epidemic. Scientific Reports; 2018.

77. Hietala AM, Børja I, Cross H, Nagy NE, Solheim H, Timmermann V, *et al.* Dieback of European ash: what can we learn from the microbial community and species-specific traits of endophytic fungi associated with ash? In: Pirttilä AM, Frank AC, editors. Endophytes of Forest Trees: Biology and Applications. Forestry Sciences. Cham, Switzerland: Springer International Publishing; 2018. p. 229–58.

78. Wylder B, Biddle M, King K, Baden R, Webber J. Evidence from mortality dating of *Fraxinus excelsior* indicates ash dieback (*Hymenoscyphus fraxineus*) was active in England in 2004–2005. Forestry: An International Journal of Forest Research 2018;91(4):434–43. doi: 10.1093/forestry/cpx059.

79. Ghelardini L, Migliorini D, Santini A, Pepori AL, Maresi G, Vai N, *et al.* From the Alps to the Apennines: possible spread of ash dieback in Mediterranean areas. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 140–49.

80. Marçais B, Husson C, Caël O, Dowkiw A, Saintonge F-X, Delahaye L, *et al.* Estimation of ash Mortality Induced by *Hymenoscyphus fraxineus* in France and Belgium. Baltic Forestry 2017;23(1):159–67.

81. Queloz V, Grünig CR, Berndt R, Kowalski T, Sieber TN, Holdenrieder O. Cryptic speciation in *Hymenoscyphus albidus*. Forest Pathology 2011;41(2):133–42. doi: 10.1111/j.1439-0329.2010.00645.x.

82. European Plant Protection Organisation (EPPO). Mini data sheet on *Chalara fraxinea* [Internet]; 2014 [cited 2019 Feb 12]. Available from: URL: https://gd.eppo.int/download/doc/991_minids_CHAAFR.pdf.

83. Coker TLR, Rozsypálek J, Edwards A, Harwood TP, Butfoy L, Buggs RJA. Estimating mortality rates of European ash (*Fraxinus excelsior*) under the ash dieback (*Hymenoscyphus fraxineus*) epidemic. Plants, People, Planet 2019;1(1):48–58. doi: 10.1002/ppp3.11.

84. Kopinga J, de Vries SMG. Ash DieBack (ADB) in amenity trees in the city of Amsterdam – development of a monitoring system and the first results of a large-scale survey in 2015. In: Vasaitis R, Enderle R, editors. Dieback of European ash (*Fraxinus* spp.) – Consequences and Guidelines for Sustainable Management. Uppsala, Sweden: Swedish University of Agricultural Sciences; 2017. p. 249–61.

85. Matisone I, Matisons R, Kenigsvalde K, Gaitnieks T, Burneviča N. Seasonal development of lesions caused by *Hymenoscyphus fraxineus* on young *Fraxinus excelsior* trees in Latvia. iForest 2018;11(1):17–23. doi: 10.3832/ifor2283-010.

86. Bengtsson SBK, Barklund P, von Brömssen C, Stenlid J. Seasonal pattern of lesion development in diseased *Fraxinus excelsior* infected by *Hymenoscyphus pseudoalbidus*. PLoS One 2014;9(4):e76429. doi: 10.1371/journal.pone.0076429. PubMed PMID: 24759550.

87. Nielsen LR, McKinney LV, Kjær ED. Host phenological stage potentially affects dieback severity after *Hymenoscyphus fraxineus* infection in *Fraxinus excelsior* seedlings. Baltic Forestry 2017;23(1):229–32.

88. Enderle R, Metzler B, Riemer U, Kaendler G. Ash dieback on sample points of the National Forest Inventory in south-western Germany. Forests 2018;9(1):1–13. doi: 10.3390/f9010025.

89. Grosdidier M, Ioos R, Marçais B. Do higher summer temperatures restrict the dissemination of *Hymenoscyphus fraxineus* in France? Forest Pathology 2018;48(4):e12426. doi: 10.1111/efp.12426.

90. Goberville E, Hautekèete N-C, Kirby RR, Piquot Y, Luczak C, Beaugrand G. Climate change and the ash dieback crisis. Scientific Reports 2016;6:35303. doi: 10.1038/srep35303. PubMed PMID: 27739483.

91. Bakys R, Vasaitis R, Skovsgaard JP. Patterns and severity of crown dieback in young even-aged stands of European ash (*Fraxinus excelsior* L.) in relation to stand density, bud flushing phenotype, and season. Plant Protection Science 2013;49 (No. 3):120–6. doi: 10.17221/70/2012-PPS.

92. Marciulyniene D, Davydenko K, Stenlid J, Cleary M. Can pruning help maintain vitality of ash trees affected by ash dieback in urban landscapes? Urban Forestry & Urban Greening 2017;27:69–75. doi: 10.1016/j.ufug.2017.06.017.

93. Kosawang C, Amby DB, Bussaban B, McKinney LV, Xu J, Kjær ED, *et al.* Fungal communities associated with species of *Fraxinus* tolerant to ash dieback, and their potential for biological control. Fungal Biology 2018;122(2–3):110–20. doi: 10.1016/j.funbio.2017.11.002. PubMed PMID: 29458714.

94. Haňáčková Z, Havrdová L, Černý K, Zahradník D, Koukol O. Fungal endophytes in ash shoots – diversity and inhibition of *Hymenoscyphus fraxineus*. Baltic Forestry 2017;23(1):89–106.

95. Schulz B, Haas S, Junker C, Andrée N, Schobert M. Fungal endophytes are involved in multiple balanced antagonisms. Current Science 2015;109(1):39–45.

96. Schlegel M, Dubach V, von Buol L, Sieber TN. Effects of endophytic fungi on the ash dieback pathogen. FEMS Microbiology Ecology 2016;92(9):1–8. doi: 10.1093/femsec/fiw142. PubMed PMID: 27364360.

97. Schlegel M, Queloz V, Sieber TN. The endophytic mycobiome of European ash and sycamore maple leaves – geographic patterns, host specificity and influence of ash dieback. Frontiers in Microbiology 2018;9:2345. doi: 10.3389/fmicb.2018.02345. PubMed PMID: 30405540.

98. ForstBW. Herausforderung Eschentriebsterben: Waldbauliche Behandlung geschädigter Bestände. [Internet]. Stuttgart; 2018 [cited 2018 Jul 18]. Available from: URL: http://www.forstbw.de/fileadmin/forstbw_infothek/forstbw_praxis/ForstBW_Praxis_Eschentriebsterben_20180327.pdf.

99. Semizer-Cuming D, Finkeldey R, Nielsen LR, Kjær ED. Negative correlation between ash dieback susceptibility and reproductive success: good news for European ash forests. Annals of Forest Science 2019;76(1):219. doi: 10.1007/s13595-019-0799-x.

100. Short I, Hawe J. Ash dieback in Ireland – A review of European management options and case studies in remedial silviculture. Irish Forestry [Internet] 2018 [cited 2019 Feb 15];75:44–72. Available from: URL: https://t-stor.teagasc.ie/handle/11019/1654.

101. Broome A, Ray D, Mitchell R, Harmer R. Responding to ash dieback (*Hymenoscyphus fraxineus*) in the UK: woodland composition and replacement tree species. Forestry: An International Journal of Forest Research 2019;92(1):108–19. doi: 10.1093/forestry/cpy040.