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**The Natural Evolutionary Potential of Tree Populations to Cope with Newly Introduced Pests and Pathogens—Lessons Learned From Forest Health Catastrophes in Recent Decades**

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4 **The natural evolutionary potential of tree populations to cope with newly introduced pests and**  
5 **pathogens – lessons learned from forest health catastrophes in recent decades**

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10

11 **Abstract**

12 Emerging diseases often originate from host shifts of introduced pests or pathogens. Genetic resistance of  
13 the host to such diseases might be limited or absent due to the lack of co-evolutionary history. We review  
14 six examples of major disease outbreaks on native tree species caused by different introduced pests and  
15 pathogens that led to large ecological and economical losses. In all six cases high tree mortality was  
16 observed in natural populations with some surviving individuals exhibiting varying levels of genetic  
17 resistance. The abundance and distribution of resistant individuals and the heritability of resistance traits  
18 varies substantially among the cases. While chestnut blight wilt combined with ink disease has virtually  
19 eliminated mature *Castanea dentata* trees from North America, other severe emerging diseases, such as  
20 the ash dieback, have left many surviving trees and genetic variation in resistance to such diseases has been  
21 documented. We argue that the evolutionary potential of tree species to respond to new emerging  
22 diseases should not be underestimated. However, the risk of increased levels of inbreeding and loss of  
23 genetic diversity caused by low population sizes is a major concern. Maintenance of broad genetic diversity  
24 is an important issue in conservation and forestry management. We expect that future research targeting  
25 the genetic background of biotic resistance towards emerging diseases, and the role of endophytic  
26 communities in protecting trees will facilitate the informed and science-based guidance required to  
27 manage and maintain forests with high resilience. International cooperation on limiting disease spread and  
28 the provision of early invasive pest or pathogen detection systems are essential.

29

30 **Introduction**

31 Forest tree species are threatened by current changes in their environment caused by climate change,  
32 overexploitation and fragmentation of their habitats, altered disturbance regimes, and by—often  
33 unintended—introductions of invasive species [1]. The expected magnitude and speed of climate change  
34 challenges the biotic and abiotic adaptations of plants, including forest tree species [2,3]. The risk of  
35 reduced fitness due to poor climatic adaptation is likely to be accompanied by an increased risk of serious  
36 biotic stress from already established, spreading or newly introduced pests and pathogens [4–6]. Several

37 examples of the dramatic effects on forest health from sudden outbreaks of emerging pests and pathogens  
38 are well documented: e.g. Dutch elm disease (on *Ulmus*; [7]), sudden oak death, (on *Quercus*, [8]), chestnut  
39 blight (on *Castanea*, [9]), ash dieback (on *Fraxinus excelsior* [10]), white pine blister rust (on *Pinus*, [11]) and  
40 on the pest side emerald ash borer (reviewed by Villari et al. [12] on *Fraxinus*), European gypsy moth on a  
41 variety of North American tree species (reviewed by Davidson et al. [13]), pinewood nematodes on Asian  
42 and European pines [14] and Hemlock wooly adelgids on Eastern and Carolina hemlock [15]. Each of these  
43 diseases has spread rapidly and created economic and ecological loss within a few decades.

44 Tree species co-evolve with many antagonist species that typically do not cause major damages. However,  
45 under certain circumstances, such as when exotic pests and pathogens are introduced to new areas, the  
46 impact on a novel host species can be severe. When an insect pest or pathogen attacks a tree the effect of  
47 the infestation can range from highly damaging and culminating in the host's death, to complete immunity  
48 of the host to any caused damage. Several factors are important for the outcome including: 1) severity of  
49 pathogen pressure, 2) probability of initial establishment or repellence, 3) success of subsequent pathogen  
50 development, and 4) tolerance to tissue invasion. These factors have been recently reviewed by Ennos [16]  
51 and will not be covered in the present article. In this review we will refer to host resistance as “the  
52 collective heritable characteristics by which a plant species, race, clone, or individual may reduce the  
53 probability of successful utilization of that plant as a host by an insect (or pathogen) species, race, biotype  
54 or individual”, defined by Beck [17].

55

#### 56 *A battle on unequal terms?*

57 Trees accumulate biomass and develop a large photosynthetic apparatus as they grow old providing  
58 attractive habitats for insects, fungi and microorganisms. Most tree species host a diverse endophytic  
59 community including mutualists, commensalists, and parasites [18]. Although the actual mechanism of the  
60 individual interactions often remains unknown, a large number of positive associations in relation to plant  
61 defenses has been documented [19]. The presence of endophytes can enhance resistance to pathogens  
62 [19,21] and beneficial microbes in the roots can improve induced resistance through priming [22].  
63 However, during co-evolution endophytes can switch multiple times between a mutualistic and a  
64 pathogenic/parasitic lifestyle on their host [23]. Pests and pathogens thus co-evolve with their host species  
65 mutually exerting and adapting to the others' selection pressures. The co-evolution of defense mechanisms  
66 and counter-defenses can lead to an “arms race” between trees and their antagonists generating ever new  
67 response mechanisms [24]. Alternatively, negative frequency dependent selection and temporally or  
68 spatially varying selection can favor the coexistence and maintenance of genetic variation in defense  
69 mechanisms (“trench-warfare”, [25]). Genetic resistance to pests and pathogens can be due to major  
70 resistance genes (R-genes), or polygenic adaptation [26,27] that determine constitutive and induced  
71 defenses. Resistance mechanisms in forest trees have been recently reviewed by Telford et al.[28].

72 Tree species share life history traits that drive and limit their tempo and mode of evolution (reviewed by  
73 Petit & Hampe [29]). The long generation time of trees provides a challenge for coadaptation since pests  
74 and pathogens usually have much shorter generation times and can evolve favorable traits during the  
75 lifespan of the host. However, trees also host mutualistic endophytes, mainly bacteria and fungi, with  
76 similarly short generation times as their pests and pathogens that can confer resistance [21,30].

77 Furthermore, tree species typically maintain large effective population sizes with high standing genetic  
78 variation [29] that are expected to allow for fast shifts in adaptive allele frequencies [31] if exposed to  
79 strong selection pressure. Newly introduced diseases can cause high mortality and thereby exert strong  
80 selection pressures favoring individual trees with low susceptibility [32]. Individual tree death provides  
81 forest gaps where a dense regeneration can take place followed by selection among a large number of  
82 offspring when mortality is high. In this sense, evolutionary change is expected to occur much faster in  
83 response to severe pests and pathogens compared to altered climatic conditions where maladapted  
84 mature trees can still persist, although not thrive, for a long time [33].

85 Typically, introduced species are only able to become a threat on host species closely related to their native  
86 host [34], while the ability of the new host to cope with novel pests and pathogens depends on its own  
87 evolutionary history. In the worst case scenario, resistance to the new antagonist may be completely  
88 absent, leaving the host species without any potential for adaptation through natural selection on standing  
89 genetic variation. However, exaptations (*sensu* Gould & Vrba [35]) might confer resistance to invasive pests  
90 and pathogens although they co-evolved in response to other selection pressures.

91 The objective of this paper is to review and discuss the adaptive potential of tree species to cope with novel  
92 insect pests and infectious diseases. Based on six case stories, which include some of the most severe  
93 epidemics on trees within the last century, we explore whether genetic variation in resistance was  
94 reported, and the relative abundance and distribution of resistant individuals. Supported by the findings  
95 from the literature, we discuss the basis of the adaptive potential of trees to deal with new diseases.  
96 Finally, we discuss how conservation and landscape management of genetic diversity can support resilient  
97 forests in the next century.

98

## 99 **Resistant trees in natural populations: lessons learned from 20<sup>th</sup> century major outbreaks.**

### 100 *Dieback of European ash*

101 Natural populations of European ash (*Fraxinus excelsior*) have over the last two decades increasingly  
102 suffered damage due to ash dieback (ADB) caused by the invasive pathogenic fungus *Hymenoscyphus*  
103 *fraxineus* (Fig. 1). The introduction history is not fully clarified, but the first reports of disease symptoms  
104 came from Poland in the mid-1990s. It is likely that the pathogen was introduced through movement of *F.*  
105 *mandshurica* plants from Asia to Eastern Europe that led to a host shift to *F. excelsior* [36]. The disease has  
106 subsequently spread rapidly across Europe with first reports of ash dieback symptoms in Scandinavia  
107 around 2001 and most recently in UK in 2012 [32]. The disease causes substantial mortality, especially in  
108 young populations [37] and the abundance of healthy individuals was found to be low in most infected  
109 areas. In Denmark, less than 5% of more than 6,000 trees in two test plantings with offspring from trees of  
110 local origin remained healthy 10 years after planting [32]. Husson et al. [38] found only 8% healthy trees in  
111 a large survey of 2400 trees across 60 forest plots in France. However, the presence of genetic variation in  
112 resistance was confirmed from several countries [39–46] with moderate to high levels of heritability ( $h^2$ )  
113 and genetic coefficient of variation ( $CV_g$ ) for susceptibility, 0.1-0.6 for  $CV_g$ , and 0.3-0.6 for  $h^2$ , respectively  
114 [39–44]. Based on breeding value estimates, the frequency of genotypes with high resistance is expected to  
115 be relatively low (1-5% or less [32]) but it is interesting that genetic variation in resistance was reported in

116 all the studied populations indicating that European ash has the potential to undergo rapid evolution  
117 towards higher levels of resistance through natural or artificial selection. Although 1-5% is a low frequency,  
118 it implies that trees with high levels of resistance are expected to be present in almost any native ash forest  
119 and selection in favor of increased resistance is likely to be ongoing. Pliūra et al. [42] found in a provenance  
120 study that ash offspring from trees from Baltic countries were significantly less affected by the disease  
121 compared to offspring from trees from the Western part of the natural distribution area when grown under  
122 the same conditions in Lithuania. Given the disease history, with the Baltic countries being the epicenter for  
123 the first symptoms and high mortality, these observed differences between Western and Eastern  
124 populations could reflect an already realized response to natural selection.

125

### 126 *Ink disease and chestnut blight*

127 American chestnut (*Castanea dentata*) was an important tree species in North East American forests until it  
128 was heavily decimated across its native range during the last century by the combined effect of  
129 *Phytophthora cinnamomi* causing ink disease and *Cryphonectria parasitica* causing chestnut blight.

130 *Phytophthora cinnamomi* was probably introduced to the native range of American chestnut two centuries  
131 ago and *Cr. parasitica* a century later [47]. Therefore, the two pathogens have been present in NE American  
132 ecosystems for a long time. Some chestnut trees have been able to survive by resprouting [9] and a few old  
133 surviving trees were observed to exhibit some degree of resistance. These candidates might be good  
134 candidates for breeding [48,49]. Alexander et al. [50] reported that old healthy trees are rare and difficult  
135 to find, and Hebard [51] expects that very few mature trees (DBH>33cm) have survived in the core of the  
136 distribution area. The variation in health among trees has been found to be influenced by the virulence of  
137 *Cr. parasitica* specific strains, because hypovirulent pathogen strains infected with the RNA virus do not kill  
138 the trees. The interaction is complicated since the susceptibility of the trees seems to depend on the  
139 interaction between the tree genotype, its growing conditions, and the virulence of the *Cr. parasitica* strain  
140 [52]. The chestnut blight is one of the most investigated emerging infectious diseases on trees and  
141 substantial genomic resources have been developed recently. However, information on the frequency of  
142 resistant trees in the natural forests or on levels and presence of additive genetic variation based on  
143 progeny trials is very limited. Quantified estimates of intraspecific variation in disease resistance is probably  
144 lacking due to a focus on hybridization with Asian species to increase resistance since American chestnut  
145 was early recognized as highly susceptible [53]. The apparently very low frequency of mature trees that  
146 have survived the disease in the native habitat and a lack of healthy recruitment from such rare survivors  
147 suggest that the potential is limited at least on a short timescale. Mature trees do still exist and some of  
148 these may have high natural resistance, but estimates of heritability are to our knowledge unknown.  
149 Recent activities have included development of genetically modified clones [54] as a potential option for  
150 increasing resistance, but here the approach is based on genes not already present in the species.

151 Hybridization with Asian chestnut species has also been deployed to obtain resistance towards *Ph.*  
152 *cinnamomi* in Europe. However, natural resistance has been observed recently among pure European  
153 chestnut (*Castanea sativa*) trees. In a European study testing 50 clones of various origins, one natural  
154 European chestnut genotype was as resistant as the hybrid used as a resistant control [55]. A very large  
155 screening across thousands of hectares in Spain led to the identification of 209 *Ca. sativa* trees of which

156 more than 100 were propagated and subjected to thorough screening. Two of these clones were classified  
157 as resistant and three clones at least partly resistant [56]. These two studies were conducted on clones so  
158 additive genetic variation could not be estimated for resistance. However, the existence of rare resistant  
159 genotypes embedded in the large gene pool of chestnut trees that were susceptible to the introduced  
160 pathogen supports the expectation that evolution towards increased resistance over time can take place.  
161 The low abundance of resistant trees in a large area is of concern because genetic bottlenecks and  
162 inbreeding could decrease genetic diversity unless very effective gene flow counteracts the effects of small  
163 population sizes.

164 *Phytophthora cinnamomi* is a pathogen that infests and causes serious damage in a large number of tree  
165 species across families and genera [57]. Frampton et al. [58] observed substantial genetic variation in  
166 susceptibility of the two closely related *Abies equi-trojani* and *Abies bornmuelleriana* species in Turkey,  
167 where *Ph. cinnamomi* is also considered to have been introduced. Based on controlled infections on  
168 offspring from single tree collections covering the native range of the two *Abies* species in Turkey,  
169 moderate to high narrow sense heritabilities (i.e. reflecting additive genetic effects) for resistance of 0.5-0.6  
170 were estimated. The authors also observed variation in the level of susceptibility among populations  
171 revealing an interesting East-West gradient. The background behind this gradient is unknown but made the  
172 authors speculate that genetic variation in resistance to the introduced *Ph. cinnamomi* pathogen could be  
173 due to exaptation due to adaptation to other *Phytophthora* species present in the region [58].

174

#### 175 *Ulmus and DED*

176 The effect of Dutch elm disease (DED) represents another example of a major calamity caused by an  
177 emerging infectious disease. Two major outbreaks caused by the pathogenic fungi *Ophiostoma ulmi* and *O.*  
178 *novo-ulmi* have led to the death of millions of European elm trees during the last century [59]. The density  
179 of large elm trees has severely decreased across Europe but occasional mature trees are still found in the  
180 landscape. Young seedlings of *Ulmus glabra* are commonly observed in at least parts of the natural  
181 distribution area [60]. The second wave of the disease caused by *O. novo-ulmi* created very high mortality  
182 [61] and breeding for resistance towards the new disease soon focused on introducing resistance through  
183 hybridization with Asian *Ulmus* species [62,63]. However, gene conservation strategies, based on an *in situ*  
184 conservation approach, have been developed to utilize the natural populations' ability to respond to the  
185 selection pressure imposed by the pathogen [64]. Also, breeding programs have been initiated to develop  
186 resistant planting material of the pure *Ulmus minor* in Spain. Unfortunately, the breeding efforts were  
187 hampered by the small fraction (0.5%) of the trees showing resistance [65], but, based on selection and  
188 testing at multiple sites, seven *Ulmus minor* clones tolerant to *O. novo-ulmi* were identified and released  
189 for use in reforestation efforts [66]. Venturas et al. [67] reported moderate to high narrow-sense  
190 heritability ( $h^2=0.54$ ) in *Ulmus minor* after controlled inoculations. Although the frequency of trees with  
191 high levels of resistance was low, the presence of genetic variation and moderate to high heritability  
192 suggest that significant evolutionary potential is still present in the natural populations of elms in Europe.  
193 Similar results have been obtained in North America, where selection and testing have identified genotypes  
194 of American elm (*Ulmus americana*) with very low susceptibility [68] and successful breeding programs  
195 have been implemented [49].

196 *The emerald ash borer*

197 The emerald ash borer (EAB, *Agrilus planipennis*) was most likely introduced to North America in the 1990s  
198 [69] but was not recognized as a new forest pest until 2002 [70]. It originates from eastern Asia [71] but has  
199 already colonized ample areas in North America and killed millions of ash trees while it continues to spread  
200 [72]. White (*F. americana*), green (*F. pennsylvanica*), and black (*F. nigra*) ash are widespread, important  
201 forest components in North America and are highly susceptible to EAB [72,73]. The larvae feed on the  
202 phloem and trees usually die 3-4 years after infestation, young trees even earlier [72]. The percentage of  
203 mortality in natural populations can exceed 99% in highly infested stands in Michigan and regeneration is  
204 extremely limited [74]. So far, all North American ash species in contact with EAB seem to be susceptible  
205 [73,75,76] though blue ash (*F. quadrangulata*) to a lower degree [77]. Asian ash species, especially *F.*  
206 *mandchurika* show distinct, induced and constitutive phloem chemistry and appear more resistant to EAB,  
207 mainly because female EAB avoid healthy trees for oviposition (reviewed by Villari et al. [12]). However, in  
208 North America so far only a few genotypes per species have been tested for resistance to EAB in studies  
209 targeting interspecific variation [73,75,76] and an extensive screening of more genotypes for intraspecific  
210 variation is essential in the future [12].

211

212 Surviving ash trees in heavily infested natural stands exist - although they are rare - (< 0.1%), [74], and  
213 these are likely promising candidates that should be tested for resistance in controlled conditions [78] and  
214 eventually used as resource for resistance in breeding programs [12]. A first bioassay study reported  
215 different mechanisms of resistance in these “lingering” ashes for *F. pennsylvanica*, and a breeding program  
216 to increase resistance in this species based on these trees has been implemented [79].

217

218 Recently, EAB has also been reported to cause damage in ash trees (*Fraxinus* spp.) in urban areas in  
219 Moscow, Russia [80]. In forests south of Moscow, widespread trees of European ash (*F. excelsior*) have also  
220 been infested and suffer dieback although they seem to be less susceptible than North American species  
221 [81]. Research evaluating intraspecific resistance of the three European ash species to EAB is pressing since  
222 the beetle is most likely to spread in Europe [81].

223

224 *Hemlock woolly adelgids*

225 The hemlock woolly adelgid (HWA, *Adelges tsugae*) was introduced to North America in the 1950s from  
226 southern Japan [82]. Over the last few decades the species has invaded vast areas in North America where  
227 it caused extensive mortality of eastern (*Tsuga Canadensis*, [83]) and Carolina hemlock (*T. caroliniana*,  
228 [84]). The damage in northeastern US has been severe with adelgid-induced mortality exceeding 95% and  
229 50–75% defoliation in surviving trees [85,86]. Attacked trees stop their growth, drop attacked needles and  
230 usually die 4–10 years after infestation [87]. Natural regeneration after HWA infestation is rare because  
231 affected trees do not produce seeds and are unable to re-sprout [85]. Forest management employing  
232 biological control agents such as *Sasajiscymnus tsugae* or *Laricobius nigrinus* have reduced HWA density  
233 only locally [88] and preventive salvage logging has additionally aggravated the impact [89]. In many  
234 regions, previously hemlock-dominated forests underwent a severe change in species composition after  
235 HWA attack [89,90]. In contrast Asian and even western North American hemlock species seem to be  
236 resistant to HWA [91]. Not surprisingly mitochondrial DNA studies revealed a long co-evolutionary history



237 between HWA and hemlock species in western North America and Asia [82]. Recently, few individual  
238 eastern hemlock trees with resistance to HWA were also found [92,93]. Nutritional foliar chemistry [94] as  
239 well as terpenoid abundance [95] might be involved in this lower susceptibility. The future impact of HWA  
240 is expected to be exacerbated by climate change since warmer winters are expected to enable an  
241 unimpeded spread of this pest also in the northern distribution range of eastern American hemlock species  
242 [96]. Knowledge about the abundance and distribution of resistant trees and the narrow sense heritability  
243 of the trait is to our knowledge so far lacking.

244

#### 245 *Pinewood nematode*

246 *Bursaphelenchus xylophilus*, the pinewood nematode (PWN) causes the pine wilt disease and is a serious  
247 threat especially but not limited to pine populations. The species is native to North America but was  
248 introduced to Asia in the early 20<sup>th</sup> century where it caused a severe dieback in pine populations [97]. In the  
249 1980s the species spread from Japan to China and Korea (reviewed by Zhao et al. [98]) and at the end of  
250 the 1990s it was brought to Portugal [99]. The Portuguese government implemented the National  
251 Eradication Program for the Pinewood Nematode (PROLUNP), which aimed to log all symptomatic trees in  
252 order to avoid further spread of the disease [100]. The program was carried out in a small affected area and  
253 a demarcation area surrounding it. When newly infested trees were detected in the demarcation area the  
254 limits were redefined, and a clear-cut corridor (3 km wide), free of all tree species that could potentially  
255 host PWN, was prepared. Despite these efforts PWN spread quickly in *Pinus pinaster* forests causing  
256 sudden wilt and tree death [101]. The disease is mainly spread by the movement of forest products [101]  
257 but in nature it is also dispersed by its vector species, a beetle of the genus *Monochamus* [102]. Apart from  
258 logging of infested trees, forest management against the pine wood nematode in Asia includes large scale  
259 insecticide spraying from helicopters or planes to prevent the spread of the vector species [14].

260

261 Infested trees usually die 40 days after infestation [103]. Some resistant trees have been observed and  
262 studied in the usually susceptible *Pinus thunbergii* and *Pinus densiflora* [104,105]. In the 1970s, breeding  
263 programs for resistance were initiated based on resistant cultivars, and seedlings from the program have  
264 been used for reforestation since the 1990s [106]. European pine species show interspecific differences in  
265 susceptibility to PWD with *Pi. pinaster* being the most susceptible and *Pinus pinea* the most resistant  
266 species [107]. However, Zas et al. [108] reported intraspecific variation in resistance to PWN in *Pi. pinaster*  
267 at the provenance level. These findings suggest that genetic variation might be present and more genetic  
268 studies targeting resistance to PWN are needed. The expression of the disease is related to temperature  
269 [109], because the nematodes can be present in trees without causing symptoms when summer  
270 temperatures are low. With global warming the disease might spread further north in Europe where *Pinus*  
271 *sylvestris* dominates in the extensive Boreal forests, and is considered a very susceptible host [110].

272

### 273 **The origin of genetic variation in resistance towards new emerging pests and pathogens**

#### 274 *Where does the variation in resistance come from?*

275 Phenological mismatch between the pathogen and its new host may result in disease escape of host  
276 genotypes in the extreme ends of the natural variation in growth rhythm. In the case of ash dieback,  
277 McKinney et al. [39] observed a strong genetic correlation ( $rg > 0.7$ ) between resistance and early autumn

278 leaf coloration. Since the fungus infects the host through the leaves during summer, early leaf senescence  
279 might increase the probability of disease escape [39]. A correlation between phenology and susceptibility  
280 was also reported for *Ulmus* species affected by DED. Here, early flushing may indicate disease avoidance  
281 due to a phenological/physiological mismatch with the occurrence of the pathogen vector (species of the  
282 genus *Scolytus* which feed and breed under the bark) [111,112]. Furthermore, smaller and narrower vessels  
283 seemed to limit pathogen growth [67] and standing variation in these anatomical traits may, therefore,  
284 explain part of the variation in susceptibility. In sudden oak death canker disease, the pathogen  
285 *Phytophthora ramorum* sporulates early in the year and requires active host cambial tissue to successfully  
286 infect *Quercus* sp. Therefore, late flushing host trees can escape the disease [113]. Genetic variation in  
287 phenology is maintained in natural populations when selection pressures shift between years, likely due to  
288 annual variation in occurrence of spring or autumn frosts. This variation can serve as buffer against damage  
289 from newly introduced pathogens or pests.

290 Most plant species have experienced and co-evolved with a large number of interacting organisms during  
291 their long evolutionary history and range shifts. Therefore, genetic variation in host resistance might have  
292 arisen during time slots of exposure to this or similar pests and pathogens. Random genetic drift is a weak  
293 force if effective population sizes are large [114], and genetic variation from previous exposure to similar  
294 pathogens can, therefore, have been maintained at a low frequency in natural populations even in the  
295 absence of selection. The ash die back pathogen *Hymenoscyphus fraxineus* is closely related to a native  
296 European fungus *H. albidus*, which is considered a harmless decomposer of leaves from European ash. It  
297 has been speculated that this relationship may have previously involved a degree of pathogenicity and  
298 adaptive polymorphisms that evolved under previous selection pressures and might, therefore, remain in  
299 the gene pool of the host species [41]. Along the same line, a large population decline in European elms, as  
300 indicated by pollen diagrams from approximately 5,000-6,000 BP, has been proposed to have resulted from  
301 an epidemic spread by *Scolytus* species. This may have been similar to the outbreak of DED in the past  
302 century [115]. It can, therefore, be speculated that such an outbreak may have generated exaptation in the  
303 host.

304 Another important aspect of the evolutionary potential of tree populations to cope with emerging diseases  
305 is their associated endophytic community, mainly fungi and bacteria [4]. Recently, endophyte communities  
306 have been proposed as indicators of tree health [116]. Gennaro et al. [117] found the endophytic  
307 communities on declining oaks infected by oak puzzle disease to be less diverse than those on healthy  
308 trees. *Tubakia dryina* was found more often on diseased trees while *Monochaetia monochaeta* was more  
309 abundant on healthy trees. In contrast, Martin et al. [118] found that *U. minor* genotypes with resistance  
310 against *O. novo-ulmi* (DED) had lower frequency and diversity of fungal endophytes in the xylem than  
311 susceptible *U. minor* genotypes. However, in laboratory conditions, Díaz et al. [119] demonstrated that an  
312 isolate of *Trichoderma atroviride* extracted from elm trees inhibits growth of *O. novo-ulmi* and was,  
313 therefore, proposed to confer resistance to DED. In *Populus* several endophytic species seemed to  
314 contribute to quantitative resistance to *Melampsora* rust [20]. The most commonly reported role of  
315 endophytes is a strong induced resistance response in hosts due to previous contact with an endophyte.  
316 For example, *Pinus monticola* seedlings were more resistant to white pine blister rust if they had been  
317 previously exposed to endophytes [30]. Arnold et al. [21] also showed that inoculation of endophyte-free  
318 leaves of *Theobroma cacao* with endophytes from naturally infected, asymptomatic trees could reduce leaf  
319 mortality of seedlings exposed to *Phytophthora* sp. The increased defense was primarily localized in the

320 endophyte-infected tissues. The use of endophytes as biological control agents to manage forest diseases  
321 has been recently discussed [120]. However, the community composition and role of endophytes in tree  
322 disease resistance, especially under natural conditions, remains poorly understood and harbors a promising  
323 field of research opportunities.

324

### 325 **Implication for forest management and conservation**

326 Most emerging infectious diseases and destructive insects are caused by accidental introductions [115].  
327 Therefore, obvious precautions include the limitation, or diligent control, of long distance transfer of plant  
328 material and products that can serve as vectors for unintended introduction of insects, fungi or other  
329 microorganisms (see e.g. Montesclaros declaration [121]). Moreover, the development of international co-  
330 operation for disease management is essential [122]. The probability of a successful host shift from an  
331 introduced species is low, but most cases of successful establishment on a new host leads to dramatic and  
332 largely negative consequences.

333 *Do not underestimate the evolutionary potential of tree species but reduce the risk of genetic bottlenecks*

334 This review of six major emerging diseases from the last century highlights the evolutionary potential of  
335 natural tree populations to respond to completely new pathogenic species. A common picture from these  
336 study cases is that the number of individual trees has been dramatically and rapidly reduced following the  
337 emergence of the new disease, but the species were not eradicated. Survival can be due to either disease  
338 escape or to different resistance mechanisms that are at least partly under genetic control and harbor  
339 moderate-to-high narrow sense heritability. This low abundance of unaffected trees, which are often  
340 scattered over large areas, can lead to severe genetic bottlenecks. The limited access to pollen from  
341 conspecific individuals may result in increased self-pollinations, increased relatedness among offspring in a  
342 given area, and decreased intraspecific genetic diversity. However, Nielsen & Kjær [123] studied surviving,  
343 scattered and solitary wind pollinated elm trees in the Danish landscape after the DED outbreak and found  
344 no genetic effects in relation to the lowered tree density. The offspring from these surviving trees were  
345 outcrossed, genetically diverse, and progeny from the same mother tree had been sired mostly by several  
346 different pollen donors. Long pollination distances have also been reported by Bacles & Ennos [124] in the  
347 wind pollinated ash (*F. excelsior*) in a fragmented landscape prior to ash dieback. Wind pollinated conifers  
348 are also expected to maintain very large effective population sizes through pollen flow over long distances.

349 Nevertheless, the potential negative effects of forest fragmentation should be taken seriously [125]. Since  
350 trees with sufficient genetic resistance may be < 1% in natural populations, the risk of decreased fitness  
351 due to inbreeding depression is a serious concern, especially in insect pollinated trees and low abundance  
352 species growing in mixed forests. This can develop into a negative feed-back loop, if loss of vigor leads to  
353 replacement by other species, which again reduces the effective population size and limits seed dispersed  
354 for next generation recruits. Silviculture in support of these endangered species may, therefore, be  
355 important. Unmanaged naturalized forests are expected to be less affected than forest plantations and  
356 resilience in these forests can be further supported through the maintenance of large population sizes, and  
357 should involve long-term land use planning to ensure continuous forest patches where gene flow can take  
358 place at the landscape level.

359

360 *Resistant, but also genetically diverse seed sources for reforestation*

361 Maintenance of substantial genetic variation in seed destined for planting programs of trees in long  
362 rotation will support the adaptive potential of planted forests and ensure a low *a priori* relatedness among  
363 planted trees [126]. On the contrary, high genetic homogeneity has been suggested to facilitate a fast  
364 disease spread, e.g. Gil et al. [127] speculated that the spread of DED on English elm in Britain was  
365 promoted by scattered plantings and vegetative reproduction of a single clone during centuries. Breeding  
366 programs must identify and test a substantial number of surviving and healthy trees based on large-scale  
367 screening to be an effective tool for the development of genetically diverse and disease resistant seed  
368 sources for forest restoration [49]. For example, the Danish restoration program for ash forests includes the  
369 testing of more than 200 trees selected among thousands of trees across the Danish landscape and similar  
370 programs are being initiated in other countries [32]. The public is very concerned about forest health, and  
371 involving citizen science is an interesting option for the identification and continuous monitoring of  
372 surviving trees [128,129]. This approach can multiply tenfold the identified number of healthy trees, as it is  
373 time consuming and expensive to find rare healthy trees scattered across large forest areas without the  
374 help of local people. Besides finding trees to be included in breeding programs for restoration, these  
375 observations from citizen science can improve data quality on disease spread. Also, local or regional efforts  
376 to protect these surviving trees can be implemented more broadly, efficiently and effectively. Volunteers  
377 have already been involved in monitoring tree health in forests suffering from ash dieback in the UK  
378 (<http://www.observatree.org.uk/portal/tree-health-citizen-science-projects/>) and sudden oak death in the  
379 US (<http://oakmapper.org/>).

380

381 *Felling of healthy trees in the neighborhood of diseased areas can be counterproductive on a large scale*

382 A classical forest management tool to avoid the spread of new pests and diseases is preemptive and  
383 salvage logging in the neighborhood of an infested forest patch. This strategy is highly relevant at the very  
384 beginning when a newly introduced pest or pathogen is detected for the first time. It should be employed  
385 in initial and locally restricted cases of first disease incidence. However, as soon as several disease centers  
386 are emerging it can become counterproductive due to the removal of high numbers of healthy and some  
387 potentially resistant trees. Since healthy mature forest trees represent commercial value to the forest land  
388 owner, the outbreak of a new infectious disease could lead to the extensive logging of many healthy trees  
389 to minimize the risk of lost revenue due to infections [32]. In this way, a new disease can trigger both  
390 natural mortality that will reduce the density of susceptible trees, and increased harvesting that will further  
391 decrease the density of all trees including the rare resistant ones. In some cases this strategy has been  
392 successful, e.g. Asian long-horned beetles were eradicated in Illinois and Jersey City after an initial  
393 introduction [130]. In other cases preemptive logging has not proven successful, e.g. the spread of PWN in  
394 Portugal 1999-2009 [131] and of the EAB in Canada [132] could not be avoided. However, more research is  
395 needed on this topic since few studies have addressed the effectiveness and impacts of preemptive logging  
396 so far. The usefulness of this management tool is highly case specific and depends e.g. on the mode of  
397 disease spread. Foster et al. [133] also pointed out that preemptive/salvage logging often imposes a bigger  
398 ecosystem impact than the disturbance itself.

400 *Cooperation and early warning systems based on observations in Arboreta*

401 Arboreta and plantings of exotic tree species can inform about potential risks of pests and pathogens  
402 before they are accidentally introduced to other jurisdictions. For example, an arboretum was used to  
403 study interspecific variation in the susceptibility to HWA among *Tsuga* species from different continents  
404 [134]. There are ongoing initiatives such as COST action FP1401 “A global network of nurseries as early  
405 warning system against alien tree pests (Global warning)” [135] or the project “REINFFORCE - REsource  
406 INFrastructure for monitoring and adapting European Atlantic FORests under Changing climate” [136] that  
407 use arboreta to detect possible future biotic threats. Furthermore, experimental plantations of Norway  
408 spruce (*Picea abies*) in North America suffered severe damage from the native white pine weevil (*Pissodes*  
409 *strobi*), an insect pest that naturally co-occurs and feeds on several North American conifer species. Within  
410 these plantations, Norway spruce revealed a degree of susceptibility similar to highly damaged local Sitka  
411 spruce populations. In an Estonian arboretum, Drenkhan et al. [137] screened exotic *Fraxinus* species for  
412 resistance to the ash dieback pathogen and observed signs of infection on *Fraxinus* species native to areas  
413 in North America where the pathogen is not present.

414

415 *Implication for gene conservation programs*

416 Conservation of genetic resources of key forest tree species is recognized as an important part of  
417 sustainable forest management [138], and various guidelines have been developed that typically target  
418 minimum effective population sizes of 50-5,000 [139]. In general, these numbers are derived from ‘the  
419 golden rule of 50-5000’, which remains controversial and hotly debated in conservation literature [140].  
420 However, if an emerging disease creates high mortality leaving only 1% living trees scattered across  
421 populations, a requirement for an effective population size of > 50-5000 in the next generation will  
422 obviously require an initial gene conservation population where 5,000 mature trees represent an absolute  
423 minimum. The effective population size is typically less than half the census number of mature trees in the  
424 landscape due to variation in fecundity, therefore the actual number of mature trees that need to be  
425 conserved is larger than often anticipated [141] Also, since resistance may be related to interactions with  
426 the endophytic society in the trees, *in situ* conservation or *in situ*-like conservation approaches [142] may  
427 have a clear preference to *ex situ* programs, which are mainly concerned with conserving the genetic  
428 variation of the targeted tree species.

429

430 **Conclusion**

431 Emerging exotic pests and pathogens pose a major challenge for future global forests and should be  
432 addressed by international cooperation reducing the risk of new introductions. However, natural  
433 populations often exhibit some level of genetic resistance even to newly introduced species. The surviving  
434 trees may be scattered and limited in number, but the evolutionary potential of host species to cope with  
435 emerging pests and pathogens should not be underestimated. Natural resistance can be facilitated by

436 maintaining genetic diversity in natural forests and by supporting connection between trees and forests to  
437 allow continuous gene flow. It will be a challenge to maintain viable natural population sizes of species that  
438 undergo dramatic mortality caused by an aggressive emerging disease. In managed forests, large-scale  
439 deployment of planting material with low genetic diversity can be extremely risky. Therefore, we suggest  
440 that breeding for resistance should not lead to genetically uniform populations with low resilience and  
441 small effective population sizes. Instead, breeding should involve large-scale screening efforts across  
442 natural populations to ensure that a high number of resistant trees are identified and included in the  
443 testing and breeding activities. Programs for the identification, protection and propagation of surviving  
444 trees will be imperative and collaboration between scientists, forest managers, public authorities and civil  
445 society will be essential. Future research on the genetics of resistance mechanisms, host-pathogen  
446 interactions, exaptation, and the role of endophytes will, hopefully, lead to a better understanding of the  
447 biotic adaptation progress that can guide effective forest management, disease control, resistance breeding  
448 and restoration efforts.

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454

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785

786 **Figure 1** Damages caused by *Hymenoscyphus fraxineus* on *Fraxinus excelsior*: A) Variation in  
787 degree of crown damage among infested trees in a planted stand in Denmark; B) Lesion on a  
788 European ash leaflet after controlled inoculation; C) Fruiting bodies of *H. fraxineus* on leaf rachises  
789 and petioles; D) Lesion on a young stem after controlled inoculation with an infested wood plug.  
790 Photos: Lars N. Hansen, Lene R. Nielsen and Lea Vig McKinney