

# First Multisite Clonal Test of Wild Cherry (*Prunus avium* L.) in Belgium

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## Abstract

A 10-year-old multisite clonal test of 19 selected clones of wild cherry (*Prunus avium* L.) was planted in winter of 1989–90 at 12 sites in Belgium.

Observations were recorded for height at 1, 4 and 9 years after planting, for height increment between years 4 and 9, for girth at 10 years, for stem straightness at years 4 and 9 and for forking and anthracnosis susceptibility at 9 years.

Multisite broad-sense heritability of the clone means was generally higher than monosite heritabilities. The multisite heritability ranged from 0.65 for forking to 0.96 for anthracnosis susceptibility. Expected genetic gains were high for most of the traits, often greater than 7%. The tallest clones had a higher girth, a straighter stem and a lower susceptibility to anthracnosis but also tended to be more forked.

A significant clone x site interaction was observed for height, girth and susceptibility to anthracnosis but it had no real negative impact on selection gain, interaction affecting mainly the clones of medium performance.

*Key words:* *Prunus avium* L., genotypic heritability, phenotypic correlation, genotypic gains, clone, clone x site interaction.

## Résumé

Un test clonal de 10 ans composé de 19 clones répartis dans 12 sites a été planté au cours de l'hiver 1989-90 et fournit les premières estimations des paramètres génotypiques et des gains potentiels que l'on peut espérer d'une sélection clonale de merisier (*Prunus avium* L.) en Belgique.

Les caractères étudiés sont la hauteur à 1, 4 et 9 ans après plantation, l'accroissement en hauteur entre 4 et 9 ans, la circonférence à 10 ans, la forme à 4 et 9 ans, la fourchaison ainsi que la sensibilité à l'anthracnose à 9 ans.

L'héritabilité génotypique sur les moyennes à travers tous les sites est, quel que soit le caractère, élevée à très élevée et supérieure aux héritabilités sur les moyennes par site. Elle est au minimum de 0,65 pour la fourchaison à 9 ans et atteint 0,95 et 0,96 respectivement pour la circonférence et la sensibilité à l'anthracnose. Les gains génotypiques conventionnels calculés sur l'ensemble des sites sont, quel que soit le caractère, assez élevés, la plupart du temps supérieurs à 7% et intermédiaires par rapport aux gains génotypiques conventionnels observés dans l'analyse unisite. Les clones plus grands ont tendance à présenter une circonférence à 1,30m plus élevée, un fût plus droit et une plus faible sensibilité à l'anthracnose. Les clones présentant les plus gros diamètres de tige tendent également à être plus fourchus.

L'interaction clone x site est significative pour les caractères de hauteur, de circonférence et de sensibilité à l'anthracnose mais cette interaction n'a pas de réel impact sur la sélection compte tenu qu'elle agit essentiellement sur les clones de niveau moyen.

*Mots clés:* *Prunus avium* L., héritabilité génotypique, corrélation phénotypique, gain génotypique, clone, interaction clone x site.

<sup>1</sup> V1 = vitro-plants of 1 year ; V1R1 = vitro-plants of 1 year, transplanted for 1 year.

## Introduction

Wild cherry (*Prunus avium* L.) is a moderately fast growing species with a rotation of 50–70 years. Its high grade wood is used for panelling and cabinet-making. Interest in this species increased at the end of 1960's in Belgium and a few years later in France due to increased awareness about the utility of noble hardwoods (HUBERT, 1982).

Today, the forest policy of many European countries encourages the production of high quality precious broadleaved species on suitable sites.

In Wallonia (Southern Belgium) where 80% of Belgian forest is located, the value of wild cherry is acknowledged and plantations are encouraged.

During the last decade, there was a constant increase in forest area which includes plantations of wild cherry. Its basal area has increased, proportional to the total deciduous forest area, from 0.78% in 1984 to nearly 2.0% in 1996. Since the implementation of an intensive silviculture involving low plantation density, protection from game, weeds and pests, the number of trees planted per hectare is low. Therefore, a high quality genetic stock at the time of planting is required.

The tree improvement programme of this species, developed at the Research Centre for Nature, Forests and Wood (CRNFB) at Gembloux, was based on the selection of 'plus trees' with a view to create seed orchards. Two seed orchards have already been planted in Wallonia (Fenffe and Ciergnon). In addition, taking into account the delay to get seeds from these orchards and the possibilities of *in vitro* multiplication for this species, the CRNFB has created a multiclinal variety to quickly supply forest managers with high-grade wild cherry plants. For a few years, this multiclinal variety, composed of 20 to 30 plus trees chosen within the 119 wild cherries of superior phenotypes selected by the CRNFB, is available for forest managers through commercial nurseries.

To investigate the quality of this material in forest conditions, several clonal tests were established by JACQUES over a range of sites covering the natural wild cherry area of Wallonia, during the 1989–90 winter.

In this paper, the first results from these clonal tests are used to estimate genotypic parameters and possible gains from a clonal selection in wild cherry.

## Material and Methods

### Material and sites

Nineteen plus trees, samples from the 119 Wild cherries selected in Wallonia, were multiplied by *in vitro* culture and planted as 1-year-old plants (V1<sup>1</sup>) at 3 m x 3 m in 14 clonal tests distributed over the potential area of wild cherry in Wallonia during the winter of 1989–90. A completely randomised design with single tree plots was adopted. The number of ramets per clone and per site varied between 5 to 15, but mortality during the first years after planting has slightly reduced the number of trees.

Since natural wild cherry generally grows as isolated trees or clumps, the test plantation area did not exceed 0.22 ha.

Of the 14 clonal tests, 2 were abandoned due to game damage and the remaining 12 were retained for analysis. A more detailed description of the clonal tests is presented in CURNEL (2000).

#### Measurements

Total height (cm) was measured at 1, 4 and 9 years after planting (respectively H90/1, H93/4 and H98/9) and the height increment (cm/year) between the 4th and the 9th years was computed ( $\Delta H93-98$ ).

Girth (cm) was measured in 1999 (C99/10), 10 years after planting, at 1.30 m in each of the 12 clonal tests.

Stem straightness (Fo) was rated in 1993 (Fo93/4) and in 1998 (Fo98/9); 3 levels were used in 1993 (1 = quite straight; 2 = slightly crooked; 3 = moderately crooked, a major crook or a bushy aspect) and 5 in 1998, according to the number of crooks observed on plants (1 = quite straight up to 5 = quite twisted).

Forking was estimated using a subjective scale in 1998 (Fc98/9). The 5-levels scale took the presence or the absence of ramicon into consideration (1 = no fork, no bayonet up to 5 = badly forked).

The degree of defoliation due to anthracnosis (*Blumeriella jaapi* (Rehm.) V. Arx.), which is the main defoliation agent of wild cherry in Wallonia, was estimated 9 years after plantation (Déf98/9) to quantify the clone's susceptibility. Four levels were noted, ranging from 0 (no defoliation) to 3 (heavy defoliation).

#### Data analysis

The analysis of variance for each of the 12 clonal tests was done on a full random scheme according to NANSON (1970),

$$x_{ij} = m + \gamma_i + \varepsilon_{ij} \quad (A)$$

where  $x_{ij}$  is the observed phenotypic value of ramet  $j$  of clone  $i$  ( $i = 1, \dots, n$ ),  $m$  is the general mean,  $\gamma_i$  is the deviation to the general mean attributable to clone  $i$  and  $\varepsilon_{ij}$  is the residual deviation.

In our clonal tests, the number of ramets  $j$  per clone was variable ( $j = 1, \dots, r_i$ ) where  $r_i$  is the number of ramets for clone  $i$  in a given site depending on the number of trees still available in the field.

In the single-site analysis of variance, the clonal effect was tested against the residual (Table 1).

Table 1. – Single-site analysis of variance (random model).

Variation source	Degree of freedom	MS	F calc.	E(MS)
Clone	$n - 1$	A	A/B	$\sigma_\varepsilon^2 + r_0 \sigma_\gamma^2$
Residual	$r. - n$	B		$\sigma_\varepsilon^2$
Total	$r. - 1$			

$r_0$  is the weighted value of the number of repetitions per clone in the clonal test (SNEDECOR and COCHRAN, 1962).

$$r_0 = \frac{1}{n-1} (r. - \sum \frac{r_i^2}{r.}) \quad (B)$$

The multisite analysis of variance (11 sites for the forking, 12 for the other parameters) was done on a completely randomised design with sites considered as blocks (table 2).

Clonal and Site effects were tested using the clone mean of each site to circumvent the difficulty of having unequal numbers of ramets per clone and site. Procedure was done according to NANSON (1970):

Table 2. – Multisite analysis of variance on means per clone and per site (random model).

Variation source	Degree of freedom	MS	F calc.	E(MS)
Clone	$p - 1$	A	A/C	$\sigma_\varepsilon^2 + q \sigma_\gamma^2$
Site	$q - 1$	B	B/C	$\sigma_\varepsilon^2 + p \sigma_\beta^2$
Residual	$(p-1)(q-1)$	C		$\sigma_\varepsilon^2$
Total	$pq - 1$			

$$x_{ij} = m + \gamma_i + \beta_j + \varepsilon_{ij} \quad (C1)$$

where  $x_{ij}$  is the phenotypic observed mean of clone  $i$  in the clonal test  $j$ ,  $m$  is the general mean,  $\gamma_i$  is the deviation to the general mean attributable to the clone  $i$  ( $i = 1, \dots, p$ ),  $\beta_j$  is the deviation to the general mean attributable to the site  $j$  ( $j = 1, \dots, q$ ) considered here as block, and  $\varepsilon_{ij}$  is the random error.

The clone and site were considered as random effects.

Interaction effects were tested upon a cross classification (Table 3) according to NANSON (1970):

$$x_{ijk} = m + \gamma_i + \beta_j + (\gamma\beta)_{ij} + \varepsilon_{ijk} \quad (C2)$$

where  $x_{ijk}$  is the phenotypic observed value of ramet  $k$  ( $k = 1, \dots, r_i$ ) of clone  $i$  in the clonal test  $j$ ,  $m$  is the general mean,  $\gamma_i$  is the deviation to the general mean attributable to the clone  $i$  ( $i = 1, \dots, p$ ),  $\beta_j$  is the deviation to the general mean attributable to the site  $j$  ( $j = 1, \dots, q$ ),  $(\gamma\beta)_{ij}$  is the deviation to the general mean due to the interaction between the clone  $i$  and the site  $j$  and  $\varepsilon_{ijk}$  is the residual deviation.

Table 3. – Multisite analysis of variance on individual values (random model).

Variation source	Degree of freedom	MS	F calc.	E(MS)
Clone	$p - 1$	A	A/C	$\sigma_\varepsilon^2 + n_1 \sigma_{\gamma\beta}^2 + q n_1 \sigma_\gamma^2$
Site	$q - 1$	B	B/C	$\sigma_\varepsilon^2 + n_2 \sigma_{\gamma\beta}^2 + p n_2 \sigma_\beta^2$
Interaction	$(p-1)(q-1)$	C	C/D	$\sigma_\varepsilon^2 + n_3 \sigma_{\gamma\beta}^2$
Residual	$pq(n' - 1)$	D		$\sigma_\varepsilon^2$
Total	$pq - 1$			

The conditions needed to apply the variance analysis (normality and equality of variances) were supposed to be fulfilled.

The software MINITAB (1998) was used for the statistical analyses of data, according to GLM procedure (Global Linear Model) for the interaction test.

The GLM procedure is a general linear model based on regression approach. This procedure allows the calculation of the mean squares where the observations are unbalanced or missing. The GLM procedure takes these factors into account, calculating for each combination of factor levels (in our case, for each combination clone-site), not the arithmetic mean but the least squares mean.

The broad-sense heritabilities were estimated according to NANSON (1970):

$$h_G^2 = \frac{\sigma_\gamma^2}{\sigma_\gamma^2 + \sigma_\varepsilon^2 / r_0} \quad (D)$$

for the single-site genotypic heritabilities based on the clone means.

$$h_{Gi}^2 = \frac{\sigma_\gamma^2}{\sigma_\gamma^2 + \sigma_\varepsilon^2} \quad (E)$$

for the single-site genotypic heritabilities based on the individual values.

$$h_G^2 = \frac{\sigma_\gamma^2}{\sigma_\gamma^2 + \sigma_\varepsilon^2 / q} \quad (F)$$

for the multisite genotypic heritabilities based on the clone means of each site.

Where  $\sigma_{\gamma}^2$  = clonal variance,  $\sigma_{\epsilon}^2$  = residual variance.

The ratios  $\sigma_{\gamma\beta}^2 / \sigma_{\gamma}^2$  and  $\sigma_{\epsilon}^2 / \sigma_{\gamma\beta}^2$  were also computed, where  $\sigma_{\gamma\beta}^2$  is the variance due to clone x site interactions. The variances  $\sigma_{\gamma}^2$  and  $\sigma_{\epsilon}^2$  were estimated using the GLM procedure. These ratios constitute two ways to estimate the relative importance of the clone x site interaction according to MURANTY *et al.* (1996), MURANTY *et al.* (1998), SANTI *et al.* (1998).

Although individual broad-sense heritability is frequently cited in literature, the broad-sense heritability of clone means is much more useful for the estimation of genotypic gains.

The direct conventional genotypic gains were estimated, in relative value, according to NANSON (1988) for a selection differential  $i$  equal to 1 (selection of 38% of the clones tested) and for a normal distribution :

$$\Delta G (\%) = i h_G^2 CV_p \quad (G)$$

Where  $h_G^2$  is the genotypic heritability on the clone means and  $CV_p$  is the phenotypic coefficient of variation in relative value.

The genotypic and phenotypic correlations on clone means of each site were computed, in the multisite analysis, from the results of the analysis of variance/covariance according to NANSON (1988).

The indirect genotypic correlated gains in trait  $y$  for selection of trait  $x$ , in relative value, were estimated according to NANSON (1967), also for a selection differential  $i_x$  equal to 1:

$$\Delta G_{y/x} = i_x (r_p - e_x e_y r_E) CV_{p_y} \quad (H)$$

Where  $r_p$  and  $r_E$  are respectively the phenotypic and environmental coefficients of correlation between traits  $x$  and  $y$ ,  $CV_{p_y}$  is the phenotypic coefficient of variation of trait  $y$ ,  $e_x$  and  $e_y$  are the environmental coefficients of direction respectively of traits  $x$  and  $y$ .

## Results and discussion

### General means per site

The total height measured 9 years after planting (H98/9), varied from 343 cm to 796 cm (Table 4).

Difference in fertility between sites were also observed with the mean height increments between 1993 and 1998 ( $\Delta H_{93-98}$ ): the mean height increments per site varied from 40 cm/year to 86 cm/year with an overall mean of 55 cm/year. Measuring height increments theoretically allows a more accurate expression of the potential height growth of the clones, as they allow partial suppression of the impact of culture or laboratory conditions, which are generally stronger in the first years of plantation.

Mean girth per site was also variable, ranging from 12 cm to 30 cm, with an overall mean of 19 cm.

Substantial differences between sites could also be observed for forking (Fc) and stem straightness (Fo). In 1998, the forking (Fc98/9) value varied between 1.9 ( $\approx$  one ramicorn) and 3.1 ( $\approx$  one fork). In the same year, the stem straightness (Fo98/9) value varied between 2.4 and 3.0. So the clones, on average and in all sites, had at least one slight crook.

For susceptibility to anthracnosis (Déf98/9), it would be risky to compare the means between sites, as scoring took place at different dates. Nevertheless, it seems clear that no site was free from this disease.

### Analysis of variance for each site

Genotypic heritability (Table 5) was generally significant for height measured 1 year after planting (H90/1). At this development stage, it seems that the observed differences between clones could be attributed to the culture conditions experienced by the clones in the laboratory and nursery. This influence decreases, in general, with time and in forest conditions, where the effects of genotype are predominant. This seemed to be the case in our experience: in 1993 (H93/4), clonal differences were less significant and in 1998 (H98/9), these differences increased again and reflected more clearly the effects due to the genotypes being fully expressed under forest conditions (Figure 1).

The residual variance was, most of time, very high for the stem straightness (Fo) and forking (Fc). For this reason, the clone effect was rarely significant for these 2 characters.

Clonal effect was, on the other hand, often significant for girth (C99/10), susceptibility to anthracnosis (Déf98/9) and height increment ( $\Delta H_{93-98}$ ). Clonal effect was even very highly significant in all of the 12 sites for susceptibility to anthracno-

Table 4. – Trait-means values, heritabilities and genotypic gains over 12 sites (data are presented as means, minima and maxima per site).

Characters	Mean			$h_G^2$			$h_{Gi}^2$			$\Delta G_C (\%)$		
	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max
Height (age 1) H90/1, cm	100.5	68.3	127.1	0.60	0.00	0.86	0.26	0.00	0.49	12.2	0.0	20.8
Height (age 4) H93/4, cm	239.9	148.3	369.5	0.49	0.30	0.74	0.16	0.07	0.32	8.0	3.5	14.4
Height (age 9) H98/9, cm	516.4	342.9	796.1	0.64	0.37	0.83	0.28	0.11	0.52	10.9	5.2	22.0
Height increment $\Delta H_{93-98}$ , cm/yr	54.9	39.8	85.8	0.69	0.45	0.88	0.35	0.13	0.63	16.1	6.9	29.8
Girth (age 10) C99, cm	18.7	12.0	29.6	0.72	0.42	0.86	0.37	0.11	0.55	24.7	6.7	34.8
Forking (age 9) Fc98/9	2.5	1.9	3.1	0.27	0.00	0.58	0.08	0.00	0.19	6.0	0.0	12.9
Form (age 4) Fo93/4	1.65	1.3	2.3	0.16	0.00	0.48	0.04	0.00	0.13	3.1	0.0	10.1
Form (age 9) Fo98/9	2.7	2.4	3.0	0.44	0.06	0.74	0.16	0.01	0.35	7.9	1.1	18.7
Anthracnosis (age 9) Déf98/9	2.3	1.6	2.8	0.84	0.72	0.92	0.55	0.31	0.79	26.8	9.6	52.9

Table 5. – Broad-sense heritabilities on the means per clone ( $h^2_G$ ) and heritabilities on the individual values ( $h^2_{Gi}$ ), over 12 sites.

Character	$h^2$	Site											
		1	2	4	5	6	7	8	9	11	12	13	14
Height (age 1) H90/1	$h^2_G$	0.47*	0.47*	0.77***	0.81***	0.59**	0.36ns	0.70***	0.00ns	0.86***	0.82***	0.75***	0.54**
	$h^2_{Gi}$	0.13	0.19	0.38	0.43	0.21	0.09	0.29	0.00	0.49	0.43	0.33	0.16
Height (age 4) H93/4,	$h^2_G$	0.30ns	0.52*	0.43*	0.56**	0.57**	0.37ns	0.42*	0.39ns	0.74***	0.52*	0.63**	0.41ns
	$h^2_{Gi}$	0.07	0.22	0.12	0.18	0.21	0.11	0.12	0.11	0.32	0.16	0.22	0.10
Height (age 9) H98/9,	$h^2_G$	0.61**	0.50*	0.62**	0.76***	0.83***	0.37ns	0.76***	0.45*	0.56**	0.71***	0.69***	0.76***
	$h^2_{Gi}$	0.21	0.21	0.25	0.37	0.52	0.11	0.37	0.14	0.18	0.31	0.28	0.40
Height increment $\Delta$ H93-98,	$h^2_G$	0.68***	0.47*	0.77***	0.77***	0.88***	0.68***	0.85***	0.53*	0.45*	0.72***	0.72***	0.79***
	$h^2_{Gi}$	0.27	0.20	0.40	0.40	0.63	0.33	0.51	0.19	0.13	0.33	0.30	0.45
Girth (age 10) C99	$h^2_G$	0.42ns	0.44ns	0.78***	0.84***	0.79***	0.69***	0.86***	0.71***	0.78***	0.78***	0.74***	0.81***
	$h^2_{Gi}$	0.11	0.18	0.43	0.43	0.46	0.33	0.55	0.34	0.38	0.39	0.33	0.48
Forking (age 9) Fc98/9	$h^2_G$	0.58**	0.07ns	0.34ns	0.47*	0.24ns	0.14ns	0.50*	0.04ns	0.06ns	0.44*	0.00ns	0.41ns
	$h^2_{Gi}$	0.19	0.02	0.09	0.14	0.07	0.03	0.16	0.01	0.01	0.13	0.00	0.13
Form (age 4) Fo93/4	$h^2_G$	0.00ns	0.36ns	0.05ns	0.05ns	0.05ns	0.33ns	0.00ns	0.05ns	0.00ns	0.23ns	0.32ns	0.48*
	$h^2_{Gi}$	0.00	0.12	0.01	0.01	0.01	0.09	0.00	0.01	0.00	0.05	0.07	0.13
Form (age 9) Fo98/9	$h^2_G$	0.42ns	0.41ns	0.29ns	0.74***	0.67**	0.35ns	0.47*	0.72***	0.06ns	0.50**	0.23ns	0.39ns
	$h^2_{Gi}$	0.11	0.16	0.08	0.34	0.30	0.11	0.14	0.35	0.01	0.16	0.05	0.12
Anthracnosis (age 9) Def98/9	$h^2_G$	0.72***	0.73***	0.89***	0.86***	0.92***	0.83***	0.91***	0.74***	0.85***	0.92***	0.86***	0.84***
	$h^2_{Gi}$	0.31	0.42	0.63	0.53	0.72	0.52	0.65	0.37	0.49	0.69	0.79	0.53

\* = significant for  $\alpha = 0.05$ ; \*\* = significant for  $\alpha = 0.01$ ; \*\*\* = significant for  $\alpha = 0.001$ ; ns = non significant.

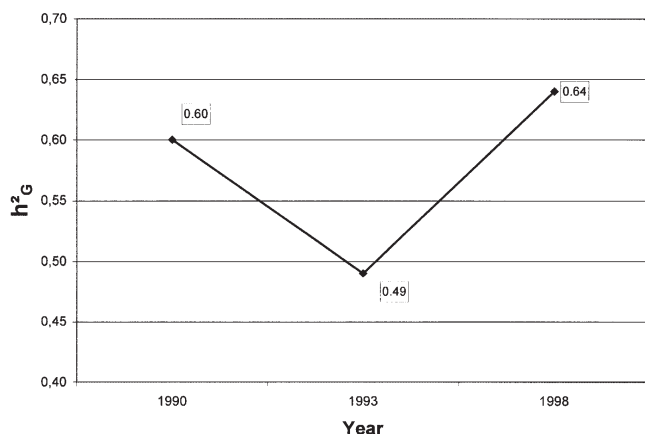


Figure 1. – Evolution of the mean of broad-sense heritabilities ( $h^2_G$ ) on the means per clone computed per site over time for height. (plantation in winter 1989–1990).

sis, which confirms the hypothesis of a strong clonal influence for this character (BOULET-GERCOURT, 1997).

#### Broad-sense heritability per site

##### Individual values ( $h^2_{Gi}$ )

The individual heritability at each site (Table 5) varied across sites for each character. Higher heritabilities were observed for the susceptibility to anthracnosis (Déf98/9), for girth (C99/10) and for height increment ( $\Delta$ H93-98). On average, the individual heritability was equal to 0.55 for the susceptibility to anthracnosis, 0.37 for girth and 0.35 for height increment (Table 4). Conversely, the individual heritabilities of forking and stem straightness were low with a mean of 0.08 for forking (Fc98/9) and 0.04 and 0.16 respectively for stem straightness in 1993 (Fo93/4) and 1998 (Fo98/9).

Individual heritability, for height, was on average equal to 0.26 in 1990 (H90/1), 0.16 in 1993 (H93/4) and 0.28 in 1998 (H98/9).

##### Means per clone ( $h^2_G$ )

For every character, the broad-sense heritability varied, at times, from one site to another (Table 5). Broad-sense heritability was generally high for the growth characters (height, height increment and girth). The broad-sense heritabilities per site for the height increment  $\Delta$ H93-98 were, most of time, higher than the observed heritabilities for the height measured in 1993 (H93/4) and in 1998 (H98/9).

Broad-sense heritability was the highest for the susceptibility to anthracnosis. It did not vary much from one site to another and reached at least 0.72.

For architectural characters such as stem straightness and forking, the genotypic heritability was low. It was even equal to zero or near zero in many sites. These low heritabilities can generally be attributed to the large residual variability that was observed for these characters. For stem straightness, an appreciable increase of the genotypic heritability between 1993 and 1998 was observed. This difference could be attributed to the difficulty in judging stem straightness at a young stage of development, to the subjectivity of the measures and to the change of assessor between 1993 and 1998.

##### Conventional genotypic gains site per site

##### Metric traits

For height in 1998 (H98/9), conventional genotypic gains (Table 4) varied from 5.2 to 22% with a mean of 10.9%. Nevertheless, a selection will be more efficient on height increment  $\Delta$ H93-98 (genotypic gains between 6.9% and 29.8% with a mean of 16.1%). For height, the gains seemed to be relatively larger in the less fertile sites.

Table 6. – Values of F-tests, genotypic heritabilities and conventional genotypic gains for the multisite analysis and the estimated percentage of the total variance due to clonal effect (%  $\sigma^2_\gamma$ ), to site effect (%  $\sigma^2_\beta$ ), to interaction effect (%  $\sigma^2_{\gamma\beta}$ ) and to residue (%  $\sigma^2_\epsilon$ ).

Character	F-test	$h^2_G$	$\Delta G_C$ (%)	% $\sigma^2_\gamma$	% $\sigma^2_\beta$	% $\sigma^2_{\gamma\beta}$	% $\sigma^2_\epsilon$
Height (age 1) H90/1	3.91***	0.74	7.9	7.28	19.45	12.85	60.42
Height (age 4) H93/4	5.53***	0.82	7.6	4.19	53.18	2.69	39.94
Height (age 9) H98/9	9.63***	0.90	10.3	8.15	57.72	3.41	30.72
Height increment $\Delta H93-98$	12.81***	0.92	15.2	12.88	47.91	4.53	34.68
Girth (age 10) C99	19.98***	0.95	25.3	19.22	38.99	3.14	38.65
Forking (age 9) Fc98/9	2.85***	0.65	6.7	3.88	13.24	3.11	79.77
Form (age 4) Fo93/4	2.90***	0.66	5.6	2.71	11.94	0.19	85.16
Form (age 9) Fo98/9	8.95***	0.89	11.4	11.23	6.09	2.31	80.37
Anthracosis (age 9) Def98/9	23.13***	0.96	25.3	33.21	21.58	13.36	31.84

The conventional genotypic gains were especially high for girth (C99/10): ranging from 6.7% to 34.8% with a mean of 24.7%.

#### Subjective traits

Genotypic gains for susceptibility to anthracosis were also high (between 9.6% and 52.9% with a mean of 26.8%) but it is important to note that conventional genotypic gains for arbitrary values are linked to a scale effect that influences the coefficient of variation.

It is not surprising that the genotypic gains were lower for the two architectural characters (stem straightness and forking), taking into account the low genotypic heritability previously observed : they did not exceed 8% on average and reached zero in many sites (Table 4).

In 1998, the genotypic gains varied from 0% to 12.9% (mean: 6.0%) for forking and between 1.1% and 18.7% (mean: 7.9%) for stem straightness.

#### Multisite analysis of variance

Once all the data were merged for a combined analysis, the clone effect was very highly significant (Table 6) for every character.

Table 7. – Estimation of the relative importance of the clone x site interaction by the  $\sigma^2_{\gamma\beta} / \sigma^2_\gamma$  and  $\sigma^2_\epsilon / \sigma^2_{\gamma\beta}$  ratios and F-test values for interaction component.

Character	$\sigma^2_{\gamma\beta} / \sigma^2_\gamma$	$\sigma^2_\epsilon / \sigma^2_{\gamma\beta}$	F-test
Height (age 1) H90/1	1.77	8.30	2.19***
Height (age 4) H93/4	0.64	14.82	1.36**
Height (age 9) H98/9	0.42	9.01	1.56***
Height increment $\Delta H93-98$	0.35	7.65	1.65***
Girth (age 10) C99	0.16	12.31	1.41***
Forking (age 9) Fc98/9	0.80	25.64	1.20ns
Form (age 4) Fo93/4	0.07	452.64	1.01ns
Form (age 9) Fo98/9	0.21	34.89	1.14ns
Anthracosis (age 9) Def98/9	0.40	2.38	3.12***

Site effect was also very highly significant for all characters studied. This demonstrates the importance of correctly choosing the plantation site and of precisely determining the potential of each site. Before establishing any forest plantation, it is highly recommended that an analysis is carried out, ensuring that those conditions necessary for optimal development are present.

The multisite analysis of variance also allowed for detection of a highly significant clone x site interaction at least for growth and susceptibility to anthracosis characters (Table 7). For stem straightness and forking, the conditions of the experiment did not allow detection of any interaction because of a high residual variance amounting to about 80% of the total variance.

#### Multisite broad-sense heritability

It is not surprising that the multisite broad-sense heritability (Table 6) was higher than the broad-sense heritability per site. The multisite method gives a more accurate discrimination of the clones in spite of a highly significant interaction for growth and susceptibility to anthracosis characters.

The multisite genotypic heritability was high for height. From 0.74 for H90/1, the heritability increased up to 0.90 for H98/9.

Compared with the analysis at individual sites, the multisite genotypic heritability for height increment ( $\Delta H93-98$ ) was slightly higher ( $h^2_G = 0.92$ ).

The multisite genotypic heritability was even higher for girth and susceptibility to anthracosis, where it was equal to 0.95 and 0.96, respectively.

For stem straightness and forking, the multisite genotypic heritability was also higher than the genotypic heritability at individual sites. It was for forking that the genotypic heritability was the lowest. However, it was still high with a value of 0.65.

#### Conventional genotypic multisite gains

The conventional genotypic multisite gains (Table 6) were intermediate compared to those observed in the analysis of individual sites. These gains were quite interesting, most of time higher than 7% and were especially high for height increment ( $\Delta H93-98$ ) and girth (C99/10), amounting to 15.2% and 25.3%, respectively. The high gain in susceptibility to anthrac-

nosis (25.3%) is probably influenced by the use of a subjective scale.

#### Clone x site interactions

Multisite analysis of variance showed an interaction at least highly significant for the characters of growth and for the susceptibility to anthracnosis (Table 7) and a low level for stem straightness and forking.

This confirms the hypothesis expressed by KREMER (1986) that characters such as vigour are more sensitive to the phenomenon of interaction than architectural characters.

The interaction study can be completed by the study of some ratios of variance (Table 7). The ratio  $\sigma_{\gamma\beta}^2 / \sigma_{\gamma}^2$  or interaction ratio is lower than 1, except for H90/1. Interaction effects are consequently always smaller than effects attributable to genotype.

The ratio  $\sigma_{\epsilon}^2 / \sigma_{\gamma\beta}^2$  was, on the contrary, always higher than 1. This ratio varied from 2.38 to 452.64. The residual variance was consequently higher, at times quite substantially, than the interaction variance.

These two observations confirm that the clone x site interaction represents the lowest part of the phenotypic variability as observed by MURANTY *et al.* (1996).

For the characters where the clone x site interaction was not statistically significant (stem straightness and forking), the ratio  $\sigma_{\epsilon}^2 / \sigma_{\gamma\beta}^2$  was very high (at least superior to 25). Consequently, the phenomenon of interaction could be in part masked by the strong residual variance (between ramets of a same clone).

This interaction ratio ( $\sigma_{\gamma\beta}^2 / \sigma_{\gamma}^2$ ) observed for height decreases as the plantation grows (from 1.77 in 1990 to 0.42 in 1998).

The interaction ratio observed for height increment  $\Delta H93-98$  (0.35) was lower than those observed by SANTI *et al.* (1998) for a height increment, between 2 and 7 years after the plantation, in 6 clonal tests (0.90) and was comparable to those (0.36) obtained by MURANTY *et al.* (1996), also for a height increment calculated between 2 and 7 years after plantation. The ratio  $\sigma_{\epsilon}^2 / \sigma_{\gamma\beta}^2$  for the same character (7.65) was comparable to those observed by MURANTY *et al.* (1996).

The interaction ratio for girth (C99/10) was, on the contrary, lower than those observed by SANTI *et al.* (1998) for a measure of girth realised 7 years after plantation ( $\sigma_{\gamma\beta}^2 / \sigma_{\gamma}^2 = 0.49$ ).

Studying the clones for the characters where the interaction is detectable allows the breeder for the determination of the relative stability of the clones. These observations are confirmed by KREMER (1986) according to whom the changes of ranking from one site to another occur most of time in the middle of the ranking, while the two extremities remaining often unchanged.

Finally, this study shows that, in spite of highly significant interactions for some characters, the corresponding genotypic gains stay at good levels. Therefore, these highly or very highly significant interactions are not necessarily important or harmful for selection.

#### Traits correlations

##### Phenotypic correlations

Coefficients of phenotypic correlation between height at different ages (Table 8) were positive and varied between 0.42ns and 0.80\*\*\*. The lowest value was observed between height 1 year after planting (H90/1) and height after 9 years (H98/9). Height in 1993, or 4 years after planting (H93/4), was the best indicator of the height at 10 years (H98/9).

Girth (C99/10) and height in 1998 (H98/9) are very well correlated (0.79\*\*\*). The correlation was even more significant between  $\Delta H93-98$  and C99/10 (0.87\*\*\*).

Clones with the highest girth tended to have more forks in 1998 ( $r_p = -0.51^*$ ).

Stem straightness seemed to be favourably correlated with the growth characters. The correlation was significant only for height ( $r_p = -0.54^*$  between H98/9 and Fo98/9) and for height increment  $\Delta H93-98$  (the phenotypic correlation between  $\Delta H93-98$  and Fo98/9 is equal to  $-0.62^{**}$ ).

The correlation between susceptibility to anthracnosis and height was significant and unfavourable (the phenotypic correlation equal to  $-0.55^*$  between H98/9 and Def98/9).

The correlation was even highly significant between the height increment  $\Delta H93-98$  and the susceptibility to anthracnosis (the phenotypic correlation between  $\Delta H93-98$  and Def98/9 is equal to  $-0.69^{**}$ ).

Height growth stops in July, before girth growth, when the fungal attack is not at its climax. So, it seems logical that the link between girth (C99/10) and susceptibility to anthracnosis was slightly more significant ( $-0.87^{***}$ ).

Table 8. – Multisite broad-sense heritabilities ( $h_G^2$ , in bold type on the diagonal), genotypic correlations ( $r_G$ , above the diagonal) and phenotypic correlations ( $r_p$ , beneath the diagonal) between the studied characters.  
 $r_p^* > 0.456$ ,  $r_p^{**} > 0.575$ ,  $r_p^{***} > 0.693$

	H90/1	H93/4	H98/9	Fc98/9	Fo93/4	Fo98/9	Def98/9	C99/10	$\Delta H93-98$
Height (age 1) H90/1	<b>0.74</b>	0.66	0.44	0.28	-0.40	0.05	-0.02	0.32	0.31
Height (age 4) H93/4	0.63	<b>0.82</b>	0.82	0.00	-0.57	-0.30	-0.09	0.39	0.66
Height (age 9) H98/9	0.42	0.80	<b>0.90</b>	0.21	-0.57	-0.58	-0.58	0.80	0.97
Forking (age 9) Fc98/9	0.22	0.03	0.18	<b>0.65</b>	0.34	0.34	-0.47	0.62	0.26
Form (age 4) Fo93/4	-0.34	-0.51	-0.47	0.22	<b>0.66</b>	0.50	-0.07	-0.22	-0.46
Form (age 9) Fo98/9	0.05	-0.26	-0.54	0.35	0.41	<b>0.89</b>	0.40	-0.45	-0.66
Anthracnosis (age 9) Def98/9	-0.01	-0.09	-0.55	-0.38	-0.04	0.36	<b>0.96</b>	-0.90	-0.73
Girth (age 10) C99	0.30	0.40	0.79	0.51	-0.21	-0.43	-0.87	<b>0.95</b>	0.89
Height increment $\Delta H93-98$	0.27	0.61	0.96	0.20	-0.38	-0.62	-0.69	0.87	<b>0.92</b>

Table 9. – Square matrix of direct conventional genotypic gains (%) (in bold type on the diagonal) and indirect correlated genotypic gains  $\Delta G_{y/x}$  (%).

x \ y	H90/1	H93/4	H98/9	Fc98/9	Fo93/4	Fo98/9	Def98/9	C99/10	$\Delta H93-98$
Height (age 1) H90/1	<b>7.9</b>	4.7	4.1	2.1	-2.4	0.5	-0.3	7.1	4.4
Height (age 4) H93/4	5.5	<b>7.6</b>	8.1	0.0	-3.5	-3.2	-2.0	9.1	9.4
Height (age 9) H98/9	3.9	6.5	<b>10.3</b>	1.7	-3.5	-6.6	-14.3	19.5	14.6
Forking (age 9) Fc98/9	2.2	0.0	1.9	<b>6.7</b>	1.8	3.2	-9.7	12.9	3.3
Form (age 4) Fo93/4	-3.1	-3.8	-4.7	2.2	<b>5.6</b>	4.8	-1.4	-4.7	-5.9
Form (age 9) Fo98/9	0.4	-2.3	-5.9	2.7	3.2	<b>11.4</b>	9.5	-11.1	-9.8
Anthracosis (age 9) Def98/9	-0.1	-0.7	-6.2	-3.8	-0.5	4.6	<b>25.3</b>	-22.8	-11.3
Girth (age 10) C99	2.8	3.1	8.4	5.0	-1.5	-5.3	-22.6	<b>25.3</b>	13.6
Height increment $\Delta H93-98$	2.9	5.3	10.1	2.0	-3.0	-7.6	-18.1	22.0	<b>15.2</b>

The other phenotypic correlations between the studied characters were not significant.

#### Indirect genotypic gains

Indirect gains between height and girth were positive, a selection on one of the two characters will involve a positive gain on the other character. Indirect gains in girth (Table 9) by selection of height at 9 and height increment  $\Delta H93-98$  were high, equal to 19.5% and 22%, respectively.

A selection on girth will lead to a slight loss in the quality of forking. Fortunately, it is always possible for the silviculturist to correct this default of forking by a correct leading of the deforking and pruning, at a certain cost however. In any case, these two operations are usually obligatory for a species such as wild cherry.

The positive indirect gain on stem straightness by selection on the height was at most 6.6% (between H98/9 and Fo98/9). This gain was superior by 1% by selection based on height increment (7.6% between  $\Delta H93-98$  and Fo98/9).

The selection of clones which are less susceptible to anthracosis will allow an indirect gain in height of about 6%. The gain on the height increment  $\Delta H93-98$  will be slightly higher (11.3%). The gain in girth will be very high (22.8%), greater than those observed with height.

#### Conclusions

1. Large differences in growth among sites were observed, which demonstrate the importance of choosing appropriate plantation site for wild cherry.

2. The single-site broad-sense heritabilities based on clone means varied substantially from one site to another, and were generally high for the growth characters (from 0.00 to 0.88) and for susceptibility to anthracosis (from 0.72 to 0.92) but were low for the architectural characters (from 0.00 to 0.74).

3. Multisite broad-sense heritabilities were high to very high; they were higher than those of single-site analysis and therefore allow more effective clonal selection. Clonal differences were always very highly significant. Very high for growth characters (from 0.74 to 0.95), the multisite broad-sense heritabilities can be qualified as high for the architectural characters (from 0.65 to 0.89). The multisite broad-sense heritability for the susceptibility to anthracosis was also very high (0.96) which reflects strong clonal differences.

4. Conventional genotypic multisite gains were intermediate to those observed per site for all the characters, which were higher than 7% most of time.

5. A highly significant clone x site interaction was detected for growth characters and for susceptibility to anthracosis. It seems that this interaction was mainly due to change in rankings for the clones of mean performances. The selection of the best clones seemed to be independent of this interaction.

6. Significant and favourable relations between stem straightness and height, and also between height and girth were found.

Significant and unfavourable relations between girth and forking, as well as growth characters and susceptibility to anthracosis were found. Fortunately, it is possible for the silviculturist to correct this defect by deforking and pruning. On the other hand, the selection of clones that present a lower susceptibility to anthracosis will be very efficient, as the multi-site broad-sense heritability is very high.

These results, which give the first indications of the improvement potential for wild cherry in Wallonia and which confirm some results obtained by SANTI *et al.* (1998), are encouraging and give an indication of real genetic improvement possibilities. The results confirm the apparent quality in forest conditions of the multiclonal variety created by the Research Centre for Nature, Forests and Wood and, due to the low influence of clone x site interactions, will also allow a reduction in the number of sites for future experiments with wild cherry.

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## Genetic Variation in Height, Branch and Needle Lengths of *Pinus sylvestris* L. from Siberia Tested in Alberta, Canada<sup>1</sup>

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### Summary

We performed the analysis of variance and covariance on height, branch length, height to branch length ratio, and needle length measurements from thirty open-pollinated families of *Pinus sylvestris* L. from Siberia, Russia. This progeny trial was replicated on three sites in central Alberta, Canada. At six years from seeds, there was statistically significant variation for height, branch length to height ratio and needle length. On individual test sites, values of individual-tree heritability for height ranged from 0.20 to 0.35, whereas values of heritability for family means ranged from 0.41 to 0.59. These low to moderate heritabilities suggest that a combination of family and within-family selection would be effective in improving height growth for this population on individual sites. Across sites, values of individual-tree heritability for height ranged from 0.03 to 0.06, whereas values for heritability of family means ranged from 0.17 to 0.29. These low heritabilities across sites were due to high genotype by environment (GE) interaction. Analysis showed that 87% to 99% of the GE interaction was due to lack of genetic correlation among sites. Heterogeneity of the genetic variance among test sites contributed 1% to 13% of the GE interaction. This shows that families in this population of Scots pine are not broadly adapted and are therefore suitable only for a site-specific breeding programme. The paper also presents and discusses results of other traits with emphasis to breeding Scots pine for production of Christmas trees, which is the main use of this species in North America.

*Key words:* *Pinus sylvestris*, genetic variation, heritability, genetic correlation, Siberia.

### Introduction

Scots pine (*Pinus sylvestris* L.) is an important Christmas tree species in parts of Canada and the United States (WRIGHT

and BULL, 1963; GIERTYCH, 1991; VAN HARVERBEKE and GERHOLD, 1991). A feasibility study in Alberta showed that Scots pine and white pine were the most valued and preferred Christmas tree species in the province (NEEDHAM et al., 1991). An economic feasibility study by KNOPF and WALL (1992) in Saskatchewan showed that Christmas tree production was a risky business, because of strong market competition and a short marketing period. However, the risk would be less by reducing the time required to raise the trees to marketable size. Thus, KNOPF and WALL (1992) concluded that because of its fast growth, Scots pine would make a better Christmas tree investment than balsam fir, white spruce and white pine, the three other species considered in the study.

A good Christmas tree is one with short internodes and a dense crown that does not expose the main stem (HAWBOLDT, 1958). This, however, may apply in places where Scots pine produces more than one internode per growing season. Where it produces only one internode per growing season, short internodes may be equated with slow growth, thus delaying return on investment relative to alternative species. In this case, longer internodes may be desirable to accelerate growth with quality characteristics being met through selection for crown characteristics such as many branches per whorl, acute branch angle, more and longer needles. Therefore, we need to develop progenies of Scots pine that combine both adequate growth to ensure early return on investment, and good qualities of Christmas trees.

Previous studies showed that Scots pine provenances from Russia were more adapted to Canadian environment than provenances from other areas (KASPER and SZABO, 1969; SOOS and BROWN, 1970; KLEIN, 1971; TEICH and HOLST, 1970). Russian provenances survived and grew better than jack pine (KASPER and SZABO, 1969; TEICH and HOLST, 1970), ponderosa pine (KASPER and SZABO, 1969), and lodgepole pine (DHIR et al., 1989). Better adaptation of Russian provenances in the Canadian Prairies can be attributed to climatic similarities between central Russia and the Canadian Prairie Provinces (GIERTYCH, 1991).

In 1988, the Alberta Forest Service initiated a progeny study of Russian Scots pine. The objective was to develop a regionally adapted Scots pine for Christmas trees and possibly timber

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