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Intra- and interspecific inheritance of some components of the resistance to leaf rust (*Melampsora larici-populina* Kleb.) in poplars

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Abstract Clonal variation for rust resistance was studied in a factorial mating design of *Populus trichocarpa* and *P. deltoides* with 17 intraspecific and 15 interspecific progenies. Susceptibility to *Melampsora larici-populina* was assessed both under natural conditions in the nursery and after leaf-disk inoculation with two pathogen races in a controlled environment. Different components of the resistance were taken into account: immunity in the field or in the laboratory, latent period, number and size of uredia in the laboratory, and field resistance at the end of the growing season. Genes controlling immunity were contributed only by the *P. deltoides* parents. The distributions of clonal means within each family suggested a polygenic inheritance of field resistance in the *P. trichocarpa* × *P. trichocarpa* crosses, but major gene effects were suspected in the interspecific progenies. Inefficiency of the quantitative mechanisms of resistance in the interspecific F_1 hybrids might have important implications for future breeding strategies. Field and laboratory trials complemented each other well, and a combined selection approach is proposed.

Key words *Populus* · Qualitative disease resistance · Quantitative disease resistance · Interspecific hybridization · Combined selection

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Introduction

Poplar culture is an example of clonal forestry. Several diseases and pests may affect production when this technique is used, such as foliar fungi, cankers (fungi or bacteria), viruses, and insects, and the management of these is one of the major preoccupations of popular growers. Several approaches can be considered, but the best results are expected to be obtained from breeding and selection (Pinon 1984; Thielges 1985). Thielges et al. (1989) and Wang and Van der Kamp (1992) found that following rust infection there was a quantitative variation in growth reduction relative to the tolerance of the poplar clones.

Melampsora larici-populina Kleb is spread all over Europe, is also present in Asia and South America, and it was recently detected on the western side of North America (Newcombe and Chastagner 1993). Different pathological races have been identified within this species (Pinon et al. 1987; Pinon and Peulon 1989; Pinon 1992) based on clone-race compatibility (immunity) after some selected clones had lost their resistance with time. This recently occured on a large scale with clone 'Luisa Avanzo', resistant to races E1 and E2, which has lost its immunity due to emergence of race E3 (Giorcelli et al. 1992).

Therefore, two different kinds of interaction can be found between *Populus* spp. and *Melampsora* spp. that we may describe as quantitative and qualitative. The breeder's objective is to select for general and durable rust resistance, and polygenic resistance is commonly assumed to be more durable. Selection for rust resistance is generally based on nursery trials under natural conditions and with an unknown mixture of inocula (possibly complemented by artificial inoculation). This field resistance may be considered to be an integrated target for selection, which includes different epidemiological parameters like a longer latent period, a smaller number of uredia per leaf area, and reduced sporulation. The prediction of the genetic values for such a complex trait may be enhanced by breaking it down into simpler components.

In the work presented here, we attempted to infer some genetic characteristics of these two types of resistance from a factorial mating design. We combined nursery trials, which integrate whole plant physiology and the interactions which climatic conditions (Prakash and Thielges 1989), with laboratory tests under a controlled environment and with various pathogen races, since the susceptibility level for a single genotype may evolve according to the inoculum content (Pinon 1992). The consequences for *Populus* breeding are discussed, both in a traditional strategy and as an illustration of a trait for which marker studies could be of great help in the near future (Nance et al. 1992).

Material and Methods

Materials

Eight *Populus trichocarpa* Torr. and Gray and 10 *P. deltoides* Bartr. clones from different origins were selected to perform a 9 × 9 factorial mating design in which each parent is involved in both intra- and interspecific crosses (Villar et al. 1990). The reverse crosses (*P. trichocarpa* as a female and *P. deltoides* as a male) were not available at the time of this study, mainly because of the difficulty in achieving these interspecific combinations.

Thus 16 *P. trichocarpa* × *P. trichocarpa* progenies and 15 *P. deltoides* × *P. trichocarpa* families were involved in the nursery trials, plus 1 *P. deltoides* × *P. deltoides* family as an indicator for that species. All of the *P. trichocarpa* × *P. trichocarpa* families were also involved in the laboratory tests. Around 30 full-sibs were clonally propagated within each family (Table 1).

Nursery trials

In order to reduce errors in disease resistance classification due to the different growth rhythm of the *P. trichocarpa* and the interspecific progenies, two separate trials were established in April 1992 with 5 commercial clones as common controls that were chosen according to their compatibility with the different *Melampsora* species and races (Table 2). The same experimental design was applied in the two trials (six randomized complete blocks, single-tree plots). The same cultural treatments were also applied: cuttings were planted under a plastic film, with 1.2 m between rows and 0.5 m within a row. Two border rows surrounded each trial; one was made with clone 'Robusta', which is known to be susceptible to all the species and races of *Melampsora*. The controls were repeated three times in each block of the two trials. Rust susceptibility was evaluated in September 1992 on each tree using the scale of Pichot and Teissier du Cros (1993b): from 1, if there are no visible uredia on any leaf of the whole plant (observed immunity), to 9, if uredia cover more than three-quarters of the surface area of the most heavily infected leaf and more than 25% of the leaves of the whole plant show this level of infection.

Table 1 Factorial mating design involved in the study. A code number (in brackets) and the progeny size are given for each family

Males	*P. trichocarpa* 36-100	19-73	19-77	101-74	*P. deltoides* L123-011
Females					
P. trichocarpa					
36-77	(11)	(12)	(13)	(14)	–
	30	30	30	30	–
36-134	(21)	(22)	(23)	(24)	–
	29	30	30	30	–
212-161	(31)	(32)	(33)	(34)	–
	30	30	26	30	–
Fritzi Pauley	(41)	(42)	(43)	(44)	–
	30	30	30	30	–
P. deltoides					
73028-62	–	–	–	(54)	–
	–	–	–	30	–
L150-89	(61)	(62)	(63)	(64)	–
	30	30	30	30	–
L132-41	(71)	(72)	(73)	–	–
	30	12	28	–	–
L155-79	(81)	–	(83)	(84)	(87)
	30	–	30	30	30
T060-42	(91)	(92)	(93)	(94)	–
	30	30	30	30	–

Laboratory tests

The *P. trichocarpa* × *P. trichocarpa* families and control clones were grown in a glasshouse with special care to avoid any contamination by rust. Leaves were taken from the fifth to tenth node below the apex, and leaf disks were taken for inoculation with different rust inocula: *M. larici-populina* races E1 and E2 that were cultivated on clones showing different specific susceptibilities. The leaf disks (3 cm in diameter) were put upside down on water in multiwell dishes. A spore suspension adjusted to 7000 +/− 1000 spores/ml was sprayed over the disks, and observations were made over the following 15 days.

For the quantitative assessment of the resistance, special attention was given to the control of environmental conditions. First, temperature (18°–20 °C) and photoperiod (16 h) were globally controlled in the chamber. Then, heterogeneity within the chamber was taken into account by experimental design: each experiment consisted of three randomized complete blocks (single-disk plots) inoculated one at a time and cultivated on different shelves. The 81 multidishes that were necessary for each block covered around 0.8 m^2. The location of each disk was recorded on a *xy* basis, and we performed a spatial analysis of the data using the iterated Papadakis method. The advantages and drawbacks of this method based on a moving average are discussed in Pichot (1993), and it seemed relevant in our experiments where there was no significant family × block interaction. Four experiments were made with races E1 and E2 between June and August 1992.

Three epidemiological components of the resistance were assessed over one cycle of the fungus: (1) the final number of uredia (log transformation was necessary for normality), (2) the latent period, roughly approached through the proportion of uredia that had already matured and fruited on day 10 or 11 (we used a square root transformation of this variable), and (3) the sporulation (the size of uredia was scored on a 1–3 scale, where the 'Robusta' control was given a score of 2).

Table 2 Compatibility of the controls with different *Melampsora* species and races (from Pinon 1991) and mean susceptibility in the two nursery trials. Clone 'Luisa Avanzo' was not infected at all in Trial 1 (all ramets scored 1) and only slightly diseased in Trial 2; the means of the controls were computed without this clone. Trial 1 contains the *P. trichocarpa* × *P. trichocarpa* families; trial 2 contains the interspecific and *P. deltoides* families

	M. larici-populina E1	E2	E3	*M. allii-populina*	Susceptibility Trial 1	Trial 2
Unal	+	+	+	+	8.5	6.1
Beaupré	–	–	–	+	2.7	2.8
Fritzi Pauley	+	+	+	+	4.4	4.6
I-214	+	+	+	+	4.6	4.5
Luisa Avanzo	–	–	+	+	(1.0)	(1.4)
Mean of the controls					5.0	4.5

Genetic analysis

As a first step, data from the two nursery trials were analysed with a fixed-effect model, and clonal means were computed. Then we estimated some genetic parameters for the *P. trichocarpa* trials. As the parents were partly selected for disease resistance, these values were only used to study the potential of the laboratory tests in the selection for field resistance.

Restricted maximum likehood estimates of the variance components were computed. The analysis of such a complete factorial mating design relies on the following model:

$$Yijkl = M + Bi + Mj + Fk + (M.F)jk + (C.M.F.)jkl + Eijkl$$

where M is the general mean; Bi, the block effect (fixed), only for the nursery trial; Mj, the male effect; Fk, the female effect; $(M.F)jk$, the male × female interaction; $(C.M.F.)jkl$, the clone effect within full-sib progeny; and $Eijkl$, the residual.

Additive and dominance genetic variances, respectively Va and Vd, can be estimated considering that in the absence of epistasis and consanguinity:

$$\mathrm{var}(M) = \mathrm{var}(F) = \mathrm{cov}HS = 1/4\,Va$$

$$\mathrm{var}(M.F) = \mathrm{cov}FS - 2\mathrm{cov}HS = 1/4\,Vd$$

$$\mathrm{Var}(C.M.F.) = \mathrm{cov\ clone} - \mathrm{cov}FS = 1/2\,Va + 3/4\,Vd.$$

Considering that variance estimates were far more precise at the clonal level (more than 350 df in all tests) than at the family level (15 df), we computed the following approximations of the genetic parameters:

$Vg = Va + Vd$ was approached by: $Vg' = 2\mathrm{var}(C.M.F.)$

$h^2 = Vg/(Vg + Ve)$ was approached by: $h^{2\prime} = Vg'/(Vg' + Ve)$.

The value of $h^{2\prime}$ is not far from h^2 except for high values of Vd. The genetic correlations were computed at the *C.M.F.* level, and coefficients of genetic prediction between two traits, i and j, were estimated as follows:

$$CGPij' = 2\mathrm{cov}(C.M.F.)ij/\mathrm{sqrt}((Vg' + Ve)i(Vg' + Ve)j).$$

Noting that $CGPii$ is the heritability of trait i, then $CGPij$ gives the reciprocal predictive value of the two traits i and j one on the other (Baradat 1976). The coefficients of genetic prediction of field resistance with each laboratory component were computed.

The fixed effect analysis was computed using the S-MODLI (1990) package and the SELECT (1992) package was used for the estimation of the variance components; other computations were made with Splus (1990).

Results

Components of the natural inoculum

The ranks of the controls were the same in the two trials (Table 2), and inoculum pressure was slightly higher in the *P. trichocarpa* trial. The different levels of infection sugested that the natural inoculum consisted mainly of *M. larici-populina* races E1 and E2, with no significant presence of race E3 and a small amount of *M. allii-populina*. Simultaneously, we sampled 225 uredia in two locations in the nursery on clones that were compatible with the three *M. larici-populina* races: individual identification of these uredia led us to the same conclusion – a predominance of race E1, with only a 5–6% prevalence for race E3.

Qualitative versus quantitative resistance

The clonal differences for resistance in the nursery were highly significant, and some clones were not infected at all (no uredia on all leaves of the six ramets) in spite of highly infected neighbours. Such immune phenotypes were found in the interspecific progenies and the *P. deltoides* × *P. deltoides* family, but not in the *P. trichocarpa* × *P. trichocarpa* progenies. These results confirmed individual observations that were previously made in the stool-beds where the whole mating design was maintained.

The laboratory tests confirmed that there was no immunity among the *P. trichocarpa* clones for either race E1 or E2. However, they did show only moderate levels of infection in the nursery trial compared to the mean of the controls. The distribution of the clonal means in these families was consistent with the underlying hypothesis of quantitative genetics for characters under polygenic control (Fig. 1).

In contrast, some interspecific clones were even more infected than the most susceptible control, 'Unal', and the distributions of susceptibility in the *P. deltoides* × *P. trichocarpa* families were highly heterogeneous, sometimes bimodal (Fig. 2). These distributions did not fit the classical quantitative approach. The families could be classified according to the *P. deltoides* female parent: female 'L150-89' gave resistant progenies (families 61, 62, 63, and 64), but female 'L123-41' gave highly susceptible ones (71, 72, and 73); the progenies from female 'L155-79' did segregate (81, 83, and 84) and those from females '73028-62' or 'T060-42' showed wide dispersal (54, 91, 92, 93, and 94). Extreme phenotypes could be found within a single full-sib progeny.

The results of the nursery trials are detailed for three related families in Fig. 3. In both intraspecific families (23 and 87) all of the clones were either immune or showed low levels of susceptibility, whereas in interspecific family 83 the clones were immune or highly susceptible with a mean score above 5, thus showing no quantitative resistance. The qualitative resistance of these interspecific genotypes was checked in the laboratory. They were found to be either susceptible to (compatible with), or resistant to, both races E1 and E2 simultaneously. The compatible clones were severely attacked in the nursery, while the resistant ones stayed immune except for four of them that were slightly diseased; their infection in the nursery might be due to the moderate inoculum of *M. larici-populina* race E3 or *M. allii-populina*.

Genetic parameters for quantitative resistance

The broad-sense heritability of the number of uredia and the size of uredia in the laboratory were higher than the heritability of resistance in the nursery (Table 3). For the latent period, heritability estimates varied between

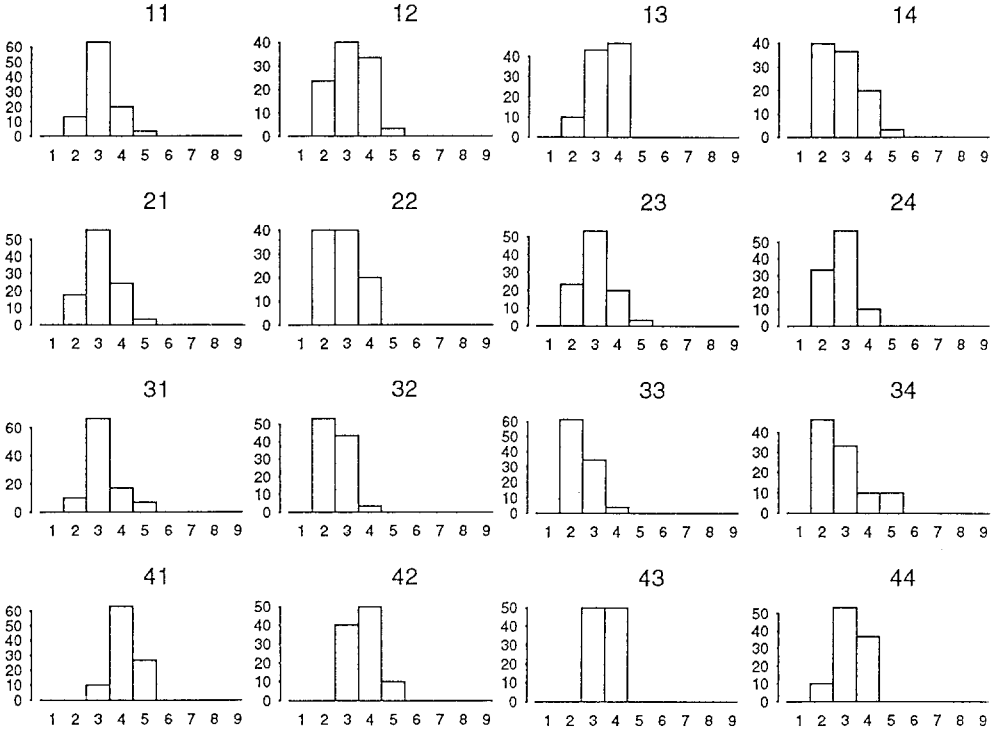

Fig. 1 Frequency disribution (%) of the clonal means within each *P. trichocarpa* × *P. trichocarpa* family: the family codes are as listed in Table 1. The mean susceptibility of the controls was 5.0 in that trial

experiments, suggesting the presence of a genotype × environment interaction. This latter problem might be more due to our measure of that component than to the component itself.

The genetic correlation between the number and the size of uredia was positive and quite stable in the different experiments (0.31–0.49). The environmental correlation between these traits was lower but in the same direction (0.13 to 0.20), thus suggesting pleiotropic effect rather than linkage disequilibrium. The latent period and the size of uredia were negatively correlated (-0.12 to -0.49). The correlation between the latent period and the number of uredia was particularly unstable in our four experiments. This is another argument for a GxE interaction, which suggests that our criteria for latent period evaluation were not correct.

The coefficients of genetic prediction integrate both the genetic covariance between traits and their heritabilities. The estimates of the coefficients of genetic prediction for field resistance were higher with the size and the number of uredia, but remained below the value of heritability for nursery resistance (Table 3).

Discussion

In our material we observed that genes conferring immunity to local pathotypes were contributed by the *P. deltoides* females but not by the *P. trichocarpa* males. When we considered only trees in the nursery that were diseased, we detected some quantitative resistance in both pure species but none in the interspecific hybrids. Therefore, we could assume that qualitative resistance exists in *P. deltoides* together with quantitative resistance, while our *P. trichocarpa* material only had quantitative resistance. Confusion between qualitative and quantitative resistances in *P. deltoides* was also found by Pichot and Teissier du Cros (1993a). This confusion makes genetic analysis more complex. The lack of polymorphism for qualitative resistance should not be generalized at once to the whole *P. trichocarpa* species: however, in screening a collection of *P. trichocarpa* clones from various origins, we never found any immunity against *M. larici-populina* isolates (data not shown).

The distribution of partial resistance within the *P. trichocarpa* material was that of a polygenic trait. In contrast, major genes might be suspected to have a predominant role in the interspecific progenies: their polymorphism would concern only the *P. deltoides* females and, at least for one family, the distribution of susceptibility in the nursery was largely explained by the segregation for compatibility with races E1 and E2. In their study on the resistance to *M. occidentalis* among intra- and interpopulation *P. trichocarpa* crosses, Hsiang et al. (1993) detected a strong maternal effect, but in our case we concluded that there is an unequal contribution of the two parental species to the variability of interspecific F_1. This has been observed in other forest tree species like eucalyptus (Bouvet et al. 1992).

We observed a negative transgression for rust resistance: the loss in the interspecific hybrids of the quantitative mechanisms of resistance that were expected on the basis of the interspecific crosses from the same parents. This phenomenon is important and should be further investigated. We might be able to explain it if we

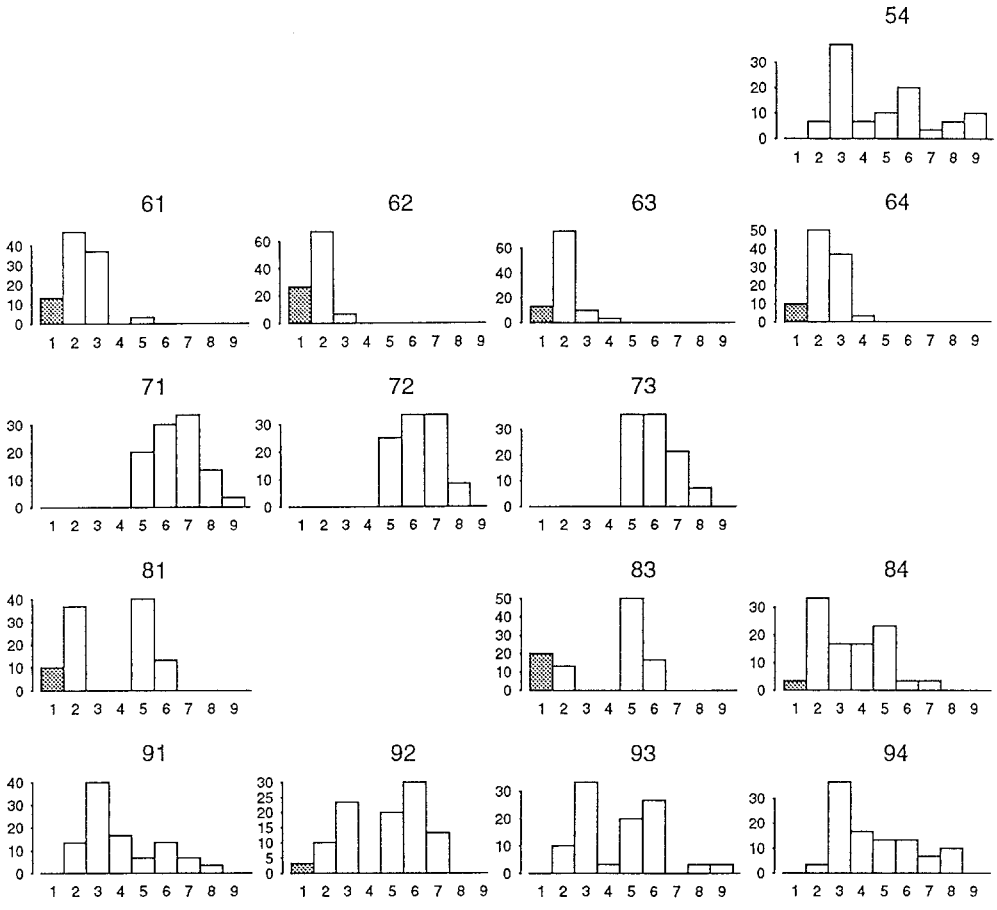

Fig. 2 Frequency distribution (%) of the clonal means within each *P. deltoides* × *P. trichocarpa* family: the family codes are as in Table 1. Clones with no infection at all on the six ramets appear in the *first dashed bar*. The mean susceptibility of the controls was 4.5 in that trial

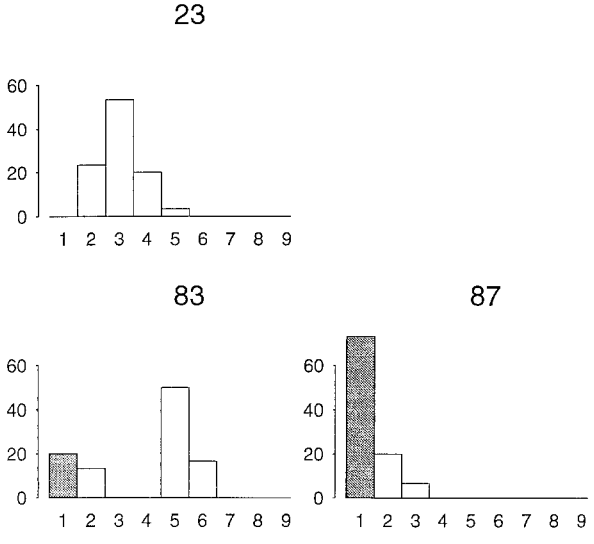

Fig. 3 Frequency distribution (%) of the clonal means within one interspecific progeny (83) and the two intraspecific-related families (23 and 87). Non-infected clones appear in the *first dashed bar*: they were not found in the *P. trichocarpa* progeny

Table 3 Broad-sense heritabilities of the resistance components and coefficients of genetic prediction of the field resistance with the different lab components

	h^2b_s	*CGP*
Field resistance	0.42	0.42
Latent period		
E1, 1st experiment	0.16	−0.07
E1, 2nd experiment	0.47	−0.09
E2, 1st experiment	0.29	−0.03
E2, 2nd experiment	0.37	−0.08
Number of uredia		
E1, 1st experiment	0.71	0.12
E1, 2nd experiment	0.82	0.17
E2, 1st experiment	0.68	0.11
E2, 2nd experiment	0.75	0.20
Size of uredia		
E1, 1st experiment	0.71	0.33
E1, 2nd experiment	0.74	0.28
E2, 1st experiment	0.56	0.27
E2, 2nd experiment	0.66	0.28

consider that quantitative resistance is a complex and polygenic trait that cannot be integrally inherited after F_1 interspecific hybridization; thus only the simpler (oligogenic) qualitative resistance mechanisms remain effective in such hybrids. Breeders have to carefully avoid selecting for the simplest resistance: considering the resistance to *M. larici-populina*, interspecific F_1 hybridization might not be the best strategy. It is interesting to note that historically the races were identified on commercial clones that are mainly interspecific hybrids.

Another strategy would consist in recovering quantitative resistance by intercrossing or backcrossing unrelated interspecific hybrids. In looking for the best clones in subsequent generations one would not necessarily lose the benefit of possible interspecific heterosis for other traits like vigor (Stettler et al. 1988). However, the fear for loss of durable resistance in the interspecific F_1 hybrids should not be generalized: good levels of resistance to *M. medusae* (Stettler et al. 1988) or *M. occidentalis* (Hsiang et al. 1993) were found in various interspecific F_1 combinations, and in the *Leuce* section, Gallo et al. (1985) showed that interspecific progenies had resistance to *M. magnusiana* intermediate between that of the intraspecific crosses.

The components of resistance that we studied had high broad-sense heritabilities. Similar heritability estimates were computed by Pichot and Teissier du Cros (1993b) for the number and size of uredia in a *P. deltoides* factorial design: 0.62 and 0.52, respectively. While the laboratory components provided some valuable information for the genetic prediction of field resistance, the direct assessment of field resistance in the nursery remained better than indirect prediction. A combined index based on both nursery and laboratory trials would be a good strategy for selection. In the laboratory, emphasis should be put on the improvement of the evaluation process for the latent period and sporulation. Prakash and Heather (1989), in a *P. deltoides* – *M. medusea* experiment, and Pichot and Teissier du Cros (1993b) found strong correlations between the laboratory components. Other parameters in the field could be relevant, like the precocity of infection. For this latter parameter, observations were also made in the two nursery trials in July: there were no significant differences among the *P. trichocarpa* families, but the most susceptible interspecific hybrids were also infected early. Working on separate components does not only improve the prediction of field resistance, it also provides an insight into the underlying anatomical, physiological, and biochemical processes to combine into a kind of "ideotype" for rust resistance. With the recent development of molecular markers in poplars (Liu and Furnier 1993; Bradshaw and Stettler 1993a, b), a marker-aided genotypic selection among poplar hybrids might be conceivable in the near future.

In this *Populus* – *Melampsora larici-populina* model, we can imagine some kind of gene-for-gene experiment, races being multiplied on specific poplar clones. However, the actual races seem to combine different virulences (Pinon 1992). As poplars may also combine several resistance genes, segregation is not so easy to analyse, and the use of genetic markers should be of great help in such a study (Villar et al. 1993). In our work, depending upon the *P. deltoides* female parent, different polymorphism might be revealed by segregation analysis. This illustrates the need for a multiple progeny approach in marker studies of forest tree species. This could be achieved at a lower extra cost for clonal species like poplars (Knott and Haley 1992; Knapp and Bridges, 1990).

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