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Effects of the cascading translocations of larch (*Larix decidua* Mill.) on canker disease due to *Lachnellula willkommii* (R. Hartig) Dennis

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Abstract

Key message Appropriate silvicultural practices combined with the use of resistant Central European provenances can reduce the prevalence of larch canker caused by *Lachnellula willkommii* (R. Hartig) Dennis, a major disease affecting larch plantations in France. However, cascading translocations have resulted in frequent admixture in European larch (*Larix decidua* Mill.) and subsequent certification errors regarding the origin of plant material. Our study highlights the urgent need to improve the certification process of seed orchards.

Context The recent history of European larch has been marked by translocations of plant stock within and beyond its native range. In order to increase stand resistance to larch canker disease, it is strongly recommended by French authorities to plant Central European provenances. However, a recent disease outbreak questioned the resistance of Central European provenances and the origin of the forest material used in these plantations.

Aims Our goal was to assess the effects of cascading translocations and mixing between larch gene pools on genetic composition of plantations and on their vulnerability to the disease.

Methods In the outbreak area, we checked the genetic origin of the trees and we estimated the percentage of Central European trees, disease prevalence and severity.

Results Intra-site genetic diversity was high. Genetic composition did not match with geographic origin certificates. A high proportion of trees could not be assigned to either the Alpine or Central European gene pools. These admixed trees were as resistant as Central European trees. Geographic origin turned out to be one of the main drivers of canker prevalence, along with abiotic factors.

Conclusion We need a precise knowledge of the origin of material used in seed orchards to mitigate canker disease and adapt forests through assisted migration.

Keywords *Larix decidua*; *Lachnellula willkommii*, Assisted migration, Admixture, Disease prevalence

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This article is part of the topical collection on Assisted Migration and Forest Seed Transfer.

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1 Introduction

Long before global change became a widespread concern, foresters have relied on translocations of tree species or populations. To cope with local seed shortages for afforestation, they have used non-autochthonous reproductive material since at least the seventeenth century (Bradshaw 2004; Jansen and Geburek 2016; Geburek and Myking 2018; Jansen et al. 2017). For instance, several hundreds of tons of conifer seeds have been imported in Scandinavian countries for reforestation purposes since the nineteenth century (Myking et al. 2016). Translocations then became management options to improve forest production using reproductive material with presumed superior genetic quality and better growth characteristics compared with local material. Other incentives for these transfers include overcoming local environmental constraints and preventing local extinctions. More recently, assisted migration was proposed as a solution to mitigate the negative effect of climate change on tree growth, by moving biological units from their current range to other areas where they are expected to perform well (Richardson et al. 2009).

Species translocations and assisted migrations have raised controversies because of the associated ecological and genetic risks (Laikre et al. 2010; Morais and Reichard 2017; Thomas et al 2014). Originally designed for the conservation and rescue of endangered species, assisted tree migration is now advocated as a means to preserve forest productivity (Pedlar et al. 2012). In such a context, the targeted species are commercially valuable forest tree species, and translocations are performed mostly within species' natural distribution range (Pedlar et al. 2012). Such translocations are less risky than outside-range translocations in terms of potential invasiveness of the introduced populations but gene exchanges between introduced and native populations can arise as a result (Petit 2004). Gene flow and intraspecific exotic introgression can break down local adaptation (Unger et al. 2016) or cause genetic homogenization or geneticcontamination (Millar et al. 2012; Ramírez-Valiente and Robledo-Arnuncio 2014; Steinitz et al. 2012). Moreover, pests and pathogens can be introduced along with their host trees during plantations. This can either extend the range of invasive bioagressors, eventually causing extinction of naïve host-tree species and populations that have not coevolved with these enemies, or increase the genetic diversity of local populations (Desprez-Loustau et al. 2007; Simler et al. 2019). Thus, within-range translocations of tree species can have long-term evolutionary consequences. Predicting their effects on native conspecific populations should be a priority. In this context, tracking the origin of populations of tree species and studying historical transfers should be particularly informative.

European larch (*Larix decidua* Mill.) is a forest tree species whose recent history was heavily marked by past translocations. Its fragmented natural distribution range includes the Alps, the Carpathians and Sudetes (three high altitude areas in Central Europe) and the Polish lowlands. Populations from the Alps are genetically different from those of the Carpathians, Sudetes and Polish lowlands, collectively referred to as CE (Central Europe) populations (Wagner et al. 2015a). European larch is famous for limited sensitivity to edaphic constraints, its resistance to cold, snow, and wind, its fast juvenile growth, and its high wood quality (Pâques et al. 2013). The interest for larch in Europe dates back to the sixteenth century when the first plantations of larch were initiated but it culminated in the nineteenth century (Rubner 1953, cited by Wagner et al. 2015a). Jansen and Geburek (2016) have summarized current knowledge on the history of the consequences of this reforestation program on international translocations of larch populations. Within-range translocations were frequent, in particular in the Sudeten area (at the border between Czech Republic and Poland). In this area, a large number of larch reforestation projects were undertaken either with local material or with alpine provenances. They aimed at mitigating the numerous disturbances consecutive to wars and overexploitation experienced by indigenous larch populations since the eighteenth century. Multiple within-range translocations, especially into CE from the Alps, resulted in admixture with local material (Wagner et al. 2015a). In addition, due to the good performance of the first larch plantations, there has been enthusiastic support for new plantations, a phenomenon called 'Lärchenmanie' or 'Lärchepassion', resulting in additional translocations (Pardé 1957; Wagner et al. 2015a). The new plantations in Germany, Northern Europe, and Britany were often performed on unsuitable soils or with seed sources poorly adapted to the local ecological conditions (Jansen and Geburek 2016; Weisgerber and Sindelar 1992). Inthe mid-nineteenth century, a disease characterized by sunken cankers and severe canopy dieback emerged in most of the afforested larch stands located in the plains (Hartig 1880; Yde-Andersen 1979). It was associated with an ascomycete fungus, *Lachnellula willkommii* (R. Hartig) Dennis, which Manners (1957) identified as the main cause of the sunken cankers (Fig. 1). Several authors suggested that this fungus was responsible for the failure of larch cultivation in the plains of Northern and Western Europe, i.e., outside the natural distribution

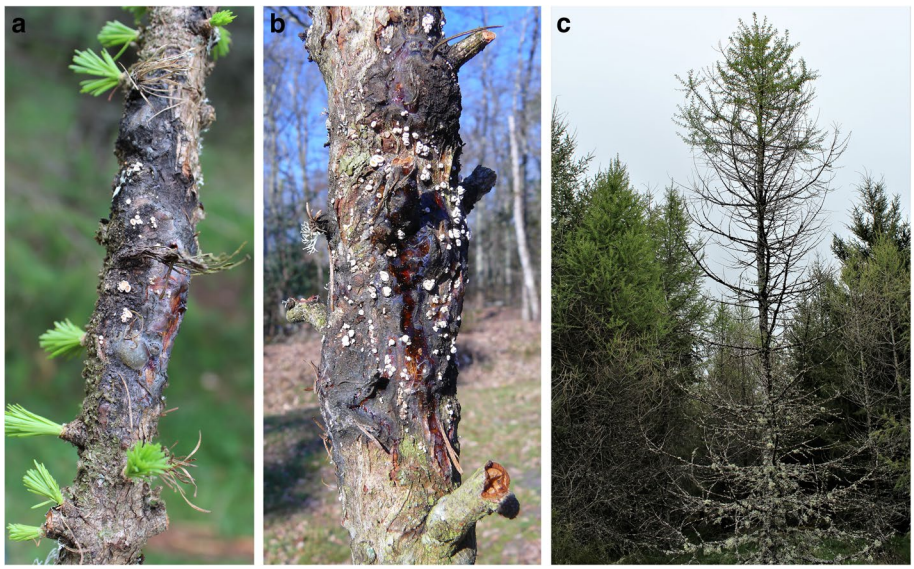

Fig. 1 Cankers caused by *Lachnellula willkommii* on European larch branch (**A**) and trunk (**B**), and symptomatic larch tree showing between 50 and 95% of symptomatic branches (**C**)

area of larch (Buczacki 1973; Pawsey and Young 1969; Schober 1977). Such failures of introduced tree species are common. For example, eucalyptus pathogens that cause virtually no damage in their area of origin induce severe damage in areas of introduction and can even be a strong limit to outside-range translocations (Burgess and Wingfield 2017). The period during which the plantations of introduced species are free of co-evolved pathogens, not yet introduced, and of indigenous pathogens, not yet adapted to a new host, is called the "honeymoon period" (FAO 2001). According to Hartig (1880), in Germany, the larch honeymoon lasted throughout the first half of the nineteenth century.

A large international cooperation was set up to address the question of these repeated and spectacular failures of outside-range translocations of larch in Europe. During two European research programs, nearly 100 common garden trials involving more than 100 provenances were launched from 1944 to 1958 (Weisgerber and Sindelar 1992). This made it possible to test larch canker resistance across provenances (Pâques et al. 2013). The main finding was that CE larch populations were more resistant than Alpine ones, with Tyrol provenances being less susceptible than those of the center or south-west of the Alps (Bürgi 1990; Pawsey and Yooung 1969; Schober 1977; Weisgerber and Sindelar 1992). For other traits such as taper, crown shape, branching, wood properties, resistance to insects, frost hardiness, and drought tolerance, provenances from CE turned out to be more promising than Alpine ones at low elevation (Pâques et al. 2013). Therefore, provenances from Eastern Europe and in particular from the Sudetes were recommended as seed sources for reforestation in plains and have been increasingly planted in Europe since the 1970s (Weisgerber and Sindelar 1992). For instance, in France, the Ministry in charge of forest policy has promoted larch for coniferous stands reconstitutions (CTGREF 1977; Ferrand 1986). For instance, in the Massif Central, following a devastating storm, a major larch reforestation campaign took place in 1982. Although CE provenances were used, in agreement with the official recommendations, an outbreak of larch canker has been observed in this region since 2010. This outbreak calls into question the origin of the forest material used for planting and the resistance of CE provenances. In the natural distribution range of larch in CE, translocations of Alpine populations have been frequent. By sampling natural populations of European larch throughout the species range, Wagner et al. (2015a) showed that the main consequence of past translocations was the admixture of local and introduced larch material.

Our objective was to assess the consequences of genetic admixture in relation with the risk of larch canker disease. We hypothesized that (1) due to successive translocations, the actual origin of larch seedlings was distinct from the one traced in the certification documents and thus, sometimes, from the one

recommended for reforestation, and (2) the prevalence of canker disease in plantations outside the natural larch zone in France is driven by the genetic composition of these plantations. To test these hypotheses, we surveyed a large set of larch stands in the outbreak area. We characterized the genetic composition of these stands and assessed disease prevalence and severity. We also provide the first evaluation of field susceptibility of interpopulation larch hybrids to larch canker. Finally, we discuss the consequences of species translocations for forest management in general and for disease management in particular.

2 Material and methods

2.1 Study sites

In 2011–2012, we selected 53 plantations in the Massif Central (stand elevation varied between 547 and 1552 m, Table 1, Fig. 2). They were 6 to 25 years-old and distant from at least two kilometers from each other. In these young plantations, thinning less than 5 years old was only observed in four plots (#12619, 12645, 12718, 12723). We included in this study as controls four additional stands corresponding to older plantations (48 to 126 years old) unaffected by the 1982 storm. We collected site and plantations descriptors (age, altitude, height of dominant trees, density, topography, site humidity; Table 4 in Appendix). We scored site atmospheric humidity indirectly using data on tree density, site topography and environment. We attributed a score of zero to all sites where air circulation between trees is sufficient to impede accumulation of humidity (e.g., tops of slope, sites not surrounded by older or higher plantations, absence of under-growth species, low tree density). Otherwise, we attributed a score of one to the stands. In each larch stand, official documents providing information on plant provenance and plantation date were collected and analyzed. We kept three classes for the following analyses: certified CE origin, certified Alpine (A) origin, and unknown origin (NA) (when no certification was available).

2.2 Assessment of larch canker prevalence and severity in plantations

The first signs of larch canker disease are bark necrosis and depressions, which develop as sunken cankers first in fine branches and then in stems. These cankers are associated with resin exudation, bark cracks and needles shriveling. Ultimate signs are needle, branch or plant mortality. Apothecia of *L. willkommii* are frequently observed in cankers (Hartig 1880; Yde-Andersen 1979). However, confusion with *Lachnellula occidentalis* (G.G. Hahn and Ayers) Dharne, a saprophytic species difficult to distinguish from *L. willkommii*, is frequent (Cech 2013). Molecular markers have recently been developed, making it possible to distinguish apothecia from the different *Lachnellula* species. We carried out fungal isolations and extractions of DNA from apothecia, sampled within the cankers developing on the branches of the diseased trees, to confirm the identity of the causal organism (N. Feau and C. Robin, unpublished results). In each stand, we recorded symptoms attributable to larch canker disease on 50 trees, distributed in five clusters, positioned every 50 m on two parallel transects distant by 50 m. We scored trees showing symptoms on branches or trunk as infected, the other trees as healthy. We calculated a disease severity score for each tree as described in Table 2. It varied from zero (healthy tree) to one (branches and stem heavily impacted by cankers). We then calculated disease prevalence (expressed as the proportion of infected vs. healthy trees) and mean disease severity for each stand (Table 1).

2.3 Larch tissue sampling and DNA isolation

We performed genetic analyses to confirm or identify the origin of trees in the larch plantations. In 2012 and 2013, we collected in each site phloem and needles from at least five trees randomly chosen within the stand. To investigate the relationship between genetic origin and canker susceptibility at the tree level, we carried out an additional sampling in 2015. For this purpose, we selected five stands (stands #11897, 11985, 12723, G, and P), according to the following criteria: detection of genetic admixture during the first campaign, altitude (from 1074 to 1213 m), age (from 14 to 19 years) and humidity score (null in all except one). We assessed disease severity and collected phloem samples for DNA extraction in 140 trees randomly chosen in these five stands. During both campaigns, we stored larch needles and phloem in plastic bags with silica gel and brought them back to the laboratory where we performed genetic analyses. Sampling technique and DNA isolation were as described by Wagner et al. (2012).

2.4 Genetic analyses and assignment to Alpine or Central European gene pools

We genotyped all sampled individuals at 13 highly variable microsatellite loci combined in two multiplexes, as described in Wagner et al. (2012). To assign individuals to genetic clusters, we used the software Structure (Pritchard et al. 2000) and 793 larch reference samples assigned with very high confidence score (Wagner et al. 2015a). Using Structure v. 2.3.4 and the analysis parameters described in Wagner et al. (2015a), we ran 10 independent Structure runs for

Table 1 Studied larch plantations: location, origin of forest material and larch canker disease assessments

Site	Locality	Long / Lat (Lambert)	Origin[a]	Disease prevalence	Disease severity
11787	Saint-Jean-Lachalm	713100 / 1993960	A	0.98	0.3
11788	Saint-Jean-Lachalm	712645 / 1994410	CE	0.34	0.04
11789	Saint-Jean-Lachalm	713313 / 1994360	NA	0.8	0.19
11826	La Chaise-Dieu	707536 / 2036000	CE	0.04	0
11827	Saint-Jean-D'aubrigoux	713169 / 2040160	CE	0.24	0.01
11828	Saint-Jean-Lachalm	710577 / 1996950	CE	0	0
11829	Saint-Jean-Lachalm	711904 / 1995770	CE	0.24	0.02
11830	Le Bouchet-Saint-Nicolas	713622 / 1990680	CE	0.02	0
11831	Cayres	714988 / 1991310	A	0.94	0.19
11832	Les Estables	744479 / 1991590	A	1	0.58
11833	Les Estables	745008 / 1992190	NA	0.06	0
11834	Saint-Front	744977 / 1992870	CE	0.02	0
11835	Saint-Front	744722 / 1993080	CE	0.64	0.15
11836	Les Estables	742204 / 1990530	NA	0	0
11837	Freycenet-La-Cuche	740649 / 1990090	NA	0	0
11880	Les Estables	742627 / 1992950	CE	0.02	0
11894	Araules	742736 / 2008040	A	0.36	0.05
11895	Araules	745781 / 2007840	CE	0.62	0.1
11897[b]	Mazet-Saint-Voy	751979 / 2006110	CE	0.32	0.03
11979	Pinols	679380 / 2004470	CE	0	0
11980	Pinols	680800 / 2002700	CE	0	0
11981	Grezes	689900 / 1992320	CE	0	0
11982	Grezes	686590 / 1989770	CE	0.04	0
11983	Les Vastres	753070 / 1999850	CE	0.36	0.03
11985[b]	Les Vastres	750280 / 1998710	CE	0.5	0.05
11987	Chaudeyrolles	748030 / 1997150	CE (SO)	0.06	0
12572	La Chaulme	727061 / 2051880	CE (SO)	0	0
12617	Saint-Antheme	723116 / 2064370	CE (SO)	0	0
12619	Le Claux	630159 / 2017600	NA	0	0
12622	Vieillespesse	663278 / 2012360	NA	0.02	0
12623	Paulhac	657291 / 1984750	NA	0.7	0.11
12624	St-Rémy-De-Chaudes-Aigues	654187 / 1975490	CE	0.06	0
12625	Cros-De-Montvert	584647 / 2007540	NA	0	0
12626	Gourdièges	643003 / 1995057	CE	0.02	0
12627	Allanche	648137 / 2027970	CE	0.96	0.28
12644	Allanche	645564 / 2026290	CE	0	0
12645	Oradour	646448 / 1995500	CE	0.02	0
12646	Anterrieux	655202 / 1980090	NA	0.04	0.01
12647	Albepierre-Bredons	639346 / 2007560	CE (SO)	0	0
12672	Valette	621711 / 2026090	CE (SO)	0	0
12673	Le Falgoux	621973 / 2016200	NA	0.16	0.01
12718	Albepierre-Bredons	636644 / 2007260	NA	0.9	0.2
12719	Vabres	669872 / 2004830	NA	0.16	0.01
12720	Narnhac	634398 / 1993790	A	0.96	0.47
12721	Millevaches	579429 / 2072900	CE	0	0
12722	Saint-Genes-Champanelle	651581 / 2083670	NA	0	0
12723[b]	Cezens	643587 / 1996990	CE	0.28	0.01
12726	Soulages	673001 / 2010490	NA	0	0
12729	Charbonnières-Les-Varennes	649970 / 2097650	CE	0	0

Table 1 (continued)

Site	Locality	Long / Lat (Lambert)	Origin[a]	Disease prevalence	Disease severity
12730	Ceyssat	647410 / 2083730	CE	0	0
12731	Orcines	651540 / 2088750	NA	0.2	0.01
12732	Saint-Ours	647250 / 2095330	NA	0	0
12733	Royat	654490 / 2084660	NA	0	0
13551	Ceyssat	644363 / 2087140	NA	1	0.8
13552	Mazaye	642002 / 2086440	NA	0.98	0.51
G	Orcival	639388 / 2070608	NA	0.78	0.16
P	Paulhac	644022 / 2000640	NA	1	0.56

[a] Origin certified *A* Alps, *CE* origin certified: central Europe, *SO* seed orchard, *NA* certified origin non available; [b]sites sampled in 2015 for the second campaign

$K=7$, with a burn-in of 200,000 followed by 1,000,000 iterations for the admixture model. We assigned the individuals to the Alpine gene pool (clusters 1 to 4 from Wagner et al. 2015a) or to the CE pool (clusters 5–7 from Wagner et al. 2015a), using an assignment threshold of 0.875 (Robin et al. 2023). We considered the individuals that we could not assign to one of these gene pools using this threshold as admixed. We then attributed a genetic susceptibility score to each larch individual. We gave a score of 0.95 (high susceptibility) to the genotypes of the Alpine gene pool and a score of 0.05 (low susceptibility) to the genotypes of the CE gene pool. For admixed genotypes, we tested three hypotheses: (i) admixed genotypes are equally resistant to the disease than CE genotypes (score of 0.05); (ii) admixed genotypes exhibit an intermediate susceptibility (score of 0.5); (iii) admixed genotypes are equally susceptible to the disease than Alpine individuals (score of 0.95). For each stand, we computed the genetic stand susceptibility (GSS) as the mean value of the genetic scores. The three hypotheses tested for admixed trees lead to three GSS values: GSS1, GSS2, and GSS3.

2.5 Statistical analyses

We tested the consistency between the certified origin of trees and their genetic assignation using chi^2 tests using the proportion of trees of each certified population as the theoretical proportion. This test was limited to the 36 stands for which we had retrieved official certification documents. For the whole sample of stands, we tested, using chi^2 test, whether the genetic composition of stands varied with the type of certification (Alpine origin, CE origin or any certification). We tested the effects of stand age, atmospheric humidity, growth index, percentage of trees assigned to the Alpine gene pool (PAP) and genetic stand susceptibility (GSS1, 2 or 3) on disease prevalence using generalized linear models (GLM). We used stand age as a proxy for stand structure (i.e., tree density and basal area). A significant linear relationship was found between the measured height (H) and age ($H=1.647+0.583\times age$, $F=68.0$; $P<0.0001$ and $R^2=0.58$). Thus, the stand growth index (GI) was calculated as $GI=(H\text{-}H_a)/H_a$, with H_a corresponding to the height adjusted to the "age effect" (Table 4 in Appendix). We considered disease prevalence as a binomial response variable. Preliminary analyses of GSS2 detected over-dispersion in prevalence data, which we accounted for by specifying a quasi-binomial error structure. We first checked for independence of explanatory variables. Stand age and humidity were not independent (ANOVA with log-transformed age: $F_{1,49}=10.1$, $P=0.003$): moist plots were older than dry ones (Fig. 7A in Appendix). Likewise, GSS2 was higher in moist stands (ANOVA: $F_{1,49}=4.58$, $P=0.037$, Fig. 7B in Appendix). GSS2 was also higher in older stands ($F_{1,49}=9.7$, $P=0.003$, Fig. 7C in Appendix), although the strength of this relationship was weak ($R^2=0.16$). To avoid spurious interpretations due to collinearity among predictors, we ran models with GSS variables fitted either before, or after, stand humidity. If GSS variables had a significant effect when fitted first, but were non-significant when fitted after humidity, we interpreted it as a confounding effect of humidity. In contrast, when GSS remained significant regardless of when it was fitted, we interpreted the effect as demonstrating a role of genetic composition on disease prevalence. We tested the significance of explanatory variables against F-distribution. In each model, we first fitted stand age used as a covariable. We compared model outputs with the three different GSS variables (GSS1, 2 or 3) with pseudo-R^2 corresponding to the degree of improvement of the fitted model over the null model ($R^2=1-$[Residual deviance/Null deviance]). We carried out all analyses in R (R Core Team 2015).

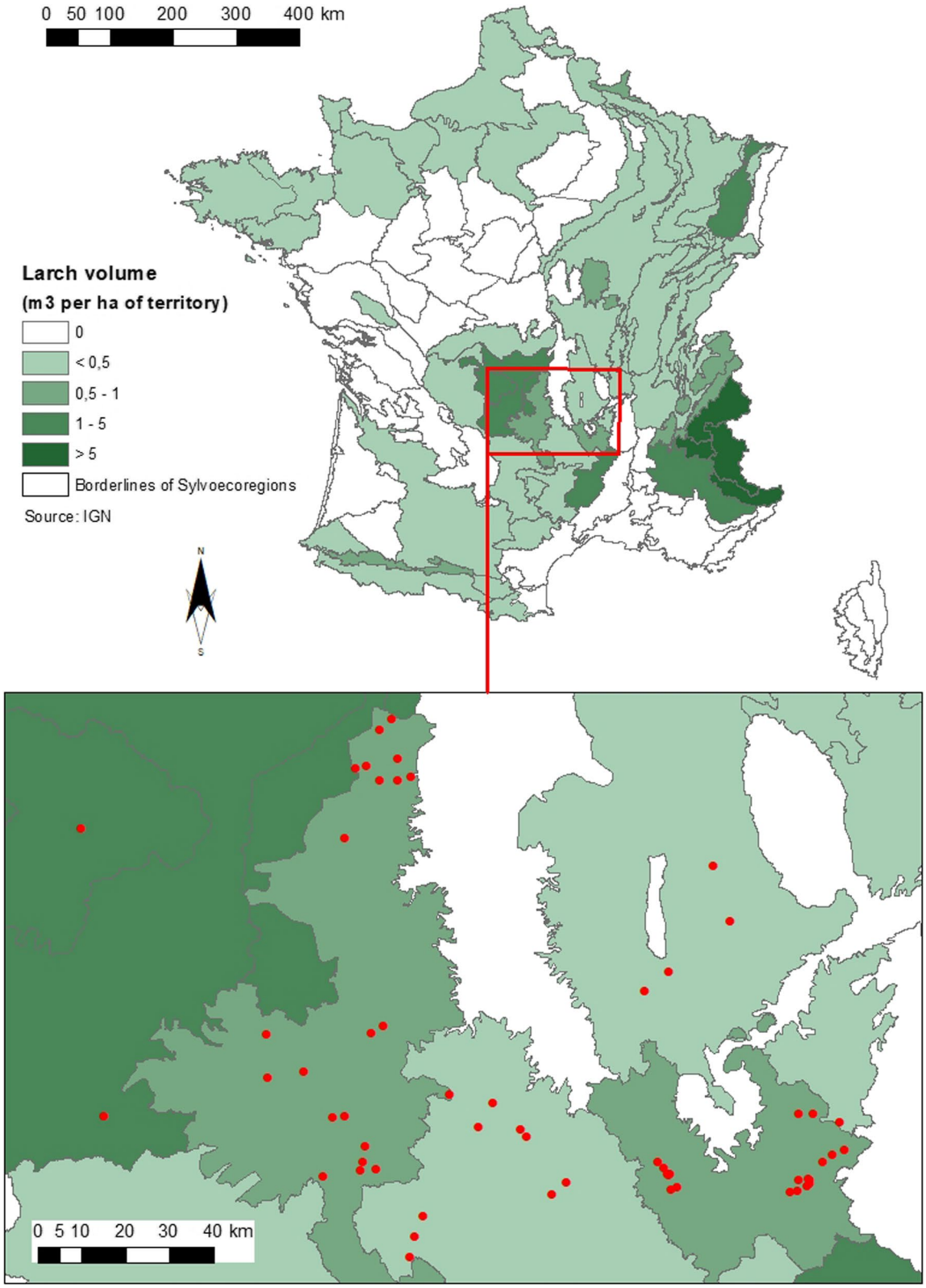

Fig. 2 Location of the *Larix decidua* plantations studied in the Massif Central (France). In the background of the map, the volume of larch wood is shown for each French forest region (i.e. territorial division where similar ecological conditions prevail in terms of forest production)

Table 2 Larch canker disease severity, according to two indicators (the occurrence of characteristic cankers on branches and the occurrence on stem), and the number (and percentage) of trees per type of symptoms scored in the study plots (2679 trees altogether)

Disease severity indicators:	**Disease severity score** **N. trees** ***(% trees)***			**Severity class**
Symptoms on branches:	**Number of stem cankers:**			
	0	**1**	**>1**	
No symptoms on branches	0	0.05	0.10	Null or low
	1939	42	5	
	(*72.4%*)	(*1.6%*)	(*0.2%*)	
Less than 10% of symptomatic branches	0.05	0.10	0.15	
	278	45	15	
	(*10.4%*)	(*1.7%*)	(*1.1%*)	
Between 10 and 50% of symptomatic branches	0.30	0.35	0.40	Moderate
	134	42	30	
	(*5%*)	(*1.6%*)	(*1.1%*)	
Between 50 and 95% of symptomatic branches	0.70	0.75	0.80	High
	61	21	21	
	(*2.3%*)	(*0.8%*)	(*0.8%*)	
More than 95% of symptomatic branches	0.90	0.95	1	Very high
	28	6	12	
	(*1%*)	(*0.2%*)	(*0.4%*)	

3 Results

3.1 Assignment of larch trees to Alpine or CE gene pools (first sampling campaign)

We found official documents certifying the origin of larch plant for 36 stands: for five stands, an Alpine origin was attested, for the other 31 stands a CE origin was found. The precise origin of a CE seed orchard is specified for only five plantations (Table 1). We successfully genotyped 287 larch trees from the 57 studied stands at 13 microsatellite loci [https://entrepot.recherche.data.gouv.fr/privateurl.xhtml?token=39f947d5-afb0-4b14-9773-becdab9f416f]). We assigned 127 trees (42%) to the Alpine gene pool and 84 (29%) to the CE one (Table 5 in Appendix). Most of the stands (44) showed a heterogeneous genetic composition. In 10 stands (four with a CE certified origin, one with an Alpine one, five with an unknown origin), all genotyped trees were assigned to the Alpine pool. In three stands with an unknown origin, all trees were assigned to the CE pool. There was no consistency between the certified origin and the genetic association ($Chi^2=8.19$, $P=0.017$, df=2 for the five stands with an Alpine certification, $Chi^2=142.75$. $P>0.0001$, df=2 for the 31 stands with a CE certification). When all trees were pooled according to their certified origin (Alpine, CE or unknown), the proportion of trees assigned to each gene pool and the proportion of admixed trees differed significantly among the three groups ($Chi^2=18.3$, $P=0.001$, df=4). Only one third of the trees with a CE certified origin were assigned to this gene pool, whereas one third were assigned to the Alpine one and one third were admixed (Fig. 3). In stands without any document, 42% of the trees were assigned to the Alpine gene pool. Whatever the stand in which they were sampled, more than a fifth of the genotyped trees were categorized as admixed individuals.

3.2 Prevalence and severity of larch canker disease in the 57 studied larch stands (first campaign)

Among the 2679 larch trees examined, 72% did not show any symptom (Table 2). The disease severity was low for 14% of the trees, moderate for 8% and high or very high for 4% and 2%, respectively. We found larch disease symptoms in 37 stands (65%). In these stands, prevalence varied from 0.02 to 1.00 (mean ± SE: 0.43 ± 0.06). Mean severity of infected trees (mean ± SE: 0.18 ± 0.03) increased exponentially with prevalence (Fig. 4). Mean severity was low (0.15–0.70) for disease prevalence below 0.70. By contrast, in the seven stands heavily impacted by the disease (with more than 95% of symptomatic trees), mean severity varied between 0.30 and 0.80. *L. willkommii* infection resulted in tree death when the main trunk or the crown were highly infected. We observed such

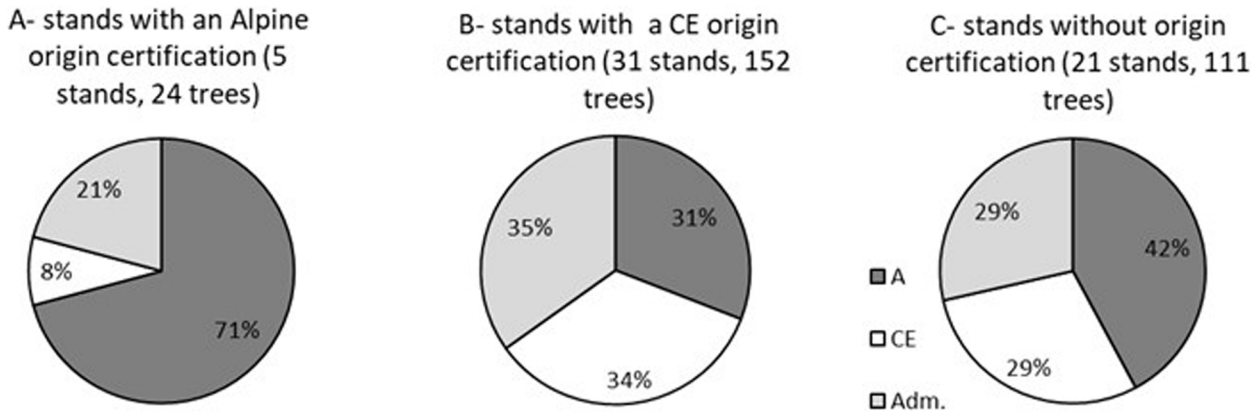

Fig. 3 Distribution of trees in the three gene pools defined by genotype analysis (A: Alpine, CE: Central Europe, Adm: admixed), according to the certification data of the stand in which they were sampled

high rates of infection for 71 dead trees (2.7%), which were all dominant or codominant and located in the most affected stands. Three explanatory variables (stand age, humidity, PAP) had a significant effect on disease prevalence, regardless of whether they were fitted first or last in GLM (Table 3), thus showing that our conclusions are not affected by problems of collinearity among variables. Prevalence was higher in moist than in dry stands and increased both with stand age and with PAP (Fig. 5). The same results were obtained whatever the GSS indices tested, all of which provided very similar outputs (with $R^2 = 0.55$ or 0.56, Table 6 in Appendix). The observations made in the four oldest stands were consistent with those made in young stands. Larch canker prevalence in the two older stands (78 and 126 years old) was 0.70 and 0.90 and the percentage of trees assigned to the Alpine pool was 80% and 100%, respectively. These stands were well aerated, with a low atmospheric humidity. In the two other stands (48 and 51 years old), we found only CE trees and we observed no symptom of larch canker disease.

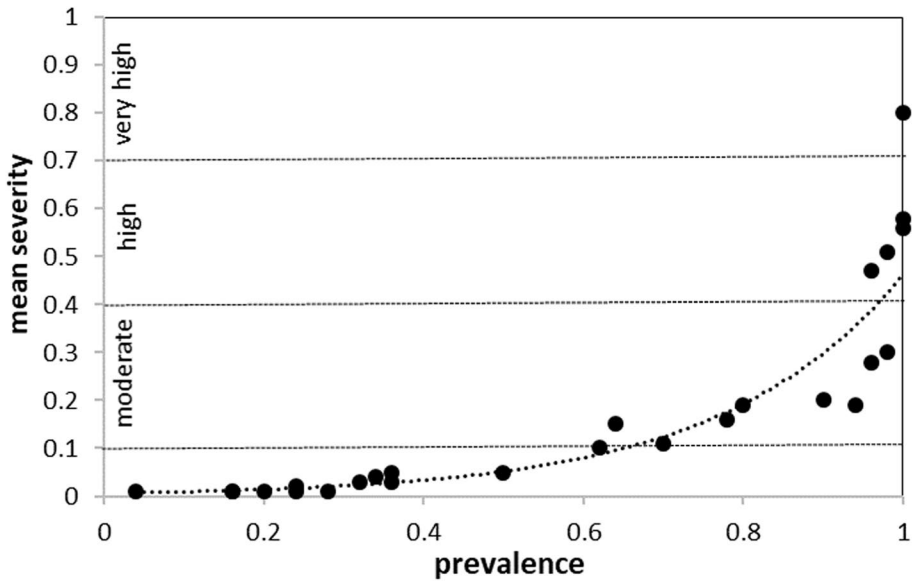

Fig. 4 Mean severity—prevalence relationship observed for larch canker disease in the infected larch stands ($n = 37$, y = 0.0058 e 4.3763, $r^2 = 0.95$, $P < 0.001$)

Table 3 Summary of the GLMs testing, in two different orders, the effect of stand age, humidity, growth index and the percentage of trees assigned to Alpine gene pool (PAP) on larch canker prevalence

Predictors	df	Deviance	Residual df	Deviance	F-value	*P*-value
Age	1	709.052	51	1052	39.727	< 0.0001
Humidity	1	184.962	50	867	10.363	0.002
PAP	1	100.252	49	767	5.617	0.022
Growth. index	1	0.754	48	766	0.042	0.838
Age	1	709.052	51	1052	39.727	< 0.0001
PAP	1	139.015	50	913	7.789	0.008
Humidity	1	146.2	49	767	8.191	0.006
Growth.index	1	0.754	48	766	0.042	0.838

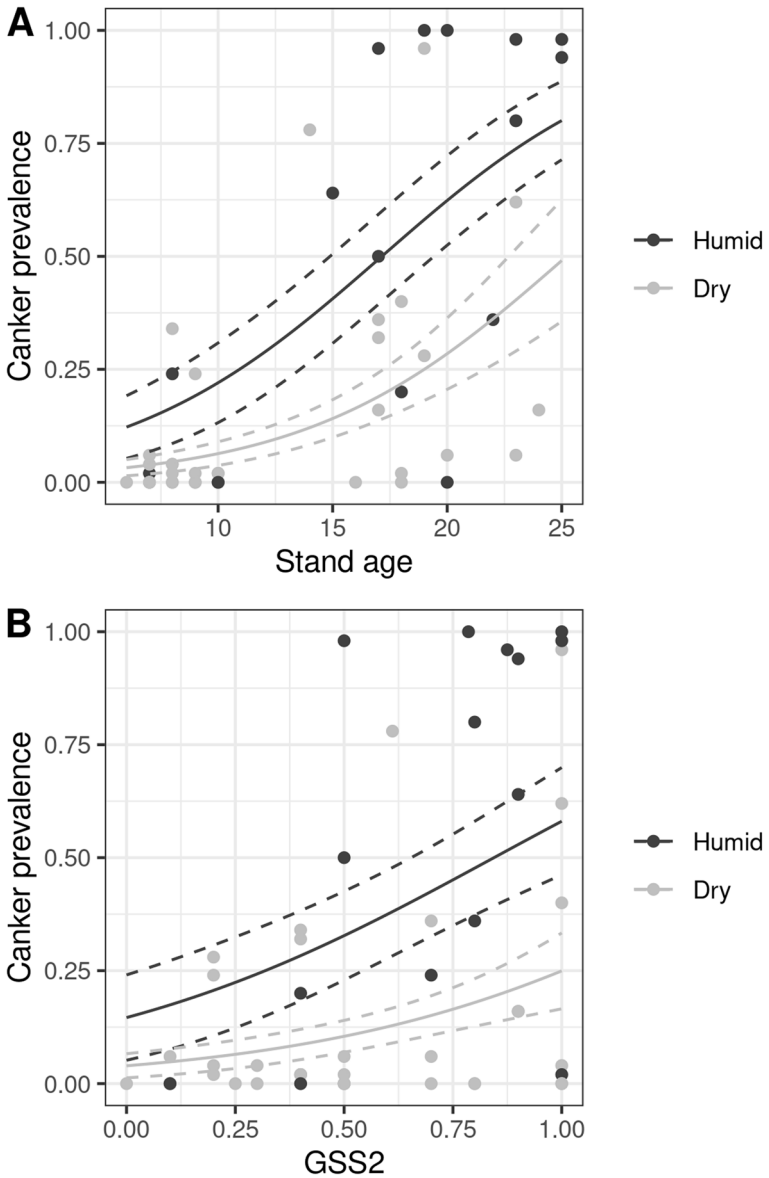

Fig. 5 Effects of stand age, atmospheric humidity and genetic stand susceptibility (GSS2) on larch canker prevalence. GSS2 is defined as the mean value of tree genetic scores (0.05, 05 and 0.95 for CE, admixed and alpine genotypes, respectively)

3.3 Field susceptibility of larch trees to larch canker and genetic assignment (second campaign)

Disease prevalence and mean severity assessed in the five stands were consistent with the first campaign disease scores (Table 7 in Appendix). Prevalence varied from 50 to 100% and the mean severity from low to high. The genetic analysis allowed to assign the 140 trees to the Alpine pool (26% of the genotyped trees), the CE pool (16%) or to the admixed group (58%). The percentage of admixed trees per stand varied from 0 to 80%. The percentage of infected trees was significantly higher for trees assigned to the Alpine pool than for admixed trees and for trees assigned to the Central European pool. There was no significant difference between these two latter gene pools (Fig. 6a, $Chi^2 = 18.5$, $P < 0.001$). Larch canker severity also differed according to genetic assignment (Kruskal–Wallis test: $H = 33.2$; $p < 0.0001$). Patterns were partly consistent with those observed for the infection status: severity was five times stronger in larches assigned to the alpine pool that in admixed trees, and 10 time stronger in Alpine trees than in Central European trees (Fig. 6b).

4 Discussion

4.1 Genetic composition and admixture in larch plantations

The assignment of trees sampled in the study stands afforested with certified material clearly highlighted the discrepancy between the expected larch origin (the origin mentioned on the official document) and the observed origin (as inferred using genetic analyses). According to the registration documents, in 31 of the stands studied, Central European larches had been used for reforestation. We confirmed this origin for about a third of the trees, while another third came from the Alps and the last was classified as admixed. In the five stands selected for the second field campaign, over 50% of the sampled trees were classified as admixed, further confirming the high percentage of admixed trees in larch plantations established outside of the natural range of *L. decidua*. Our results provide evidence that this second generation of outside-range translocations into France was carried out with forest material that was not of pure CE origin. It was pointed out by different authors that the first wave of within- and outside-range translocations (at the time of the "Lärchenmanie" period, which started in the 1850s) opened up the possibility of mixing between translocated and native populations (Geburek and Myking 2018; Jansen and Geburek 2016; Wagner et al. 2015a). It is likely that provenance certification made for material issued from CE seed orchard was based on false premises since it was not always possible, before the advent of genetic traceability tool, to distinguish native trees from translocated or admixed ones (Lines 1992). According to Cieslar, cited by Oudin and Fourchy (1952), as early as 1904, the numerous introductions of Alpine larch into the Sudetes made the origin of Sudeten trees uncertain. In such a context, misidentification of larch populations and wrong labelling of seed lots were inevitable. Cascading larch translocations in Europe also resulted in mixed planted forests. Such genetic heterogeneity, although already reported in other forest tree species (König et al. 2002), was not expected. It is however in agreement with the strong human influence on larch populations.

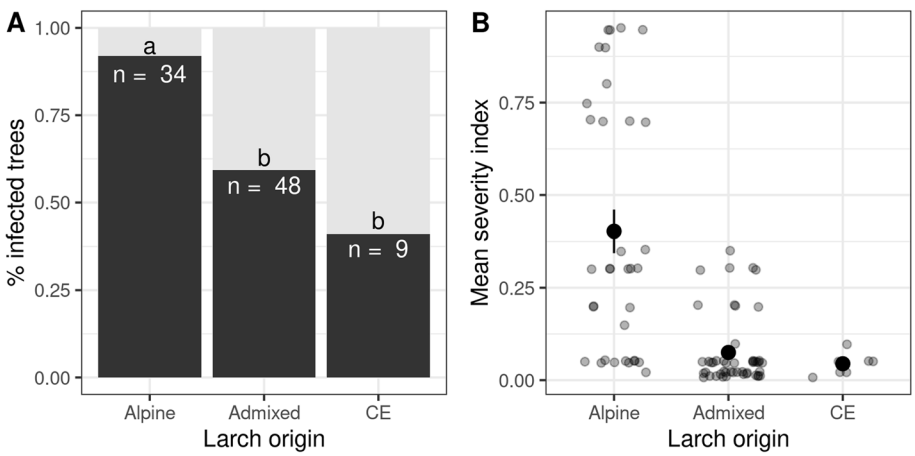

Fig. 6 Percentage of trees infected by *Lachnellula willkommii* (**A**) and disease severity (**B**) according to their inferred gene pool (Alps, CE and admixed individuals)

4.2 Drivers of larch canker prevalence in plantations

Predicting potential losses induced by a disease is of high priority before defining management and silvicultural options. Although many authors (e.g., Bürgi 1990; Schober 1977) have argued that it is difficult to note symptoms of larch canker on branches in adult stands, we calculated prevalence by scoring both the presence of symptoms on branches and stem cankers. This scoring method is relatively straightforward because the lower branches are most infected. It is more relevant than previous approaches since only one third of symptomatic trees showed cankers on the trunk. It also probably brings greater sensitivity to statistical evaluations. Measuring the prevalence of larch canker proved to be more simple and accurate than a score of mean severity at the stand level. This suggests that modelling this prevalence-severity relationship should be valuable to improve the assessment methods of other forest tree diseases (such as ash dieback or Diplodia shoot blight, for example) and to better understand their epidemiological trends. The prevalence-severity relationship followed a 'type C' curve, according to Seem's nomenclature (Seem 1984). Such relationship is associated with wind-borne pathogens and involves quantitative resistance in plants, which matches well with the known traits of *L. willkommii* × *L. decidua* pathosystem.

Our epidemiological study confirmed the role of larch genetic origin, site humidity and stand age on disease prevalence. Prevailing conditions in humid regions of intermediate altitude favor *L. willkommii* multiplication and dispersal (Schober 1977; Bürgi 1990; Petercord and Straßer 2012). Our study shows that silvicultural management can mitigate their effect. In disease-conducive sites, characterized by cool and moist climatic conditions, the use of Central European larch trees resulted in a low canker prevalence. On the other hand, in sites planted with Alpine trees, any operation to reduce atmospheric humidity can help maintain a low level of disease, as observed for the two oldest studied forests. In our study, the larch growth-index had no significant influence on disease prevalence. This is not consistent with the data collected by Weiberger and Šindelář (1992) or Weisberger (1995) who observed in common gardens a negative correlation between growth and canker frequency. However, our results corroborate the experimental work of Sylvestre-Guinot and Delatour (2002) who concluded that the vigor of the trees did not affect disease development. Altogether, our results support the proposal that integrated larch canker disease management can be achieved by monitoring provenance origin and by using site-specific management.

4.3 Larch tree susceptibility to larch canker disease

Assessing variation in disease resistance among geographic provenances is not an easy task, because disease resistance (the ability to limit infection and colonization by a pathogen) is a complex phenotype, which depends, as any host–pathogen interaction trait, on the host (especially its genetic make-up and its age), the pathogen and the environment. Proxies or components of this resistance can be assessed by controlled inoculation of seedlings or mature trees (Potts et al. 2016; Telford et al. 2015) or by field observations of damage in provenance trials (Boshier and Buggs 2015; Sniezko and Koch 2017). We confirmed the higher resistance of Central European larch genotypes, which express less severe and less frequent symptoms than Alpine larch genotypes. Furthermore, by highlighting the low genetic purity of larch stands, our findings help elucidate some contradictory results from provenance tests. Indeed, the occurrence of Alpine larch material in these genetically diverse plantations might explain the disease outbreaks involving Sudeten plantations in the United Kingdom (Buczacki 1973; Pawsey and Yooung 1969) or in Germany (Schober 1977). We report here the first field evaluation of the resistance of intraspecific hybrids to larch canker disease. They display a high level of resistance comparable to that of central European trees. In interspecific hybrids between *L. decidua* × *L. kaempferi*, the resistance of the hybrid larch is inherited from its Japanese genitor, whatever the direction of the cross (Sylvestre-Guinot et al. 1999). In the future, it would be worth testing the hypothesis that dominant genes are involved in resistance of Central European provenances.

4.4 Lessons for assisted migration and for disease management

Whether resistance to pathogens can be improved in native populations by using exotic source of gametes has rarely been tested with long-term trials (Boshier and Buggs 2015). Similarly, within-range translocations of populations with higher disease resistance traits have rarely been documented. On the contrary, reports of natural introgression of resistant exotic species gene pools into native susceptible gene pools exist (Brunet et al. 2013). Our results suggest that translocating disease-resistant provenances into areas where the causal pathogen is widespread can increase tree fitness and favor forest regeneration. However, the high frequency of Alpine larch trees in plantations made with certified Central European material underlines limitations in the recommendations that can be made for larch plantations. First, the methods ensuring the traceability of

forestry material must be improved, since in some sites the certificates of origin could not be found. Second, the orchard certification procedure needs to be revised in Europe. As long as larch certification will continue to consider only the geographic origin and not the genetic composition of seed lots, purity and homogeneity of the larch plant material will not be guaranteed, and consequently adaptation to local conditions will not be guaranteed either. Seed orchards should be set up only with genetically controlled material and in sites where genetic pollution by exotic gene flow is sufficiently low. Third, in low elevation plantations, outside the native range of larch, where larch canker disease is a strong limitation for afforestation, a silvicultural monitoring is required to limit as much as possible disease development, especially in disease-conducive sites. Finally, the high frequency of intraspecific hybrids found did not compromised the health status of larch plantations. This is due to their high field canker resistance, an important result. However, before including these plants in the list of recommended larch material for planting, it is necessary to confirm and better characterize this resistance. For this purpose, controlled inoculations could be carried out (Sylvestre-Guinot et al. 1999), using progenies from controlled crosses between different genetic pools.

Our study can also contribute to the debate on assisted migration of forest species or provenances to locations that could better suit them climatically in the future (mostly northward migrations in the northern hemisphere). First, to effectively assist gene flow (Aitken and Whitlock 2013), the origin of translocated material must be checked to allow traceability and proper certification (see recommendations above). For this purpose, genomic resources combined with recent technologies allow the development of a large number of promising genetic markers (*e.g.* Laurent et al. 2020). Second, our study underlies the risk of genetic pollution (Thomas et al 2014; Unger et al 2016; Williams and Dumrose 2013). Local larch populations (e.g., in the Alpine and Sudetes range) can indeed be threatened by the translocation of non-indigenous populations. The conservation of genetic resources in protected areas as well as in orchards would be the best insurance against this risk, provided that non-contamination by exotic pollen is assessed. For outside-range translocations, regardless of whether they correspond to assisted range expansion or assisted species migration (see Williams and Dumrose 2013 for definitions), the main risk is maladaptation of the introduced population or species. A strong requirement is that the target habitat must correspond to the future requirements of the target species. European larch transfers and larch canker outbreaks beyond the species native range illustrate that biotic interactions (including diseases) can counteract the expected effects of assisted migration (Simler et al. 2019).

Identifying the geographic origin of *L. willkommii* should provide us with information on the coevolutionary trajectories of the larch and its pathogen and on the origin of intraspecific variation in larch canker resistance. This is a recommended step to implement sustainable forest management (Desprez Loustau et al. 2016; Ennos 2015). In natural ecosystems, coevolution and reciprocal adaptation are shaping the evolutionary dynamics of plants and their pathogens. Resistance to a sympatric pathogen is expected in areas where the host and the pathogen coexist since a long time. European larch history was reconstructed thanks to fossil and genetic data (Wagner et al. 2015b). However, the origin of *L. willkommii* is still unclear: historical records of this pathogen come from both Japan, Asia (on *L. sibirica*) and Europe (Yde-Andersen 1979). Considering the resistance of Japanese larch (*L. kaempferi*) and the finding of a 104 years old canker in a Japanese larch, Kobayashi (1970) suggested that *L. willkommii* was native from Japan. This hypothesis is still frequently raised (e.g., Giroux and Bilodeau 2020). However, Hartig (1891) suggested that the pathogen was present in the high Alpine regions since the end of the eighteenth century, much before any importation of Japanese larch trees in Europe started. The hypothesis of a European origin for *L. willkommii* is consistent with the reduced severity of the disease in larch natural European range. The disease caused by this pathogen is a limiting factor only outside larch natural range and has never been a problem inside this range. European larch current distribution range could therefore be partly the result of the sanitary pressure exerted on larch by *L. willkommii.* The limits of this range did not change significantly over the last few centuries in the Alps, which is also in disagreement with the hypothesis of an introduced exotic pathogen. The hypothesis that the Sudetes, western Carpathians and southern Carpathians were refuge areas for larch during the ice period (Dostalek et al. 2018) is also an argument for a European origin of *L. willkommii*. New investigations of *L. willkommii* populations from Europe and Asia are required to test this hypothesis and to elucidate where and when larch resistance has evolved.

Appendix

Table 4 Plot characteristics

Plot	Age (years)	Altitude (m)	Height (m)	Adjusted height, Ha	Growth index	Density (number of stems/ha)	Slope	Atmospheric humidity
11787	23	1337	15.2	14.9	0.02	1800	0.18	1
11788	8	1375	3.6	6.3	−0.43	900	0.2	0
11789	23	1338	16.2	14.9	0.09	1800	0.12	1
11826	8	1072	7.6	6.3	0.21	625	0.05	0
11827	8	976	6.8	6.3	0.08	2500	0.12	1
11828	10	1223	9.4	7.4	0.27	1100	0.1	0
11829	9	1287	6.6	6.8	−0.04	1100	0.12	0
11830	10	1260	8.2	7.4	0.10	1100	0.05	0
11831	25	1231	19.6	16.1	0.22	1100	0.3	1
11832	19	1458	8.2	12.6	−0.35	1800	0.1	1
11833	20	1552	5.6	13.2	−0.58	1100	0.18	0
11834	7	1529	5	5.7	−0.12	1800	0.1	1
11835	15	1514	5.6	10.3	−0.46	1100	0.14	1
11836	10	1387	4	7.4	−0.46	1100	0.25	0
11837	10	1417	4.6	7.4	−0.38	1100	0.2	0
11880	8	1466	3.2	6.3	−0.49	1100	0.1	0
11894	22	1260	19.6	14.3	0.37	1800	0.18	1
11895	23	1292	15.6	14.9	0.05	1800	0.01	0
11897	17	1074	13.6	11.5	0.19	1100	0.01	0
11979	10	1300	7	7.4	−0.06	1100	0.18	0
11980	10	1323	6.8	7.4	−0.08	900	0.08	0
11981	7	1227	4.8	5.7	−0.16	1100	0.05	0
11982	7	1338	5	5.7	−0.12	1800	0.08	0
11983	17	1142	18.2	11.5	0.59	875	0	0
11985	17	1178	17.4	11.5	0.52	1100	0.05	1
11987	7	1287	5.2	5.7	−0.09	625	0.18	0
12572	8	1171	8	6.3	0.28	1100	0	0
12617	9	1232	7.8	6.8	0.14	1100	0	0
12619	18	1204	13.5	12.0	0.12	900	0.6	0
12622	18	1060	14.7	12.0	0.22	1100	0.14	0
12623	78	1021	30.6	30.7	0.00	100	0.2	0
12624	23	1246	10.8	14.9	−0.28	500	0.15	0
12625	51	547	31.1	28.7	0.08	480	0.05	0
12626	9	1060	7.2	6.8	0.05	1000	0	0
12627	19	1015	8.3	12.6	−0.34	500	0	0
12644	7	1106	8.3	5.7	0.46	1100	0.04	0
12645	10	994	10.9	7.4	0.47	1000	0.1	0
12646	8	1046	8.2	6.3	0.31	1100	0.05	0
12647	8	1251	4.9	6.3	−0.22	1000	0.05	0
12672	10	1159	9.6	7.4	0.29	800	0	0
12673	24	1199	13.3	15.5	−0.14	800	0.6	0
12718	126	1323	34.2	34.3	0.00	100	0	0
12719	17	1133	11	11.5	−0.04	1000	0.15	0
12720	17	1057	11.6	11.5	0.01	1000	0.05	1
12721	9	875	5	6.8	−0.27	1100	0	0
12722	16	948	7.5	10.9	−0.31	1300	0.35	0

Plot	Age (years)	Altitude (m)	Height (m)	Adjusted height, Ha	Growth index	Density (number of stems/ha)	Slope	Atmospheric humidity
12723[a]	19	1110	13.9	12.6	0.10	1000	0.2	0
12726	48	1047	26.2	28.5	−0.08	80	0.1	0
12729	10	800	9.5	7.4	0.28	1000	0.1	1
12730	7	1095	6.3	5.7	0.11	500	0.35	0
12731	18	974	14.3	12.0	0.19	1200	0.15	1
12732	6	960	5	5.1	−0.02	1000	0.35	0
12733	20	752	20	13.2	0.52	1000	0.1	1
13551	20	864	8	13.2	−0.39	1000	0	1
13552	25	820	14.8	16.1	−0.08	1000	0	1
G	14	1288	8.8	9.7	−0.10	1100	0.15	0
P	18	1213	9	12.0	−0.25	700	0.12	0

Table 5 Larch tree assignation to gene pools after analysis with structure

Plot	Certified origin	N. genotyped trees	N.A[a]	N.CE[b]	N.Admix[c]	GSS1	GSS2	GSS3
11787	A	5	2	2	1	0.41	0.5	0.59
11788	CE	5	1	2	2	0.23	0.41	0.59
11789	NA	5	4	1	0	0.77	0.77	0.77
11826	CE	5	5	0	0	0.95	0.95	0.95
11827	CE	5	2	0	3	0.41	0.68	0.95
11828	CE	5	0	2	3	0.05	0.32	0.59
11829	CE	5	0	3	2	0.05	0.23	0.41
11830	CE	5	2	3	0	0.41	0.41	0.41
11831	A	5	4	0	1	0.77	0.86	0.95
11832	A	5	5	0	0	0.95	0.95	0.95
11833	NA	5	2	0	3	0.41	0.68	0.95
11834	CE	5	5	0	0	0.95	0.95	0.95
11835	CE	5	4	0	1	0.77	0.86	0.95
11836	NA	5	5	0	0	0.95	0.95	0.95
11837	NA	5	5	0	0	0.95	0.95	0.95
11880	CE	5	0	3	2	0.05	0.23	0.41
11894	A	5	3	0	2	0.59	0.77	0.95
11895	CE	5	5	0	0	0.95	0.95	0.95
11897	CE	5	0	1	4	0.05	0.41	0.77
11979	CE	5	1	2	2	0.23	0.41	0.59
11980	CE	5	2	2	1	0.41	0.5	0.59
11981	CE	5	0	4	1	0.05	0.14	0.23
11982	CE	5	1	3	1	0.23	0.32	0.41
11983	CE	5	2	0	3	0.41	0.68	0.95
11985	CE	5	1	1	3	0.23	0.5	0.77
11987	CE	4	0	0	4	0.05	0.5	0.95
12572	CE	5	0	3	2	0.05	0.23	0.41
12617	CE	5	3	1	1	0.59	0.68	0.77
12619	NA	5	3	0	2	0.59	0.77	0.95
12622	NA	5	1	2	2	0.23	0.41	0.59
12623	NA	5	4	0	1	0.77	0.86	0.95

Plot	Certified origin	N. genotyped trees	N.A[a]	N.CE[b]	N.Admix[c]	GSS1	GSS2	GSS3
12624	CE	5	0	4	1	0.05	0.14	0.23
12625	NA	5	0	5	0	0.05	0.05	0.05
12626	CE	5	2	2	1	0.41	0.5	0.59
12627	CE	5	5	0	0	0.95	0.95	0.95
12644	CE	5	0	4	1	0.05	0.14	0.23
12645	CE	5	1	2	2	0.23	0.41	0.59
12646	NA	5	0	3	2	0.05	0.23	0.41
12647	CE	3	0	0	3	0.05	0.5	0.95
12672	CE	5	1	1	3	0.23	0.5	0.77
12673	NA	5	4	0	1	0.77	0.86	0.95
12718	NA	5	5	0	0	0.95	0.95	0.95
12719	NA	5	4	0	1	0.77	0.86	0.95
12720	A	4	3	0	1	0.725	0.8375	0.95
12721	CE	5	4	1	0	0.77	0.77	0.77
12722	NA	5	1	1	3	0.23	0.5	0.77
12723	CE	5	0	3	2	0.05	0.23	0.41
12726	NA	5	0	5	0	0.05	0.05	0.05
12729	CE	5	0	1	4	0.05	0.41	0.77
12730	CE	5	0	4	1	0.05	0.14	0.23
12731	NA	5	1	2	2	0.23	0.41	0.59
12732	NA	5	0	5	0	0.05	0.05	0.05
12733	NA	5	0	4	1	0.05	0.14	0.23
13551	NA	7	5	1	1	0.69	0.76	0.82
13552	NA	5	5	0	0	0.95	0.95	0.95
G	NA	9	3	1	5	0.35	0.6	0.85
P	NA	5	5	0	0	0.95	0.95	0.95
Total		287	121	84	82			

[a] *N.A* number of trees assigned to the Alpine gene pool, [b]*N.CE* number of trees assigned to the Central European gene pool, [c]*N. Admix* number of admixed trees

Table 6 Summary of GLMs testing the effect of stand age, humidity, genetic stand susceptibility (GSS), and growth index on larch canker prevalence

Model	Predictors	df	Deviance	Residual df	Deviance	*F* value	*P* value	R^2
GSS1	Age	1	709.052	51	1052	39.47	< 0.0001	0.56
	Humidity	1	184.962	50	867	10.296	0.002	
	GSS1	1	93.287	49	774	5.193	0.027	
	Growth.index	1	1.299	48	773	0.072	0.789	
GSS1	Age	1	709.052	51	1052	39.47	< 0.0001	0.56
	GSS1	1	129.924	50	922	7.232	0.01	
	Humidity	1	148.326	49	774	8.257	0.006	
	Growth.index	1	1.299	48	773	0.072	0.789	
GSS2	Age	1	709.052	51	1052	39.727	< 0.0001	0.56
	Humidity	1	184.962	50	867	10.363	0.002	
	GSS2	1	100.252	49	767	5.617	0.022	
	Growth.index	1	0.754	48	766	0.042	0.838	
GSS2	Age	1	709.052	51	1052	39.727	< 0.0001	0.56
	GSS2	1	139.015	50	913	7.789	0.008	
	Humidity	1	146.2	49	767	8.191	0.006	
	Growth.index	1	0.754	48	766	0.042	0.838	

Model	Predictors	df	Deviance	Residual df	Deviance	*F* value	*P* value	R^2
GSS3	Age	1	709.052	51	1052	39.977	< 0.0001	0.55
	Humidity	1	184.962	50	867	10.428	0.002	
	GSS3	1	78.579	49	789	4.43	0.041	
	Growth.index	1	1.825	48	787	0.103	0.75	
GSS3	Age	1	709.052	51	1052	39.977	< 0.0001	0.55
	GSS	1	108.766	50	943	6.132	0.017	
	Confinement	1	154.775	49	789	8.726	0.005	
	Growth.index	1	1.825	48	787	0.103	0.75	

Table 7 Assessment of larch canker and results of tree genetic assignment in the five larch stands studied in 2012 and 2015

Larch stand	Disease scoring						Genetic assignment					
	Sampling size		Prevalence (%)		Severity		Sampling size		% of Alpine trees		% of admixed trees	
	2012	2015	2012	2015	2012	2015	2012	2015	2012	2015	2012	2015
#11897	50	41	32	61	0.03	0.04	5	41	0	10	80	59
#11985	50	50	50	56	0.05	0.06	5	50	20	20	60	72
#12723	50	20	28	50	0.01	0.01	5	20	0	0	40	80
G	50	9	78	89	0.16	0.18	9	9	33	33	56	56
P	50	20	100	100	0.56	0.56	5	20	100	100	0	0

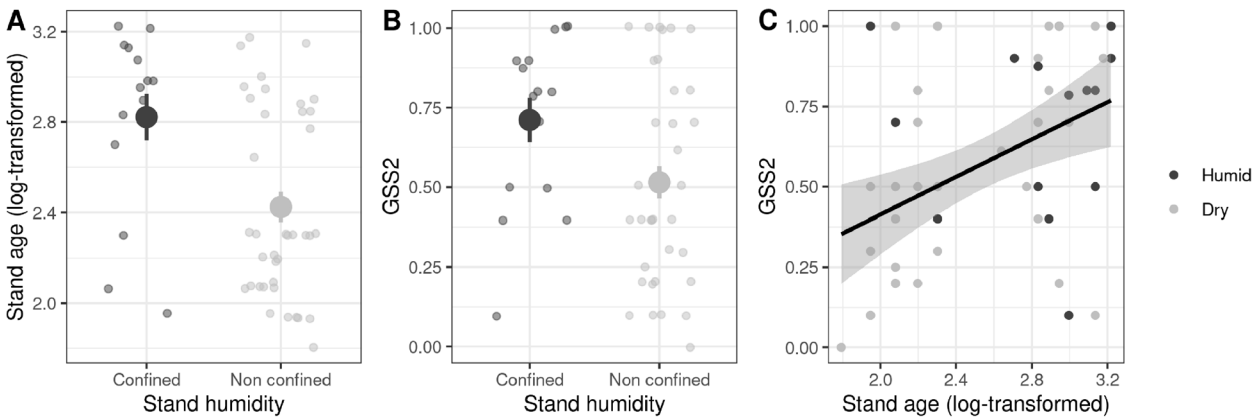

Fig. 7 Relationship between larch stand age, humidity, canker prevalence and genetic stand susceptibility (GSS2)

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Consent to participate
Not applicable.

Authors' contributions
Conceptualization: Dominique Piou, Cécile Robin, Stefanie Wagner; Methodology: Dominique Piou, Cécile Robin, Stefanie Wagner; Data acquisition, formal analysis, and investigation: Dominique Piou Cécile Robin, Stefanie Wagner, Olivier Baubet, François Ehrenmann, Bastien Castagneyrol, Xavier Capdevielle, Olivier Fabreguettes, Rémy Petit; Writing—original draft preparation: Cécile Robin, Stefanie Wagner, Bastien Castagneyrol, Rémy Petit, Dominique Piou; Writing—review and editing: all authors. Funding acquisition: Dominique Piou, Cécile Robin. All authors read and approved the final manuscript.

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Availability of data and materials
The datasets generated during and/or analyzed during the current study are available from the repository Dataverse INRAE (https://doi.org/10.57745/2VRAD6).

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
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Competing interests
The authors declare that they have no conflict of interest.

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