dew), and genetic maps of some of these have been obtained through molecular markers.

So, we can reasonably assume that many resistance genes will be discovered within species of poplar as well as virulence genes in *Melampsora* species.

To reduce the probability of major damage caused by new rust races, race-specific, totally resistant clones should be avoided in selection. Knowledge of the gene-for-gene relation in the qualitative reaction to rust will allow poplar breeders to select quantitatively resistant clones that should be buffered to attain acceptable equilibria with heterogeneous, ambient rust inocula.

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Susceptibility of P. deltoides Bartr. to Melampsora larici-populina and M. allii-populina

II. Quantitative Analysis of a 6x6 Factorial Mating Design

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(Received 15th September 1992)

Summary

Quantitative susceptibility to *Melampsora* spp. was observed in a 6x6 *P. deltoides* factorial mating design. Artificial inoculation in the nursery proved inefficient for estimating partial resistance. However, in the laboratory test, susceptibility was efficiently partitioned into number of uredia size of uredia and latent period, and these 3 traits were correlated to natural rust infestation in the nursery. Partial resistance to rust caused by *Melampsora larici-populina* was highly heritable in *P. deltoides*. No correlations between growth and susceptibility were observed in our study. Additive variances estimated from the half sib effect overpassed additive variances estimated from clonal effect in the full sib families, which suggested that genetic differentiation occured between stands within the natural range of *P. deltoides*. The large amount of intra- and inter-specific partial rust resistance must be futher studied and categorized to better understand the *Populus-Melampsora* system and to optimize the use of these natural resources in poplar improvement.

Key words: *Populus deltoides*, *Melampsora larici-populina*, partial resistance, uredia, latent period, vigour, heritability, genetic correlation, factorial mating, natural variability.

Introduction

This quantitative analysis of *P. deltoides* susceptibility to rust follows a qualitative report based on the analysis of the same *P. deltoides* mating design (Pichot and Teissier du Cros, 1993). Quantitative polygenic (or horizontal resistance should be preferred over qualitative mono or oligogenic (or vertical) resistance, especially for forest

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tree species with long breeding cycle. The former is assumed to offer good resistance over time, while the latter may easily overcome by natural selection or by mutation of the pathogen (NELSON, 1978). This loss of resistance was observed as early as 1916 (KOMMEDAHL et al., in: PARLEVLIET and ZADOKS, 1977). Quantitative resistance can be defined as a combination of complementary traits of the host that reduces the pathogen development and multiplication, thus limiting total damage on hosts. Two traits are primarily considered: 1) latent period, and 2) rate of multiplication.

Genetic determination of these traits is of major importance for breeding strategies. For poplar susceptibility to rust, very few quantitative results have been published compared to qualitative ones. In this paper, we relate results of quantitative tests on susceptibility of *Populus deltoides* BARTR. to *Melampsora larici-populina* race E1 in both laboratory and nursery tests, and to *Melampsora* spp. in the nursery. Genetic parameters such as number of uredia, mean size of uredia and latent period were estimated as well as was the correlation between rust susceptibility and poplar growth in the nursery.

Materials and Methods

A) Origin of plant material and rust inuculum

The plants tested belong to an intraspecific *P. deltoides* factorial mating experiment involving 6 males and 6 females. Details on the crosses are given in PICHOT and TEISSIER DU CROS (1989).

Melampsora larici-populina race E1 was the only inoculum used for quantitative tests. Origin of inoculum and its multiplication are included in PICHOT and TEISSIER DU CROS (1993).

B) Experimental design

B 1) In the nursery

Experimental design was described in PICHOT and TEISSIER DU CROS (1993).

B 2) In the laboratory

Only clones that were susceptible in the qualitative tests (PICHOT and TEISSIER DU CROS, 1993) were quantitatively analysed.

Foliar disk were prepared and inoculated as described for the qualitative tests. Quantitative tests were conducted in multidish cultures with six dishes of 33 mm diameter, for foliar disks of 30 mm in diameter. Every multidish plate contained disks of 3 clones, each represented by 2 neighbouring disks. Six complete blocks were inoculated with various inoculum concentrations (5000, 7500, 7500, 15000, 15000 and 20000 spores/ml). Tests were conducted in a controlled chamber at a 15h photoperiod at 17° C to 20° C. Spore germination rate was always over 90%.

C) Observations

C 1) In the nursery

Susceptibility to *Melampsora* was estimated by:

a) single score per tree of the extent of rust infection at the end of the growing seasons (10th to 11th October 1990 and 4th to 6th September 1991);

b) the number of uredia on the inoculated leaf 20 days after inoculation with race E1 (9th July 1991).

For the rust infection scoring, the following scale was used:

1) 0 uredia.

2) Less than 10 uredia on the heaviest infected leaf, less than 50% of the leaves have uredia.

3) Less than 10 uredia on the heaviest infected leaf, more than 50% of the leaves have uredia.

4) Uredia cover less than 50% of the surface area of the heaviest infected leaf, and less than 50% of the leaves have this rust development.

5) Uredia cover less than 50% of the surface area of the heaviest infected leaf, and more than 50% of the leaves have this rust development.

6) Uredia cover from 50% to 75% of the surface area of the heaviest infected leaf, and less than 50% of the leaves have this rust development.

7) Uredia cover from 50% to 75% of the surface area of the heaviest infected leaf, and more than 50% of the leaves have this rust development.

8) Uredia cover more than 75% of the surface area of the heaviest infected leaf, and less than 50% of the leaves have this rust development.

9) Uredia cover more than 75% of the surface area of the heaviest infected leaf, and more than 50% of the leaves have this rust development.

Total height in 1990 and 1991 and diameter at 1m above the ground were also measured to estimate correlations between vigour and rust susceptibility.

C 2) In the laboratory

The number of uredia on each disk was counted every day. Three traits were analysed: total number of uredia per disk, latent period (delay between inoculation and sporulation of half of all the uredia) and mean size of the uredia classified as small, medium or large. The size of the uredia was assumed to be correlated with the number of urediospores per uredia. Thus, the size of the uredia was a crude estimation of multiplication rate. The tests ended 15 days after inoculation.

D) Variance analysis

Data were analysed according to the following statistical model:

$$X_{ijklm} = MU + B_i + M_j + F_k + M_jxF_k + CL_l/M_jxF_k (+ UP_{il}) + EPS_{ijklm} \quad (1)$$

MU	: general mean
B_i	: Block effect
M_j	: Male effect (half sibs)
F_k	: Female effect (half sibs)
M_jxF_k	: Male x Female (full sibs) interaction
CL_l/M_jxF_k	: Clone effect in full sib families
UP_{il}	: Unit Plot effect
EPS_{ijklm}	: error

Unit Plot effect was taken into account only for nursery traits. UP_{il} was defined as $block_i$ x $clone_l$ interaction, because each clone in each block was represented by 2 neighbouring ramets. We assumed that there were no genetic x environment effects.

E) Estimation of genetic parameters

Phenotypic value = Genetic value + Environmental value = Additive value + Dominance value + Environmental value

$$PH = G + E = A + D + E$$

We assume that A, D and E are independent, so:

$$\sigma^2_{PH} = \sigma^2_G + \sigma^2_E = \sigma^2_A + \sigma^2_D + \sigma^2_E \quad (2)$$

Considering that in our statistical model, all effects except block can be taken as random, genetic variances and, thus, genetic parameters can be estimated as follows:

$$\sigma^2_M = \text{cov. half sibs} = 1/4\ \sigma^2_A \quad (3)$$

$$\sigma^2_F = \text{cov. half sibs} = 1/4\ \sigma^2_A$$

$$\sigma^2_{MxF} = \text{cov. full sibs} - 2\ \text{cov. half sibs} = 1/4\ \sigma^2_D$$

$$\sigma^2_{CL/MxF} = \text{cov. clones} - \text{cov. full sibs} = 1/2\ \sigma^2_A + 3/4\ \sigma^2_D$$

$$\sigma^2_A = 4\ ((m-1)\ \sigma^2_M + (f-1)\ \sigma^2_F)/(m+f-2)$$

$$\sigma^2_A = 2\ \sigma^2_{CL/MxF} - 6\ \sigma^2_{MxF}$$

$$\sigma^2_D = 4\ \sigma^2_{MxF}$$

(m-1) and (f-1) are the degrees of freedom associated with male and female effects.

σ^2_M, σ^2_F, σ^2_{MxF} and $\sigma^2_{CL/MxF}$ were estimated according to the expected mean squares and mean cross-products (BECKER, 1984).

F) Correlations between traits

In addition to genetic additive correlations between rust susceptibility in autumn 1991 and vigour, correlations between all the traits observed in the different tests were also computed with the estimated genetic values of the clones and of the half-sib families.

G) Computations

ANOVA analyses were performed with S-MODLI (1990) and all others computations and figures were realised with Splus (1990) installed on a Sun 4/390.

Results

A) Laboratory test

A.1) Distribution of the traits

The rust is expected to grow exponentially in the foliar tissue according to the susceptibility of the clone and the level of inoculation. A log transformation made the distribution more normal. Because of the 0 level, log(x+1) was used. The distribution was largely assymmetrical because of the great number of clones with very few or no uredia. Absence of sporulation may be due to low susceptibility and/or to an incorrect level of inoculum. To avoid the latter, and to improve normally of the distribution, we eliminated disks with no uredia whose neighbouring disk of the same clone also had no uredia. Clones which supported sporulation only on 1 of the 6 disks were also eliminated. And finally, to reduce error variance, we used the mean of the 2 neigbouring disks instead of the individual values. The same transformations were used for necrotic flecks. *Figure 1* shows the effect of the transformation on normality. In spite of their discrete distribution, latent period (LP) and size of uredia (SU) were quantitatively treated. These 2 traits did not need to be transformed (*Figure 2*).

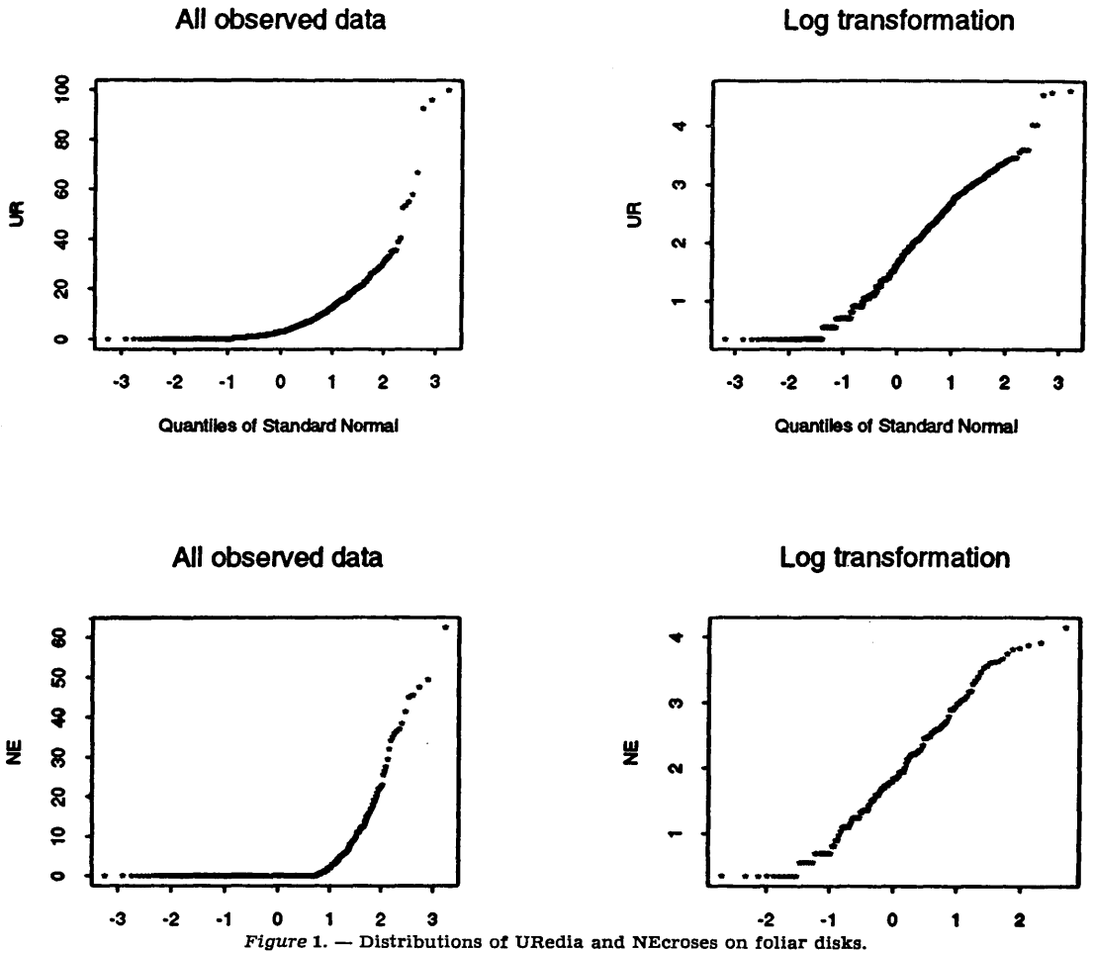

Figure 1. — Distributions of UReda and NEcroses on foliar disks.

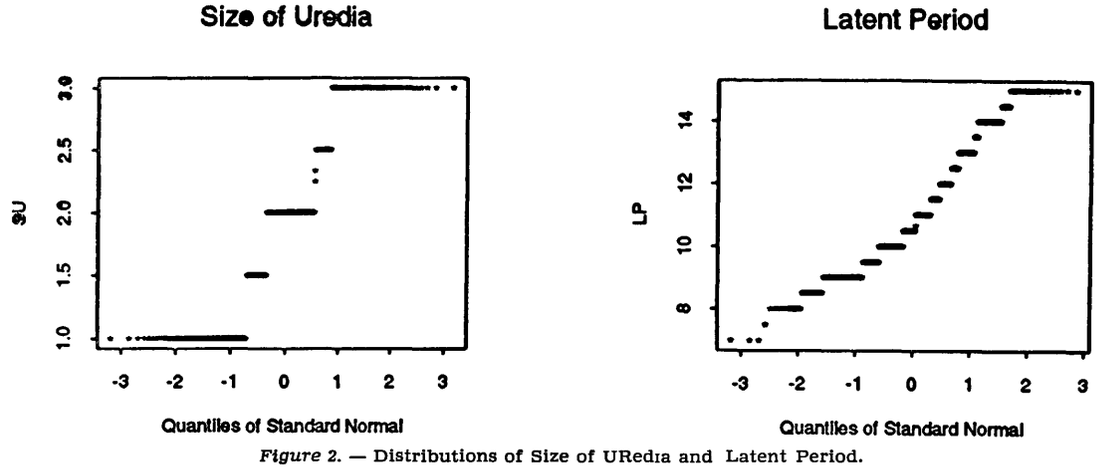

Figure 2. — Distributions of Size of URedia and Latent Period.

A.2) Variance analysis

From now on, the following notations will be used for simplicity:

UR = mean of log (number of URedia + 1) on 2 neighbouring disks,

NE = idem for NEcrotic flecks,

UN = idem for (Uredia + Necrotic flecks),

SU = mean of Size of Uredia on two neighbouring disks,

LP = mean of Latent Period on two neighbouring disks.

Homogeneity of variances was evaluated with the BARTLETT test (*Table 1*).

Significance of the factors *(Table 2)*.

Male (M) and female (F) effects were very important for UR, SU and LP, and interaction (MxF) was never significant. We note that the male effect, estimated on the 4 male half sib families which were susceptible to rust, was high for UR and NE but was nil for UN. Thus, the more rust sporulation in male half sib families, the less necrotic flecking, but numbers of uredia plus necrotic flecks were not significantly different from one male to the other.

A.3) Genetic parameters

There are no significant MxF interactions, thus no

Table 1. — BARTLETT test. P. values (%).

	CL/MxF	MxF	F	M
UR	02	84	85	44
SU	86	03	70	26
LP	00	00	47	24
NE	40	36	99	26
UN	17	32	26	63

Table 2. — Variance analysis. F. (FISHER) values and significance level. B., M, F, MxF and Cl/MxF are respectively the Block, Male, Female, Male x Female interaction and Clonal (in full sib family) effects.

	B	M	F	MxF	Cl/MxF
UR	3.1 **	13.5 **	5.7 **	0.6 NS	5.1 **
SU	18.4 **	31.1 **	11.8 **	0.7 NS	3.7 **
LP	1.5 NS	11.6 **	10.2 **	1.4 NS	5.1 **
NE	1.7 NS	6.8 **	1.6 NS	1.0 NS	2.8 **
UN	3.7 **	0.7 NS	5.8 **	0.7 NS	5.2 **
df	*5*	*3*	*5*	*7*	*120*

** : significant at 1%; * : significant at 5%; NS : non significant; df : degrees of freedom.

Table 3a. — Heritabilities and correlations in the laboratory test with *M. larici-populina* race El.

	Heritability(x100) H^2_{ns} (a)	(b)	H^2_{bs}	additive.cor.(x100) SU (a)	(b)	LP (a)	(b)	envir.cor.(x100) SU	LP
UR	52	62	62	94	48	-88	-34	24	-18
SU	65	52	52	100	100	-100	-78	100	-37
LP	68	55	61	/	/	100	100	/	/
NE	47	49	49	/	/	/	/	/	/
UN	46	62	62	/	/	/	/	/	/

(ns : narrow sense; bs : broad sense; (a) : from half sib effects; (b) : from clonal effect.)

Table 3b. — Heritabilities and additive correlations in the 4 most susceptible full sib families.

	Family 22 *			Family 42		
	H^2(x100)	Add. cor.(x100) SU	LP	H^2(x100)	Add. cor.(x100) SU	LP
UR	47	-04	28	59	28	-32
SU	56	100	-63	33	100	-45
LP	69	/	100	28	/	100
UN	65	/	/	61	/	/

	Family 64			Family 66		
	H^2(x100)	Add. cor.(x100) SU	LP	H^2(x100)	Add. cor.(x100) SU	LP
UR	42	56	-58	80	74	-65
SU	65	100	-100	69	100	-97
LP	56	/	100	78	/	100
UN	43	/	/	80	/	/

* Family number designates mating design; i.e., 42 = male 4 x female 2.

dominance variance ,and the clonal effect in the full sib families therefore gives us directly $\sigma^2_A/2$.

Heritabilities and additive correlations were estimated both from parental and clonal effects (*Table 3a*). To evaluate stability of estimates from clonal effect, heritabilities and correlations were also estimated in each of the 4 most susceptible full sib families, 22, 42, 64 and 66 (*Table 3b*).

Narrow sense heritabilities from clonal or parental effects are very similar, and estimations remain relatively constant from one family to the other except for LP in family 42. The additive correlation between size of uredia and latent period is always high and negative (*Figure 3* for families 64 and 66). Thus, the later sporulation occured, the smaller were the uredia.

Furthermore, the number of uredia per disk is positively correlated to the size of the uredia and negatively related to the latent period, particularly in families 64 and 66. Family 22 reveals a different reaction. UR and SU are independent and the later the clones sporulate, the more numerous are the uredia.

B) Nursery tests

B.1) Distribution of the traits

A log transformation was applied to rust notations:

Table 4. — Bartlett test. P. values (%).

	CL/MxF	MxF	F	M
UR2	13	05	00	22
NE2	71	83	40	34
UN2	53	30	69	44
R90	100	07	71	47
R91	13	00	82	94
D90	00	02	94	06
H90	00	14	62	58
H91	00	09	49	25

Table 5. — Variance analysis F. (Fisher) values and significance level. B, M, F, MxF, Cl/MxF and UP are respectively the Block, Male, Female, Male x Female interaction, Clonal (in full sib family) and Unit Plot effects.

	B	M	F	MxF	Cl/MxF	UP	EPS
UR2	70.0 **	0.1 NS	1.2 NS	0.5 NS	1.8 **	3.1 **	
df	*2*	*1*	*5*	*4*	*62*	*101*	*111*
NE2	59.3 **	0.8 NS	1.3 NS	0.6 NS	1.2 NS	2.5 **	
df	*2*	*4*	*5*	*7*	*66*	*68*	*92*
UN2	99.7 **	0.9 NS	2.8 *	0.8 NS	1.2 NS	2.9 **	
df	*2*	*4*	*5*	*8*	*99*	*149*	*179*
R90	2.2 NS	12.4 **	12.0 **	0.7 NS	1.9 **	1.5 **	
df	*2*	*5*	*5*	*12*	*150*	*184*	*221*
R91	9.3 **	16.4 **	26.1 **	1.6 (10%)	4.1 **	3.0 **	
D90	0.7 NS	14.3 **	2.0 (10%)	1.4 NS	2.8 **	1.1 NS	
H90	0.9 NS	10.7 **	3.8 *	2.4 **	2.5 **	1.4 **	
H91	3.8 *	7.0 **	5.1 **	2.0 *	2.1 **	1.5 **	
df	*2*	*5*	*5*	*17*	*218*	*371*	*415*

** : significant at 1%; * : significant at 5%; NS : non significant, df : degrees of freedom.

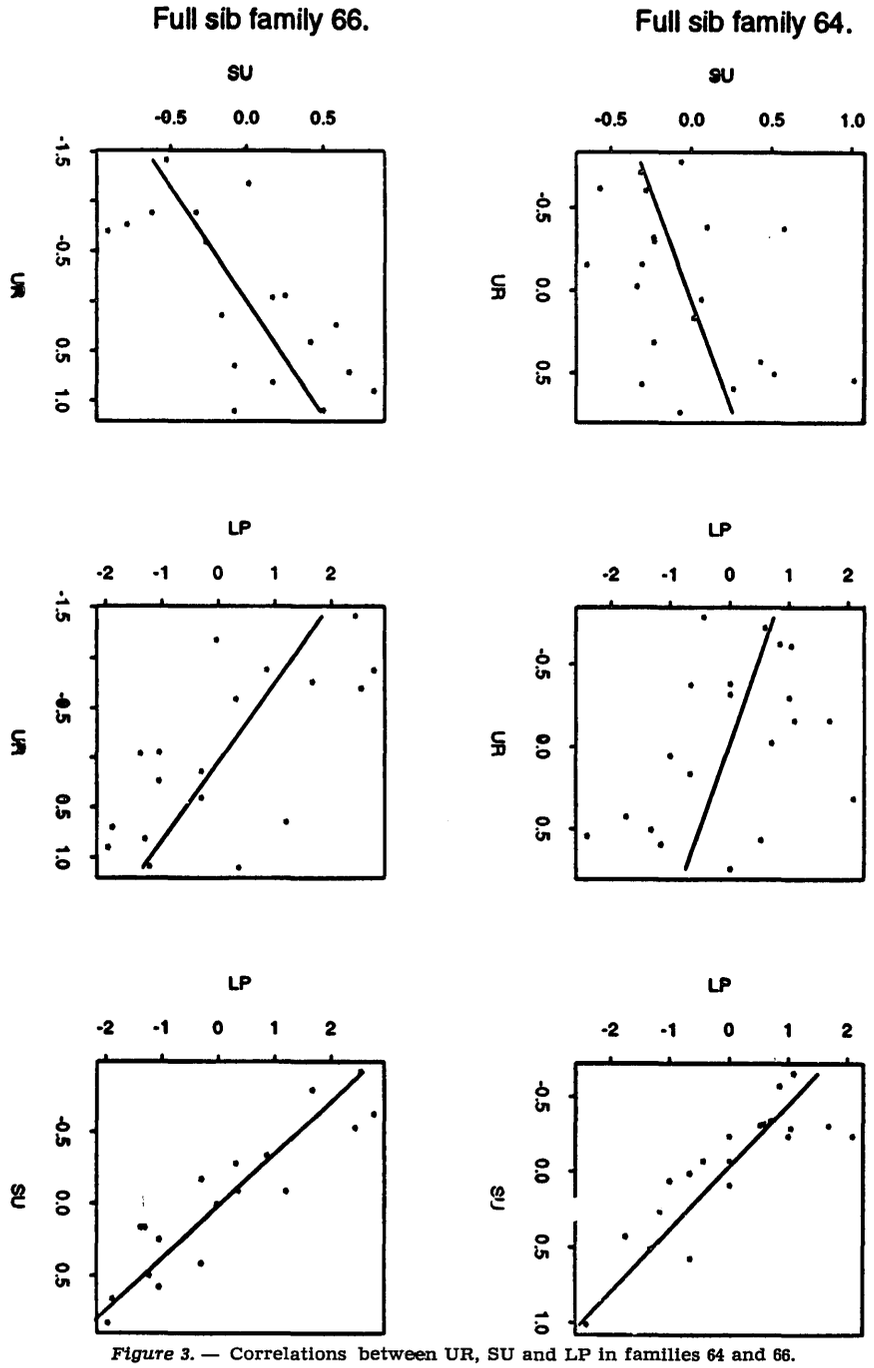

Figure 3. — Correlations between UR, SU and LP in families 64 and 66.

— number of uredia on the inoculated leaf (UR2) 20 days after inoculation;
— number of necrosis ... (NE2);
— number of uredia plus necrosis (UN2);
— susceptibility to rust in 1990 (R90), at the end of the growing season;
— susceptibility to rust in 1991 (R91), at the end of the growing season.

Only non 0 values were taken into account in the variance analysis. Furthermore, clones with only one susceptible ramet were considered as resistant (106 clones for R90 and 38 for R91 were concerned).

Growth traits: for normality diameter (D90) and height (H90 and H91) were square transformed.

B.2) Variance analysis

Homogeneity of variances (*Table 4*).

Significance level of the factors (*Table 5*).

Except for UR2 where the clonal effect is significant, there is no genetic effect for reaction to rust 20 days after inoculation, while block and unit plot effects are very high. On the contrary, observations of susceptibility to rust at the end of the growing seasons reveal significant genetic effect while block effect is this time lower.

B.3) Genetic parameters

Because of the inefficiency of the inoculation as determined by lack of sporulation 20 days after inoculation, no genetic parameters were estimated for UR2, NE2, and UN2.

For R90, R91 and growth in height, additive variances estimated from half sib family effect are higher than those obtained from clonal effect in full sib families (*Table 6*).

Table 6. — Ratio of the estimated additive variances.

	R90	R91	D90	H90	H91
$\sigma^2_{A\ par.}/\sigma^2_{A\ clon.}$	1.8	2.8	1.0	5.0	3.0

Table 7. — Heritabilities (x100) and correlations (x100) between traits in the nursery trial.

	heritability						add.cor.									envir.cor.		
	H^2_{ns}			H^2_{bs}			D90			H90			H91			D90H90		H9
	a	b	c	a	b	c	a	b	c	a	b	c	a	b	c			
R90	57	32	45	57	32	45	/	/	/	/	/	/	/	/	/			
R91	99	35	71	108	43	75	-19	-31	-21	25	-48	-17	12	08	13	03	02	-04
D90	37	38	38	43	45	45	100	100	100	82	92	71	72	-05	12	100	89	32
H90	48	10	38	69	30	50	/	/	/	100	100	100	79	-08	55	/	100	36
H91	34	12	28	51	28	39	/	/	/	/	/	/	100	100	100	/	/	100

(a) and b) : from half sib and clonal effects with F=0; c) : with estimated F).

Table 8. — Correlations (x100) between traits.

	UR	SU	LP	UN	UR2	UN2	R90	R91	D90	HT90	HT91
UR		41	-26	79	-06	02	22	33	02	01	04
SU	97 91		-69	15	07	-15	24	49	-01	02	11
LP	-96 -88	-100 -100		-03	-20	10	-02	-33	-03	-09	-14
UN	06 96	31 77	-35 -74		-03	02	18	27	08	01	04
UR2	/ /	/ /	/ /	/ /		75	17	17	06	11	17
UN2	/ /	/ /	/ /	/ /	/ /		02	05	09	03	01
R90	75 78	79 75	-80 -75	31 77	/ /	/ /		41	16	05	-05
R91	24 76	47 82	-51 -81	97 65	/ /	/ /	61 87		-06	-05	09
D90	-61 28	-69 03	68 -06	-39 34	/ /	/ /	06 12	25 -04		84	37
HT90	-68 32	-74 17	72 -16	-31 29	/ /	/ /	20 40	48 54	92 54		55
HT91	-48 -16	-45 -25	42 21	08 -18	/ /	/ /	47 -08	75 -07	79 77	93 70	

Upper triangular matrice : estimations based on clonal means in full-sib families.
Lower triangular matrice : estimation based on male then female half-sib family means.

This was previously observed in the analysis of another nursery trial with the same full sib families and could be explained by some allelic fixations in the different natural stands from where the parents were taken (Pichot and Teissier du Cros, 1989).

Considering F as the mean value of these fixations, we have:

$$\sigma^2_M = \text{cov. half sibs} = (1+F)/4\ \sigma^2_A \quad (4)$$

$$\sigma^2_F = \text{cov. half sibs} = (1+F)/4\ \sigma^2_A$$

$$\sigma^2_{MxF} = \text{cov full sibs} - 2\ \text{cov half sibs} = (1+F)^2/4\ \sigma^2_D$$

$$\sigma^2_{CL/MxF} = \text{cov clones} - \text{cov full sibs} = (1-(1+F)/2)\ \sigma^2_A + (1-(1+F)^2/4)\ \sigma^2_D$$

Thus, estimation of F leads to: 0.28, 0.39, 0, 0.30 and 0.22 for respectively: R90, R91, D90, HT90, HT91.

Taking into account (or not) the estimated values of F changes the estimation of genetic parameters (*Table 7*).

When ignoring F in the calculations, heritabilities estimated from the half sib family variances are higher than those evaluated from clonal effect and broad sense estimation exceeds 1.0 for rust susceptibility in 1991. Furthermore, genetic additive correlations estimated from clonal effect between growth in height in 1990 and 1991, is near to 0, but increases to 55% when fixations are considered. In either case, correlation between D90 and H91 remains very low.

Finally, R91 and vigour are very weakly correlated; negatively with growth in 1990 and positively in 1991.

C) Correlation between laboratory and nursery tests (Table 8)

Concerning *M. larici-populina* race E1, there is no correlation between the susceptibility level of the plant material tested in the laboratory and in the nursery 20 days after inoculation, but the nursery test was not efficient. Susceptibility of plant material tested under laboratory conditions is highly correlated to rust infection at the end of growing season in the nursery, when estimated on half-sib families from females or males with.

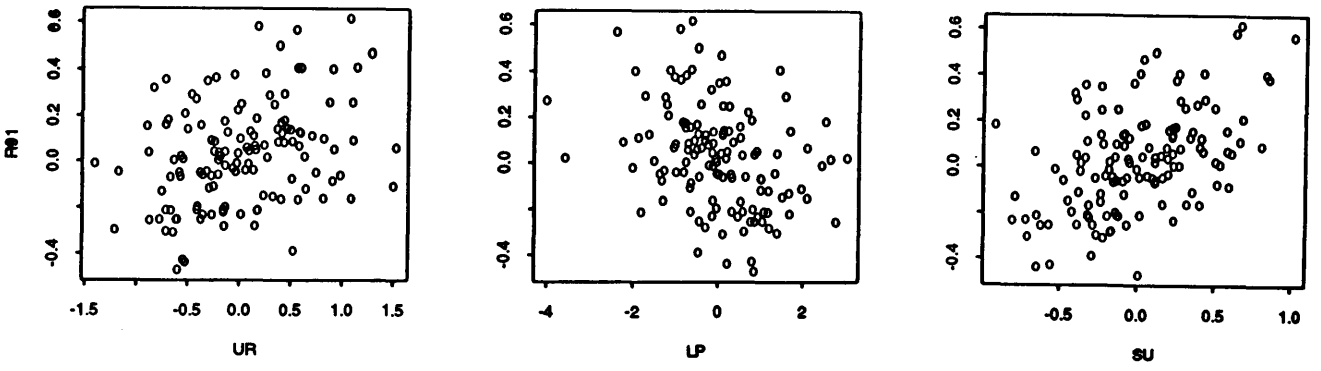

Figure 4. — Clonal (within full-sib families) correlations between rust infection in the laboratory (UR, LP, SU) and in the nursery in 1991 (R91).

respectively, only six females and four males. Estimates on clonal means (in full-sib families), lead to a lower but significant correlation. Correlations between the rust infection in the nursery and the two partial components of resistance (SU and LP) in the laboratory agree with the laboratory results. Families and clones which present short latent period and large uredia are more susceptible to rust in nursery (*Figure 4*). We note that clonal correlations between laboratory and nursery observations are lower in 1990 than in 1991. Moreover, R90 and LP are independent. Susceptibility of the plant material at the end of the growing seasons is more strongly correlated with laboratory estimations than with observations made in the nursery 20 days after inoculation. Finally, the correlation between the R90 and R91 is highly significant and, once again, higher when estimated from parental means.

Susceptibility to rust does not seem to be correlated with growth. Male and female half-sib families give very different and inconsistent results. The sample of males associates growth with low susceptibility in the laboratory and with relatively high susceptibility in the nursery, while the females associate resistance to rust with low growth in 1990 but with high growth in 1991.

Discussion

A) Variances within families and allelic fixations

Significantly unequal variances within half sib families are encountered only for UR2 and female effect.

When considering the full sib families, significant differences are found with SU, LP, R91, D90 and H91. Two points could explain this feature. First, the number of clones in full sib families is variable. The smallest and largest variances are generally encountered in the families with fewer clones (*Figure 5*). Second, this could reflect the variability of the level of heterozygosity of the parents used in the factorial mating. Thus, the more homozygous the parents, the less variable are their progenies. This might occur either with inbred parents or with parents originating from genetically different populations. But if heterozygosity were really variable from one parent to the other, we should have found significantly different variances between half sib families. Since we did not, we must assume that the allelic fixations revealed by the variance analysis are present in all the parents.

Literature about susceptibility of poplars to rusts suggests that populations from the large natural range of *P. deltoides* are naturally differentiated. Testing 81 open pollinated progenies, Farmer (1970) estimated that 51% of the total variance of susceptibility to *Melampsora* spp. was due to half sib effect (σ^2_{HS}). If families had come from heterozygous and unrelated mother trees randomly chosen in a panmictic population, σ^2_{HS} should have been equal to $1/4\ \sigma^2_A$ (additive variance). Thus, the remaining variance ($3/4\ \sigma^2_A + \sigma^2_D$ (dominance) $+ \sigma^2_E$ (environment)) (we ignore epistasis) should have been far greater ($> 3\ \sigma^2_{HS}$). In the same paper, variance among half sib families represented 90% of the total variance for foliation date in 1965 and 1966. Thielges and Adams (1975) reported results from a nursery trial involving 228 clones from 76 open polinated families collected in the northern part of the natural range of *P. deltoides*. Variances of half sib families and clones within these families represented, respectively, 72% (69% to 75%) and 21% (19% to 23%) of the total variance. Once again σ^2_{HS} should have been $1/4\ \sigma^2_A$ and σ^2_{CL}: $3/4\ \sigma^2_A + \sigma^2_D$, while results gave $\sigma^2_{HS} = 3.5\ \sigma^2_{CL}$.

In a hierarchical provenance-half sib family analysis of *P. deltoides*, Nelson and Tauer (1987) estimated the ratio variance between: variance within families (within stands) to $1/15$ for susceptibility to rust, and the highest values, obtained for branch number, did not exceed $1/4$. This result agrees with the observations of Thielges et al. (1989) in another provenance and progeny test, where stands and families within stands represented, respectively, 44% and 13% of the total variance, and σ^2 between/σ^2 within families was equal to 0.3. Thus, half sib family variances did not exceed the maximum expected value when they were estimated within stands. There would not be genetic structuration, which is not surprising at this level, and there would not be significant consanguinity neither.

According to Gallo et al. (1985), half sib family effects are largely predominant in the Leuce section also. The analysis of susceptibility to *Melampsora magnusiana* Wagner, in an inter- and intra-specific factorial mating with *P. tremula* L. and *P. tremuloides* Michx., revealed that σ^2_{HSmale} and $\sigma^2_{HSfemale}$ represented respectively 40% and 38% of the total variance.

In our experiment, both susceptibility to rust at the end of the growing seasons and growth in height are characterised by overestimations of σ^2_A when calculated on half sib effects. These results are in agreement with our previous observations (Pichot and Teissier du Cros, 1989) on the same material. It was noted that the more natural selection could affect a trait, the higher "allelic fixations" were. Thus, phenologic traits are most affected, followed by growth and wood quality traits. But in the laboratory test, where *M. larici-populina* race E1 was inoculated,

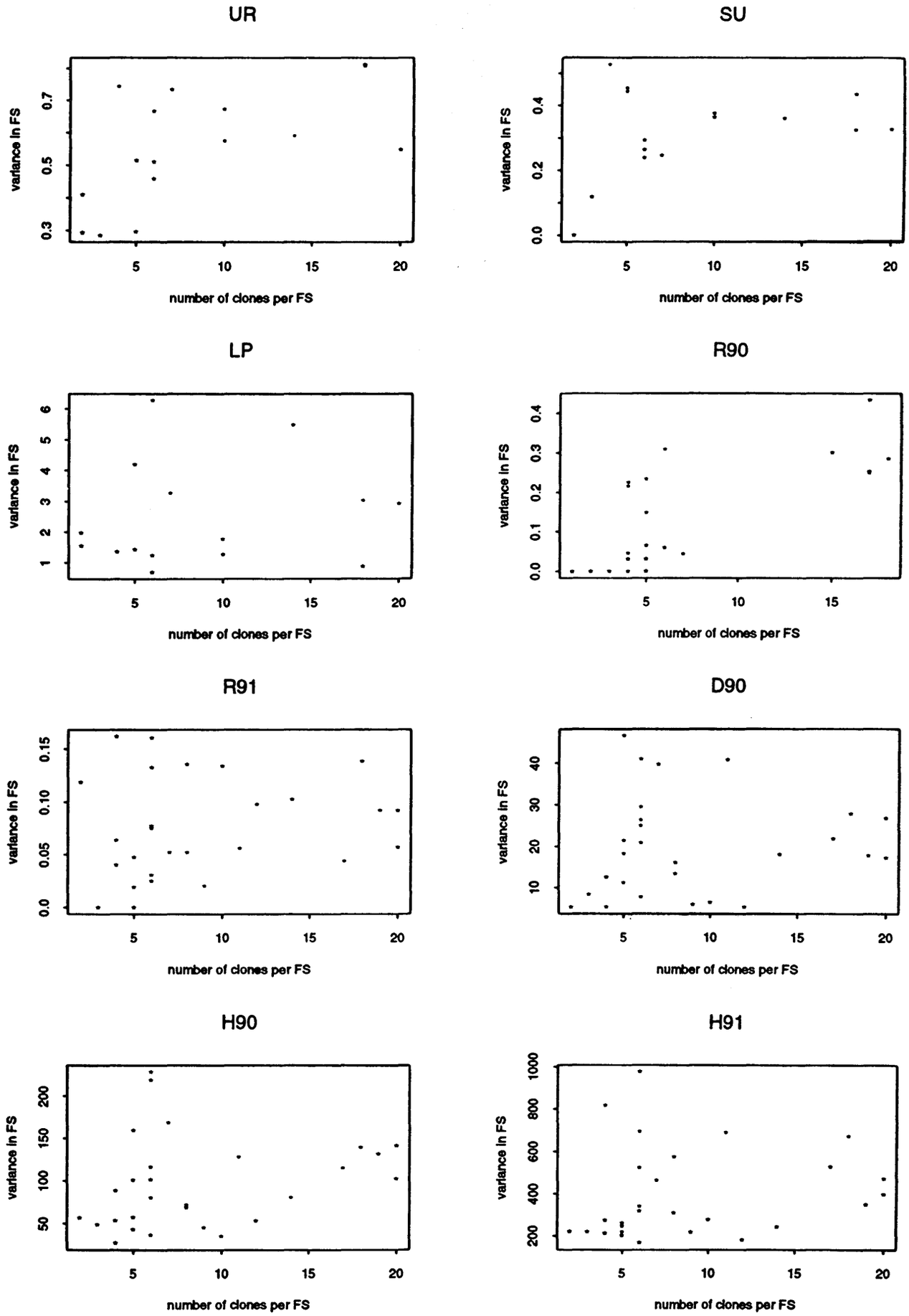

Figure 5. — Effect of the number of clones on the within FS family variances.

additive variance from half sib effects does not exceed additive variance from clonal effect. For resistance to race E1, there is no significant differentiation between the populations where the parents originated. Without further specific tests, it is impossible to explain the differentiation observed in the nursery trial for overall resistance to *M. larici-populina* and *M. allii-populina*. From an evolutionary point of view, natural selection in *P. deltoides* for partial resistance to rust in a geographic area where these particular rust species are absent, suggests some common genes of aggressiveness from one rust species to the other and/or some retention in *P. deltoides* of resistance genes from an earlier evolutionary context wherein these rust genes were encountered.

B) Heritabilities

Susceptibility of poplars to rusts is always reported to be a highly inherited trait. According to CHIBA and NAGATA (1972) broad sense heritability of susceptibility of *P. maximowiczii* to *Melampsora larici-populina* is 0.99. PINON and TEISSIER DU CROS (1976) observed a 0.68 correlation between females and half sib progenies of *P. nigra* L. in 2 successive years. Estimates are generally based on half sib family effect, so dominance is rarely reported, but additive variance seems largely to previal (GALLO et al, 1985) as observed in our laboratory and nursery tests. On the contrary, growth traits reveal significant dominance effect (PICHOT and TEISSIER DU CROS, 1989). Thus, broad sense and narrow sense estimates of partial resistance to *Melampsora* spp. are very close and clonal tests are adequate for additive estimates, but may lead to bias for correlation with other traits.

The natural range of *P. deltoides* includes genetically different populations however, thus estimates based on parents originating from various stands give overestimated heritabilities if stand effect is ignored. This is why we obtained higher estimations on half sib effect than on clonal (within full sib families) effect. Broad sense heritability of R91 based on parental effects exceeded 1.0. THIELGES and ADAMS (1975) estimated broad sense heritability as:

$$H^2_{bs} = (\sigma^2_F + \sigma^2_C)/(\sigma^2_F + \sigma^2_C + \sigma^2_E) \quad (5)$$

with: σ^2_F: female parent variance; σ^2_C: clone; σ^2_E: environment. But mother tree progenies were collected in different stands and stand effect was ignored. It might be more correct in that case to estimate:

$$H^2 = (4/3\ \sigma^2_C)/(4/3\ \sigma^2_C + \sigma^2_E), \quad (6)$$

thus removing both stand and family effects. Expected variance is:

$$E(\sigma^2_C) = \sigma^2_{genotypic} - \sigma^2_{half\ sibs} (= 3/4\ \sigma^2_A + \sigma^2_D). \quad (7)$$

$$\text{So, } H^2 = (\sigma^2_A + 4/3\ \sigma^2_D)/(\sigma^2_A + 4/3\ \sigma^2_D + \sigma^2_E).$$

Except for large σ^2_D, H^2 is very close to H^2 broad sense.

When reestimating H^2 in that manner, we obtained a mean value of 82% instead of 94%, which remains very high.

NELSON and TAUER (1987) evaluated H^2 of susceptibility of *P. deltoides* to *Melampsora* spp. at 0.38 for family mean within stand, which corresponds to only 0.24 for individual narrow sense H^2. Analysing an analogous provenance-progeny trial, THIELGES et al. (1989) related H^2 values from 0.48 to 0.54. IN this study, H^2 was estimated as:

$$H^2 = (4\ \sigma^2_F)/(4\ \sigma^2_F + \sigma^2_{FxR} + \sigma^2_E) \quad (8)$$

(FxR: familyxreplication interaction),

instead of:

$$H^2 = (4\ \sigma^2_F)/(\sigma^2_F + \sigma^2_{FxR} + \sigma^2_E)$$

as expected and which leads to $H^2 = 0.90$.

Heritabilities of rust traits observed in our laboratory test are high but not as high as we expected based upon our nursery estimations (R91) and on those previously mentioned. It is reasonable to think that environmental conditions could be more controlled, especially the regulation of the inoculum using a spore settling tower (SHARMA et al., 1980) which might effectively reduce the error variance.

C) Correlation between traits

C.1) Components of partial resistance to *Melampsora*

Number of uredia (UR) is correlated to latent period (LP) and size of uredia (SU), and correlation between these 2 last traits seems to be even higher. This agrees with the results reported by PRAKASH and HEATHER (1989) where a —0.68 correlation between LP and UR and —0.71, —0.79 between LP and the number of spores was found for the 2 races tested. SINGH et al. (1991) observed similar relationships between the components of partial resistance to wheat leaf rust caused by *Puccinia recondita*. According to our results, determination of the degree of resistance by UR, SU and LP in the laboratory appears to be effective for predicting of susceptibility in the nursery. PINON (1991) insists on the importance of selecting for both a long reproductive cycle of the rust on host tissue (extended latent period) as well as for a low level of susceptibility in partial resistance of clones (number and size of uredia). Components of resistance in the laboratory and susceptibility in the nursery are quantitatively distributed (PRAKASH and HEATHER, 1989; THIELGES et al., 1989), but CHALINE (1991) found a bimodal distribution, especially for latent period, in one *P. trichocarpa* TORR. and GRAY. family, suggesting that some genes may have major effects on quantitative susceptibility.

According to CHALINE (1991) correlation between number of uredia per disk after inoculation with *M. larici-populina* race E1 and race E2 varied from 0.3 in a *P. trichocarpa* full sib family to 0.5 in two *P. interamericana* families. Latent periods were better correlated (0.35 and 0.65, 0.80). Results suggest a common genetic basis for partial resistance to races E1 and E2, and these 2 races are very close from a qualitative point of view as well. Two races of *Melampsora medusae*, tested on 61 seedlings from one F1 progeny of *P. deltoides*, revealed significant effects of F1 plants and plant x race interactions for the 3 traits observed: latent period, uredia per disk and urediospores per sq. mm (PRAKASH and HEATHER, 1989). Thus, only some of the partial resistance to the first race was effective in the resistance to the second race.

Finally, our observations in the nursery 20 days after inoculation revealed the great difficulty of carrying out a large scale homogenous artificial inoculation *in situ*.

C.2) Growth and susceptibility to *Melampsora*

Some negative correlations between growth and susceptibility to rust are reported (CHIBA and NAGATA, 1972; FRIEND, 1981, in: NELSON and TAUER, 1987; THIELGES et al., 1989). In general, growth is reduced by rust attack and the negative correlations can be due to a secondary effect of rust infection such as a change in the allocation of photosynthate. Root growth is earlier and more reduced than stem growth (WANG and VAN DE KAMP, 1992). According to WILCOX and FARMER (1967), CHIBA and NAGATA (1972) and NELSON and TAUER (1987), diameter increment is especially affected by rust attack in previous year(s). In our experiment, there was no significant genetic correlation between susceptibility to *Melampsora* and growth in the nursery, where vigour was estimated with a low level of rust infection.

Conclusion — Consequences for Breeding Programs

Correlations between laboratory-nursery, nursery-plantation and juvenile-mature observations proved adequate for predicting the susceptibility of *Populus* spp. to various *Melampsora* rusts. Thus, early testing can be efficient if conducted properly.

However, it appears that both host and rust species are characterized by a great degree of variability. Many rust pathotypes have been identified for *Melampsora* species, and the number of virulence genes that can be detected depends on the variability of the poplar hosts tested. A gene-for-gene relationship must correspond to this qualitative aspect of virulence and susceptibility. It is clearly dangerous to select poplars on this type of resistance; partial resistance must be favored.

The great variability of the poplar genus allows breeders to improve productivity through both intra and inter specific hybridizations. The natural range of *P. deltoides* represents a broad spectrum of genetically different stands relative to susceptibility to *Melampsora spp.* (CELLERINO, 1976, for M. *allii-populina;* WILKINSON and VAN KRAAYENOORD, 1979, for *M. larici-populina;* THIELGES and ADAMS, 1975; PINON and TEISSIER DU CROS, 1976; NELSON and TAUER, 1987; THIELGES et al., 1989, for *M. medusae).* Effect of geographic origin has also been reported for *P. trichocarpa* (PINON, 1976; WANG and VAN DER KAMP, 1992): clones with coastal or moist-site origins tested more resistant. However, no geographic effect was found for *P. nigra* originating from southeastern France (PINON and TEISSIER DU CROS, 1976) or from Italy (CELLERINO et al., 1986), where geographic ranges are more restricted and where decades of poplar cultivation may have masked natural variation through hybridization among a few commercial clones.

Between species, great differences in susceptibility are noted. Compared to *P. nigra, P. trichocarpa* is more resistant to *M. allii-populina,* and *P. deltoides* to both *M. allii-populina* and *M. larici-populina.* On the other hand, *P. nigra* is more resistant to *M. medusae* than is *P. deltoides. P. maximowiczii* is relatively resistant to most *Melampsora* spp.. Unfortunately, initial results of testing interspecific hybrids suggests the dominance of susceptibility over partial resistance, especially in crosses of *P. deltoides* x *P. trichocarpa* (CHIBA, 1964, in: PINON, 1976; KRZAN, 1976; WILKINSON and VAN KRAAYENOORD, 1979). But there is actually a lack of information about the nature of genes involved in partial resistance to *Melampsora* rusts from one poplar species to another, and even among different intra-specific populations of poplar. In the same way, we know nearly nothing about the nature of the intra-specific poplar genes which control partial resistance to one *Melampsora* species or race versus another. This opens a wide field of research for a better and more efficient use of poplars as a natural resource. The objective would be to find pools of partial resistance with a wide spectrum of resistance, and to select poplar under artificial inoculations of various races of the species encountered in the geographic area for which poplars are selected.

In addition to the genetic improvement, other techniques can also reduce the possible damage caused by rust infection (PINON, 1984). Among them, the culture of several clones in a mixture or mosaic is very often suggested, but this often conflicts with habits of poplar growers and industry views on veneer production. Hence, educational programs and field demonstrations of these new practices are of main importance.

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Results of Sequoiadendron giganteum ([Lindl.] Buch.) Provenance Experiment in Germany

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Abstract

30 provenances of *Sequoiadendron giganteum* from the natural range and 2 single trees from Germany were tested with seedlings and cuttings on 3 different sites in the mountain region of southern Lower Saxony in elevation between 300m and 400m.

Mortality and height growth up to age 12 were evaluated. Average mortality ranged from 16% in Bad Grund to 60% in Uslar with considerable differences between provenances. There was no significant correlation between latitude, longitude, and elevation of origin and mortality.

Mean height growth ranged between 2.94 m and 3.38 m. Site and provenance effects are significant. There was no significant difference between seedlings and cuttings. The fastest growing provenances were Whitaker's Forest, Standard USA and Mountain Home. Slow growing provenances were Merced Grove, Grant Grove and Windy Gulch.

The best growth was found in provenances from the central and southern central portion of the natural range. Certain provenances were poor survivors and performers on most sites.

The results are discussed especially with regard to the results of FINS and possible inbreeding.

The experiments must be followed further before recommendations for seed import can be derived.

Key words: Sequoiadendron giganteum, provenance experiment, mortality, growth, geographic variation.

Zusammenfassung

30 Herkünfte von *Sequoiadendron giganteum* aus dem natürlichen Verbreitungsgebiet und 2 Einzelbaumbeerntungen aus Deutschland wurden als Sämlinge und Stecklinge auf 3 verschiedenen Standorten im südniedersächsischen Bergland in Höhenlagen zwischen 300 m und 400 m über NN. geprüft.

Ausfälle und Höhenwachstum bis zum Alter 12 wurden ausgewertet. Die durchschnittlichen Ausfälle je Herkunft reichten von 16% in Bad Grund bis zu 60% in Uslar. Es gab keine signifikanten Zusammenhänge zwischen geographischer Länge und Höhenlage des Herkunftsortes mit der Mortalität.

Die Mittelhöhen reichen von 2,94 m bis 3,38 m. Standort- und Herkunftseinflüsse sind signifikant. Es gibt keine signifikanten Unterschiede zwischen Sämlingen und Stecklingen. Die raschwüchsigsten Herkünfte sind Whitacker's Forest, Standard USA und Mountain Home. Langsamwüchsige Herkünfte sind Merced Grove, Grant Grove und Windy Gulch.

Die bestwüchsigen Herkünfte stammen aus dem zentralen und südlich-zentralen Teil des natürlichen Verbreitungsgebietes. Einige Herkünfte zeigen geringes Überleben und schlechtes Wachstum auf den meisten Standorten.

Die Ergebnisse werden besonders im Hinblick auf die Ergebnisse von FINS und mögliche Inzucht diskutiert.

Die Versuchsflächen müssen weiter verfolgt werden, ehe Empfehlungen für Saatgutimport gegeben werden können.

Introduction

Giant sequoia (*Sequoiadendron giganteum* (LINDL.) BUCH.) is the most massive living organism known and is second only to bristlecone pine (*Pinus aristata* ENGELM.) in verified