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Genetic Parameter Estimates for Resistance to Rust (*Cronartium quercuum*) Infection from Full-Sib Tests of Slash Pine (*Pinus elliottii*), Modelled as Functions of Rust Incidence[1])

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Abstract

Data from 171 slash pine progeny tests, incorporating over 700 different families from more than 2100 first-generation parents and approximately 170000 trees, were used to estimate variance and covariance components by Restricted Maximum Likelihood (REML) in both single-site and paired-site analyses. From these REML estimates, genetic parameters (heritabilities, proportion of dominance, type B genetic correlations, and age-age genetic correlations) were estimated for resistance to fusiform rust infection at 4 to 15 years of age. Predictive models were developed for biased (single-site) heritability, unbiased (paired-site) heritability and the type B genetic correlation. Biased heritability exhibited a maximum of 0.20 at an average rust infection of 72%. Unbiased heritability estimates from paired-site analyses increased linearly with increasing average rust infection in the tests; however, in very few test pairs did the average rust infection exceed 75%, and

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extrapolation beyond 67% would be unreliable. Although genotype-environment interaction was present, it was of little consequence except at low rust infection levels. The proportion of dominance variance (when compared to the total phenotypic variance) was not related to rust infection levels or age, and across all tests and ages averaged 0.087 and 0.053 in single-site and paired-site analyses respectively. Since dominance variance was small relative to additive variance, except when rust infection levels were low, it was considered to be of little practical importance. Test age was not a significant factor affecting any genetic parameter examined, and all age-age genetic correlations were near +1.0. The results endorse the current breeding strategy, which is based on recurrent selection for general combining effects, and stresses the importance of restricting improvement efforts to sites with moderate to high rust infection levels.

Key words: Fusiform rust, genetic parameters, dominance, genotype-environment interaction, disease resistance, predictive models, *Pinus elliottii.*

FDC: 232.11; 165.4; 443; 165.53; 174.7 *Pinus elliottii.*

Introduction

The most serious disease affecting slash (*Pinus elliottii* ENGELM. var. *elliottii*) and loblolly pines (*P. taeda* L.) within their natural ranges is fusiform rust (WALKINSHAW and ANDERSON, 1988; WALKINSHAW and ROLAND, 1990). Fusiform rust is caused by the fungus *Cronartium quercuum* (BERK.) MIYABE ex SHIRAI f. sp. *fusiforme*. Of the commercially important *Pinus* species grown in the southern United States, slash pine is one of the most susceptible to fusiform rust (SCHMIDT *et al*., 1981), and the economic impacts of the disease are substantial. Yield losses result from mortality and stem breakage (following the formation of stem-girdling galls), reduced marketability, and decreased growth rates (SCHMIDT *et al*., 1981; HODGE and WHITE, 1986; WALKINSHAW and ROLAND, 1990). Annual losses in commercial slash pine and loblolly pine (*P. taeda* L.) stands have been estimated at 562 million board feet (1.3 million m^3) of sawtimber, and 194 million cubic feet (5.5 million m^3) of growing stock (PHELPS and CZABATOR, 1980), and have been estimated to cost approximately 9 million and 26 million US dollars annually in slash and loblolly pine, respectively, grown in Florida, Georgia, South Carolina, North Carolina and Virginia (ANDERSON *et al.,* 1986).

Although useful levels of genetic variation in resistance to fusiform rust infection are known to exist within slash pine (DINUS and GRIGGS, 1975; GODDARD *et al.*, 1975; SCHMIDT *et al.,* 1981; WALKINSHAW and BEY, 1981), and substantial genetic gains have been achieved in the resistance of slash pine to fusiform rust (SLUDER, 1986; HODGE *et al.* 1989, 1990), relatively little concerning the quantitative genetics of rust resistance in slash pine has been published. ROCKWOOD and GODDARD (1973) obtained individual-tree heritability estimates ranging from 0.035 to 0.262 in 10 progeny tests with an average of 0.167, SOHN and GODDARD (1979) reported heritabilities in the range 0.1 to 0.4 in 8 tests with an average of 0.25, and HODGE *et al.* (1990) reported realised heritabilities between 0.3 and 0.4. For non-additive variance, KRAUS (1973) found a significant amount of dominance variance in the number of rust galls observed per tree in 2 factorial tests.

In other *Pinus* species there is likewise relatively little published information on the quantitative genetics of disease resistance. SLUDER (1988, 1993) estimated the family-mean heritability of fusiform rust resistance in a single loblolly pine test to be 0.69 at both 10 and 15 years of age, and found that the amount of dominance variance was small relative to additive variance. In 9 progeny tests of *P. radiata* involving resistance to *Dothistroma* needle blight, the individual-tree heritability of resistance varied between 0.13 and 0.40 (average of 0.26), and dominance was very small compared with additive variance (CARSON, 1989). In a single test of *P. muricata*, individual-tree heritability of resistance to *Dothistroma* needle blight was estimated to be 0.29 (ADES *et al.,* 1992).

There is evidence that genetic parameter estimates may be influenced by the mean infection level. When rust incidence is near either extreme (0% or 100% infection) there is little genetic or phenotypic variation, and consequently genetic parameters will be poorly estimated. Some authors have reported a positive association between the heritability of rust resistance in slash pine and the mean level of rust infection (ROCKWOOD and GODDARD, 1973; SOHN and GODDARD, 1979; WHITE and HODGE, 1989, p. 185). Genotype-environment interaction (GxE) of rust resistance is not thought to be important in slash pine, even thought statistically significant in some cases (GODDARD and SCHMIDT, 1979). Nevertheless family-mean correlations between 2 wind-pollinated tests with reasonable levels of rust infection (over 20%) were higher when both tests had similar levels of rust infection (SCHMIDT and GODDARD, 1971; SOHN *et al*., 1975). Therefore it appears that genetic parameter estimates must be considered in conjunction with the abundance and virulence of the pathogen as measured by the average infection level in the stand.

This paper uses data from 171 separate full-sib tests of slash pine established by the Cooperative Forest Genetic Research Program (CFGRP) based at the University of Florida, to: i) provide reliable genetic parameter estimates for rust resistance in slash pine, and ii) investigate the relationship between the mean incidence of rust in a test and the genetic parameter estimates. The genetic parameters included in this study are biased (single-site) and unbiased (paired-site) estimates of heritability, the proportion of dominance, type B genetic correlations which are an inverse measure of GxE (BURDON, 1977), and age-age genetic correlations.

Materials and Methods

Full-sib progeny test data

First-generation slash pine parents used by the CFGRP in full-sib matings originated from 2 separate phases of mass selection: the first (1956 to 1963) concentrated on stands with a low incidence of fusiform rust, while the second (in the early 1970s) was restricted to stands where the average fusiform rust infection exceeded 70% (HENDRICKSON, 1976). Some 2500 and 550 phenotypically superior trees were selected in the first and second phases, respectively. The second phase of mass selection, of "rust-free" trees in high rust-incidence areas, was necessary because relatively few of the initial first-generation parents proved resistant to fusiform rust (GODDARD, 1980). The CFGRP established over 200 replicated full-sib progeny tests between 1966 and 1989, which involve: i) factorial matings amongst the initial first-generation trees, ii) diallel or factorial matings among only the better-quality initial first-generation trees, and iii) factorial crosses using the rust-free trees as male parents and the better initial first-generation trees as female parents.

As of April 1994, 171 different full-sib tests were old enough (> 3 years old) to be included in this study. These tests are mainly in Florida, Georgia, and Alabama, but a few are in Mississippi and South Carolina. All tests were established in randomised complete block designs with 3 to 12 blocks (mean = 5.4 blocks), and each full-sib family was represented by plots of 5 to 10 trees (mean = 6.9 trees) arranged as either row-plots or non-contiguous plots. In any one test there were between 6 and

86 full-sib families (mean = 30.6 families), which were derived from crosses amongst 6 to 47 parents (mean = 18.2 parents). In total the tests included progeny from over 700 parents (165 rust-free parents), represented by over 2100 different full-sib families (550 families involving rust-free parents), and approximately 170000 individual trees.

The presence (score = 100) or absence (score = 0) of rust infection was assessed visually on each tree at approximately three-year intervals between 4 and 15 years of age, with most individual tests assessed at least twice. Data were grouped into 4 3-year age classes, centred at 5, 8, 11 and 14 years, and each of the 362 test-age class combinations was treated as a separate data point. Tests were also grouped into 5 classes based on the mean level of rust infection in the test (0% to 20%, 20% to 40%, …).

Estimation of variance components

There was considerable imbalance within this set of full-sib data as a result of incomplete mating designs, unequal representation of parents in tests, and mortality. Restricted maximum likelihood (REML) estimation (PATTERSON and THOMPSON, 1971) is generally considered to be a better choice in such circumstances (SEARLE *et al.*, 1992). GAREML, a computer program developed by HUBER (1993) which utilises GIESBRECHT's algorithm (1983) to provide REML estimates, has been shown to provide variance component estimates with desirable properties when applied to data sets with the amount and type of imbalance commonly found in genetic tests of forest trees (HUBER, 1993, p. 82). Therefore GAREML was used to analyse the individual-tree rust scores from these 171 full-sib slash pine tests.

REML variance component estimates were obtained from i) single-site analyses, and ii) analysis of connected pairs of tests, i.e., test pairs with a least 5 common parents. All possible connected test pairs were analysed, utilising data from all ages. However data of different ages obtained from the same test were never analysed together, thereby avoiding the potential problem of non-independent error terms due to environmental correlation within a single site (HODGE and WHITE, 1992). When test pairs were constructed using data from 2 tests measured at the same age, the REML estimates were variance components; however when the pair of tests were of different age the estimates obtained were considered to be covariance components. This method of analysis forced variance and covariance components to be non-negative, and may upwardly bias the parameter estimates compared with ANOVA estimators which are inherently unbiased (HUBER, 1993; SEARLE *et al.,* 1992). However, when HUBER (1993) simulated 1000 data sets with known variance components and varying levels of imbalance, the bias associated with the use of REML to analyse individual tree data was shown to be small. Also, DIETERS *et al.* (1995) used REML in this same manner to estimate variance and covariance components for tree volume from the same full-sib tests reported in this study of rust resistance. The heritability estimates obtained by DIETERS *et al.* (1995) were consistently lower than previous ANOVA-based estimates from half-sib tests (HODGE and WHITE, 1992). This suggests that any potential bias would be small.

For binominal traits the unit of analysis can be plot means, possibly combined with a transformation such as arcsin or logistic (SOHN and GODDARD, 1979; DE SOUZA, 1990; DE SOUZA *et al.,* 1991), because according to the central limit theorem such means are approximately normally distributed (MENDENHALL *et al.*, 1990, p. 325). However as one of the ultimate goals of this work is the prediction of genetic gain from within-family selection, estimates of the individual heritability and the within-plot variance are required, and these can only be determined by using individual observations as the unit of analysis. There is evidence to support the contention that REML analyses using individual observations of binomial traits will yield satisfactory results: i) REML estimation has been shown to be relatively robust to violations of the underlying normality assumptions (BANKS *et al.,* 1985; WESTFALL, 1987), ii) BANKS *et al.* (1985) using categorical data (with 2 to 6 classes) generated from an underlying normal distribution, demonstrated that REML estimation was acceptable, at least in terms of heritability estimates, iii) in simulation studies using REML estimation, the use of individual observations has been shown to be generally superior to the use of plot means as the unit of analysis (HUBER, 1993), iv) results from previous studies have demonstrated that using an arcsin transformation of the plot means does not greatly affect the relative size of variance component estimates (ROCKWOOD and GODDARD, 1973; SOHN and GODDARD, 1979), and v) the approximate variance of heritability estimates, which depends on underlying normality assumptions, was found to be closely rated to empirical estimates of variance even when individual binomial rust scores were used in the analysis (DIETERS, 1994; DIETERS *et al.,* 1995). Therefore, for these reasons and to avoid the problems of back-transformation, all analyses were conducted using untransformed individual rust scores.

Threshold theory suggests that heritability estimates calculated from binominal data should be transformed to a "true", underlying normal scale (ROBERTSON and LERNER, 1949; DEMPSTER and LERNER, 1950; VAN VLECK, 1972; MCGUIRK, 1989; MANTYSAARI *et al*., 1991). However, this theory requires that there is only one causal agent affecting the expression of the trait (ROBERTSON and LERNER, 1949), and assumes the effects of gene substitutions are individually small and strictly additive (DEMPSTER and LERNER, 1950). In the case of rust resistance in slash pine, neither of these conditions appears to be met: there is considerable variation in the way slash pine reacts to infection by fusiform rust (WALKINSHAW and ANDERSON, 1988; DE SOUZA *et al.,* 1991); the pathogen differs in virulence between isolates (WALKINSHAW and BEY, 1981; WALKINSHAW and ROLAND, 1990); the observed incidence is a function of abundance and virulence of the pathogen; and this study provides evidence for the existence of dominance genetic variance influencing rust resistance. Even though it is common to apply this transformation when these conditions are not met (MCGUIRK, 1989), the transformation was not able to remove the considerable variation observed between individual heritability estimates. Therefore, it was considered to be more appropriate to model the untransformed (binominal) heritability estimates. If necessary, the transformation can be applied to any models developed.

Linear model

The most complex model used was that for paired-site analyses of factorial tests, while all other models can be thought of as a subset of this model. This model was:

$$y_{ijklmn} = \mu + t_i + b_{ij} + set_n + f_k + m_l + tf_{ik} + tm_{il} + fm_{kl} + tfm_{ikl} + p_{ijkl} + e_{ijklm}$$

where, y_{ijklmn} is the m^{th} tree in the kl^{th} family in the n^{th} set and j^{th} block of the i^{th} test,

μ is the population mean,

t_i is the random effect of the i^{th} test environment, $E(t_i) = 0$ and $Var(t_i) = \sigma^2_t$,

b_{ij} is the random effect of the j^{th} block in the i^{th} test, $E(b_{ij}) = 0$ and $Var(b_{ij}) = \sigma^2_b$,

set_n is the random effect of the n^{th} disconnected set of full-sib families, $E(set_n) = 0$ and $Var(set_n) = \sigma^2_s$,

f_k is the random effect of the k^{th} female, $E(f_k) = 0$ and $Var(f_k) = \sigma^2_{gca}$,

m_l is the random effect of the l^{th} male, $E(m_l) = 0$ and $Var(m_l) = \sigma^2_{gca}$,

fm_{kl} is the random effect of the interaction between the k^{th} female and the l^{th} male, $E(fm_{kl}) = 0$ and $Var(fm_{kl}) = \sigma^2_{sca}$,

tf_{ik} is the random interaction between the i^{th} test and k^{th} female, $E(tf_{ik}) = 0$, and $Var(tf_{ik}) = \sigma^2_{tgca}$,

tm_{il} is the random interaction between the i^{th} test and l^{th} male, $E(tm_{il}) = 0$, and $Var(tm_{il}) = \sigma^2_{tgca}$,

tfm_{ikl} is the random interaction between the i^{th} test and kl^{th} family, $E(tfm_{ikl}) = 0$, and $Var(tfm_{ikl}) = \sigma^2_{tsca}$,

p_{ijkl} is the random effect of the $ijkl^{th}$ plot, $E(p_{ijkl}) = 0$ and $Var(p_{ijkl}) = \sigma^2_p$, and e_{ijklm} is the random effect within the $ijkl^{th}$ plot, $E(e_{ijklm}) = 0$ and $Var(e_{ijklm}) = \sigma^2_w$.

In this model it was assumed that there was no covariance between the random effects in the model, that variances due to the female and male effects were equal, and that the female- and male-by-environment interactions were the same. GAREML produces one estimate of σ^2_{gca} and σ^2_{tgca} by pooling the estimates from the male and female parents, and when there are disconnected sets, all subsequent terms in the model are nested within sets. Note also, that where different-aged test measurements were included in a paired-site analysis, all variance components defined above were viewed as age-age covariance components.

In the case of diallel tests, individual parents are used as both males and females in the crosses. By assuming the absence of any reciprocal effects (i.e. that it does not matter whether a parent is used as a male or as a female), it is possible to estimate the variance components for gca and gca-by-test location (GRIFFING, 1956). Thus, when analysing diallel tests the terms f_k, m_l, tf_{ik} and tm_{il} in the above linear model were replaced by g_k, g_l, tg_{ik} and tg_{il} respectively for the general combining ability of the k^{th} (or l^{th}) parent, and fm_{kl} was replaced by s_{kl} for the specific combining ability. Finally, when conducting single-site analyses, the models used for factorial and diallel experiments were as described above, except that all terms involving main and interaction effects due to the i^{th} test were dropped from the model.

Genetic parameter estimates

Four types of genetic parameters were estimated: heritability (h^2 and h^2_b from paired-site and single-site analyses respectively), the proportion of dominance (d^2 and d^2_b), type B genetic correlation (r_B), which measures GxE, and genetic correlation between ages (r_g). Type B genetic correlations were estimated only from paired-site analyses.

From each paired-site analysis, with both tests measured at the same age, heritability and the proportion of dominance were estimated as:

In the absence of epistasis and maternal effects, with non-inbred parents, these estimates of narrow sense heritability (h^2) and the proportion of dominance (d^2) are unbiased, because i) σ^2_{gca} is an estimate of 1 quarter of the additive genetic variance, σ^2_A (COCKERHAM, 1963; WRIGHT, 1985; COTTERILL *et al.,* 1987), ii) σ^2_{sca} is an estimate of 1 quarter of the dominance genetic variance, σ^2_D (COCKERHAM, 1963; WRIGHT, 1985; COTTERILL *et al.,* 1987), and iii) the denominator in equations [1] and [2] is an estimate of the total (relevant) phenotypic variance, σ^2_p. Note that the ratio of additive to dominance variance (σ^2_A/σ^2_D) can be estimated by dividing h^2 by d^2. Details of other assumptions required in the genetic interpretation of the GCA and SCA variance components are described by COCKERHAM (1963) and WRIGHT (1985).

When variance components are estimated from single-site analyses of progeny tests, it is impossible to separate the genetic (both additive and dominance genetic effects) from the genetic-by-environment interaction. Therefore estimates of variance due to general and specific combining abilities (from single-sites) are biased upward (COMSTOCK and MOLL, 1963), since the estimates include $\sigma^2_{gca} + \sigma^2_{tgca}$, and $\sigma^2_{sca} + \sigma^2_{tsca}$ respectively. Thus for single-sites we can define the variance components attributable to the general and specific combining abilities as: $\sigma^2_{GCA} = \sigma^2_{gca} + \sigma^2_{tgca}$, and $\sigma^2_{SCA} = \sigma^2_{sca} + \sigma^2_{tsca}$.

Thus upwardly biased heritability (h^2_b) and the corresponding proportion of dominance (d^2_b) were calculated separately for each measurement of all 171 progeny tests using the equations for paired-sites given above (*Equations [1]* and *[2]*, but dropping all variance components involving interactions with tests, and substituting σ^2_{GCA} and σ^2_{SCA} for σ^2_{gca} and σ^2_{sca} respectively.

For all pairs of tests where both tests were measured at the same age, a Type B additive genetic correlation (BURDON, 1977), termed r_B, was estimated in the following manner:

$$r_B = \frac{\sigma^2_{gca}}{(\sigma^2_{gca} + \sigma^2_{tgca})} \qquad [3]$$

This measures the degree of GxE at the additive level ($r_B = 1$ implies no GxE, while $r_B = 0$ implies that there is no consistent rust resistance performance of families across test sites).

From the results of the paired-test analyses it was also possible to estimate additive genetic correlations, in this case age-age genetic correlations, from each test-pair where both tests were measured at age_1 and age_2:

$$r_{g(age1,age2)} = \frac{\sigma_{gca(age1,age2)}}{\sqrt{(\sigma^2_{gca(age1)}) \cdot (\sigma^2_{gca(age2)})}} \qquad [4]$$

where the additive genetic covariance between two ages ($\sigma_{gca(age1,\ age\ 2)}$) is the quadratic average covariance for that test-pair, i.e.,

$$h^2 = \frac{4\sigma^2_{gca}}{(2\sigma^2_{gca} + \sigma^2_{sca} + 2\sigma^2_{tgca} + \sigma^2_{tsca} + \sigma^2_p + \sigma^2_w)} \qquad [1]$$

$$d^2 = \frac{4\sigma^2_{sca}}{(2\sigma^2_{gca} + \sigma^2_{sca} + 2\sigma^2_{tgca} + \sigma^2_{tsca} + \sigma^2_p + \sigma^2_w)} \qquad [2]$$

$$\sqrt{\sigma_{gca_{(age1,age2)}} \cdot \sigma_{gca_{(age2,age1)}}} \quad [5]$$

and $\sigma^2_{gca(age1)}$ and $\sigma^2_{gca(age2)}$ are estimated from the same test pair where data are the same age in both tests. This requires both tests to have been measured at both age_1 and at age_2.

Linear regression analysis was used in an attempt to develop predictive equations for each of the genetic parameters described above, using mean rust incidence of each test, and various transformations and combinations of mean rust incidence as regressors. Individual estimates of the genetic parameters were not weighted in any manner because it proved to impossible to identify any test parameter(s) that were closely related to the quality of the data. All final models were required to be significant at p = 0.0001 (F-test) with no substantial lack of fit, to have all independent variables significant at p = 0.01 (t-test), and to be biologically plausible across the entire range of possible regressors. If such models could not be identified, then simple unweighted average parameters across all tests or test pairs are reported.

Results and Discussion

Useful regression models (*Table 1*) were identified for biased and unbiased heritability of rust resistance (h^2_b and h^2, from single- and paired-sites with values of the coefficient of determination, r-square, of 0.33 and 0.43 respectively), and type B genetic correlation between 2 sites (r_B with r-square = 0.18). However, no models adequately predicted the proportion of dominance variance (either biased or unbiased estimates) or the age-age genetic correlation (r_g). Where possible an approximate test for lack of fit was constructed by subdividing the independent variable into classes (WEISBERG, 1985, p. 95) and plots were used to assess the validity of the models. For the models reported no important lack of fit was detected even though the r-square values reported are generally only modest.

Age, mating design (diallel or factorial), and the type of cross (crosses between initial first-generation parents or crosses with rust-free parents) were not found to be significant (p-value ≤ 0.05) in any model attempted. Therefore all data regardless of age, mating design and cross-type were used together for both single- and paired-site models. Parameters were successfully modelled using simple functions of the mean level of rust incidence in a test (R), the sum of the mean rust incidence in 2 tests (RSUM = $R_1 + R_2$), the average rust incidence in 2 tests (RAVG = RSUM/2), or the absolute difference in the mean rust incidence of the 2 tests (RDIFF = $|R_1 - R_2|$), expressed in percentage units. The relatively low r-square of the model predicting the type B genetic correlation (*Table 1*), is reasonable given the difficulty of estimating genetic correlations (FALCONER, 1989, p. 317) and experimental error associated with the estimation of parental performance. All the predictive models presented are believed to have greater utility than either the parameter estimates themselves, or simple average parameters which take little or no account of difference in rust infection.

Heritability

Biased heritability (h^2_b) was predicted by a quadratic function of the mean rust in the test (*Table 1*). The model presented was better than simply using the mean rust incidence and its square: this alternative model had significant lack of fit, and a lower r-square value. The predicted maximum biased heritability is 0.20 when the mean rust incidence equals 72% (*Figure 1*). The model predicting unbiased heritability (h^2) from paired tests is a simple linear function of the average rust incidence in the 2 tests (*Table 1*), and unlike the predicted biased heritability no significant quadratic effect was detected (*Figure 1*). It is possible that the lack of a quadratic trend in the h^2 model is related to the very limited amount of data which was available for the upper range of average rust infection levels. The maximum value of RAVG observed was 87.5%, and only in 29 out of 955 test-pairs did RAVG exceed 75%. Since pairs of connected tests with high levels of rust in both tests were relatively rare, *figure 1* is only plotted for average rust incidence between 0 and 80%. If additional data had been available from test-pairs with high levels of rust infection it is

Table 1. – Regression models to predict single-site biased heritability (h^2_b), paired-site unbiased heritability (h^2), and type B genetic correlations (r_B), for resistance to fusiform rust developed from 171 slash pine full-sib tests. (Regressors in models: R = mean rust incidence at one site; RSUM = sum of the mean rust incidence at 2 sites; RAVG = RSUM/2; RDIFF = absolute difference in mean rust incidence at two sites.).

Dependent Variable	Age	Regression Model	r^2	n
Heritability of rust in one test (h^2_b)	All (4-15)	$\hat{h}^2_b = 0.000156 + 0.00130(R)^{1.5} - 0.000115(R)^{2.0}$	0.33	362
Heritability of rust in a pair of tests (h^2)	All (4-15)	$\hat{h}^2 = -0.0223 + 0.00323(RAVG)$	0.43	955
Type B genetic correlation of rust between two tests (r_B)	All (4-15)	$\hat{r}_B = 0.405 + 0.00871(RSUM) - 0.0000316(RSUM)^2 - 0.00712(RDIFF)$	0.18	908

possible that the predicted h^2 might have declined in a manner similar to the predicted h^2_b above 70% rust infection levels. However, in defence of the linear model presented it should be noted that the predicted h^2 at RAVG = 100% is 0.30, which is similar to realised heritability estimates; $h^2_r \cong 0.35$ at rust infection levels exceeding 90% (HODGE *et al.,* 1990). At a rust infection level of 67% both models predict heritability to be 0.19 (*Figure 1*). Finally, the models were checked to determine whether or not they conformed to the relationship: $h^2_b = h^2 \div r_B$. At average rust infection levels (50%) the models conform well to this relationship; however, above 67% rust infection levels, h^2 exceeds h^2_b (which is theoretically impossible) with the absolute difference increasing rapidly to over 0.16 at 100% rust infection. Thus it would appear that the model for unbiased heritability is most reliable only for RAVG values below 67%.

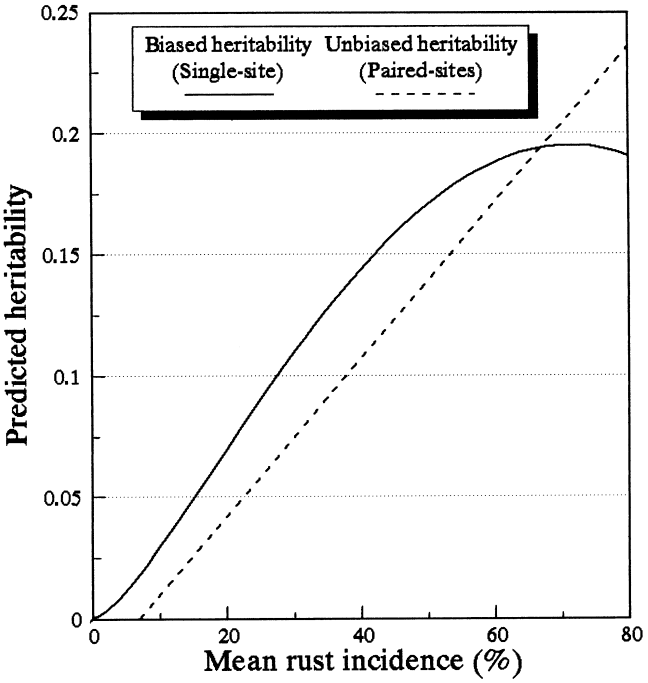

Figure 1. – Predictive models for the single-site biased heritability (h^2_b) and paired-site unbiased heritability (h^2) of resistance to fusiform rust in slash pine. Models developed using data from 171 full-sib slash pine progeny tests measured between 4 and 15 years after field planting. (Refer to *Table 1* for more details of the models).

Proportion of dominance

Attempts to develop predictive models for biased and unbiased estimates of the proportion of dominance (d^2_b and d^2 respectively), and for the ratio of additive to dominance variance (h^2/d^2), were fruitless. No model with an r-square exceeding 0.09 could be found for d^2_b, d^2, or h^2/d^2. Analysis of variance revealed that neither age classes, rust classes, nor the interaction between age and rust classes was significant in the case of d^2_b. For paired-site analyses age classes were also not significantly different and test pairs could not be readily classified on the basis of rust incidence. In the absence of reliable models, or meaningful classification variables, the unweighted mean proportion of dominance was estimated to be 0.087 and 0.053 for d^2_b and d^2 respectively, in tests where the mean rust incidence was between 10% and 90% (*Table 2*). Tests with extreme levels of rust were excluded for the purpose of estimating these means, because variance is low in such tests (SOHN and GODDARD, 1979). Average biased estimates were higher than unbiased estimates reflecting the presence of dominance-by-environment interaction which is about 64% of the dominance variance. Dominance-by-environment interaction of this size may have important implications if full-sib families are deployed operationally at some future date.

Table 2. – Mean proportion of dominance (± standard error of mean) in the fusiform rust resistance of slash pine, estimated from single and paired full-sib tests. (Means include only tests where the mean rust incidence is between 10% and 90%.).

Analysis type	Number of tests or test pairs	Proportion of dominance
Single-tests	307	0.087 ± 0.007
Paired-tests	674	0.053 ± 0.003

If the proportion of dominance in fusiform rust resistance of slash pine is constant across levels of rust incidence and age classes between 4 and 15 years, as these data indicate, then the ratio of additive to dominance variance ($\sigma^2_A/\sigma^2_D = h^2/d^2$) can be estimated by diving the predicted heritability by the mean proportion of dominance. For single tests within the limits of 10% to 90% rust incidence, o^2_A/o^2_D has a maximum of 2.2 at a rust incidence of 71.5%, and a minimum of 0.34 at 10% rust incidence.

Genetic correlations

Type B genetic correlation for fusiform rust resistance between 2 tests was modelled as a function of the sum of the mean rust incidence in the 2 tests (RSUM), and absolute difference in the mean rust levels in the 2 tests (RDIFF), with an r-square of 0.18 (*Table 1*). Although the r-square for this model is relatively small, the model is biologically plausible. The predicted r_B is at a maximum when the 2 tests have the same mean rust infection level, and decreases as the difference in the rust incidence between the 2 tests increases (*Figure 2*). When rust levels in both tests are intermediate (30% to 70%) then type B genetic correlations are predicted to exceed 0.67. SHELBOURNE (1972) suggested that when the variance of GxE is at least half the size of the additive variance (i.e. type B = 0.67), breeding strategies should be modified to address this interaction, or potential genetic gains will be compromised.

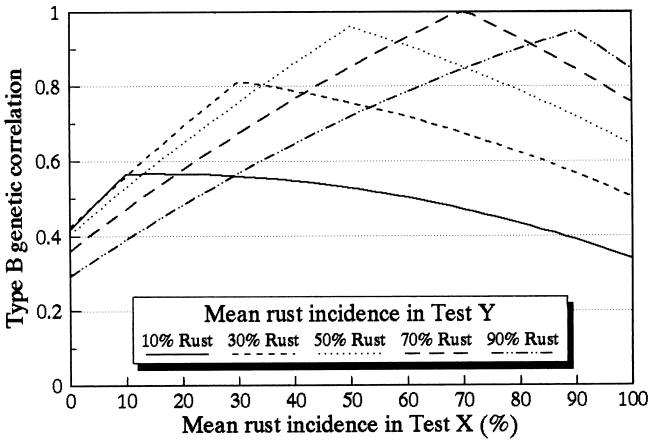

Figure 2. – Predicted type B genetic correlation (r_B) for resistance to fusiform rust, between 2 slash pine progeny tests (Tests X and Y). Predictive model developed using data from 171 full-sib slash pine progeny tests between 4 and 15 years after field planting. (Refer to *Table 1* for more details of the model).

Therefore, at intermediate levels of rust infection, GxE interaction would appear to be of little importance for rust resistance in slash pine.

No significant regression models were found for genetic correlations (r_B) of rust resistance at 2 different ages between 5 and 14 years. To prevent estimates well outside the parameter space from unduly influencing average parameter estimates, individual estimates of r_B exceeding 1.3 were set to 1.3. Unweighted average age-age genetic correlations estimated from tests where the mean rust incidence in both tests was between 10% and 90% were not significantly different from + 1.0. These very strong age-age correlations are reasonable in view of the fact that for rust resistance no other genetic parameter showed any relationship to age.

It is possible that these genetic correlations are biased downward due to heterogeneity of genetic variances between sites (EISEN and SAXTON, 1983). This would mean that GxE is less important than suggested by the magnitude of the type B genetic correlations. Therefore, any potential downward bias is of no practical concern in this study.

Implications for breeding strategy

For most quantitative traits breeders attempt to identify a minimum selection age which will maximise economic and genetic gains from improvement work. Data presented here show that, for resistance to fusiform rust in slash pine, the mean infection level is a more important determinant of future gains than is age: age was not important in determining any genetic parameter estimated, nor were age-age genetic correlations significantly different from +1.0. When selecting for resistance to fusiform rust infection, provided the trees have been exposed to at least moderate infection levels, there appears to be no benefit in delaying selection beyond 5 years. Also, because infection levels are a primary determinant of heritability, and hence genetic gain, it is important that tests be established in areas where they are likely to be exposed to the pathogen, i.e. moderate to high rust hazard sites.

Genotype-environment interaction for fusiform rust resistance was detected, but appears to be inconsequential. At intermediate levels of rust infection levels (30% to 70%) type B (between-site) genetic correlations are always sufficiently high to negate concerns about GxE reducing potential gains. Also as long as selection for rust-resistant individuals is restricted to sites with high rust infection levels, then heritability is high (*Figure 1*) and type B genetic correlations with other tests are high (*Figure 2*).

The proportion of dominance (d^2) in slash pine was not found to change with age or rust infection levels, but to be constant at relatively low levels across all parameters examined. Dominance variance is only expected to be important relative to additive genetic variance where the mean infection level is low, i.e. where the amount of additive variance is low. However, because resistance to rust is of little importance on sites with low levels of fusiform rust, dominance variance is regarded as unimportant to the breeding of resistant genotypes for future deployment.

These results reaffirm assumptions about the relative unimportance of dominance variance underlying the current CFGRP breeding strategy (WHITE *et al.*, 1993), and endorse this strategy in that it is based on recurrent selection for general combining ability. However, the results do reinforce the need to establish progeny tests on sites where rust infection is likely to be moderate to high if substantial future gains in rust resistance are to be achieved, and that selection "age" should be determined by mean rust infection levels rather than the test age.

Conclusions

Significant regression models for fusiform rust resistance were identified to predict biased and unbiased heritability, and the type B genetic correlation between 2 sites, as functions of the mean rust incidence in the tests. The maximum predicted single-site (biased) heritability is 0.20 at 72% rust infection. A simple linear relationship was modelled between the unbiased (paired-site) heritability of rust resistance and the mean rust infection level. However data from situations where both tests encountered high levels of rust infection were rare, and the model for unbiased heritability appears to be most reliable for mean rust infection levels below 67%. Type B genetic correlations indicated the existence of GxE; however, at intermediate to high levels of rust infection (> 50%), GxE did not appear to be important in the rust resistance of slash pine. The proportion of dominance could not be related to rust incidence or age, and average estimates of 0.087 and 0.053 were obtained for d^2_b and d^2 respectively. Dominance variance was small relative to the additive variance except at low levels of rust infection. Therefore dominance variance is not considered to be of any importance to the improvement of rust resistance in slash pine. Age was not a significant factor affecting the estimation of any genetic parameter examined, and all age-age genetic correlations for rust resistance were close to 1.0. Therefore age seems to be unimportant in determining the quantitative genetics of rust resistance in slash pine. Breeding strategies that concentrate on the utilisation of additive variance, and which restrict testing and selection efforts to sites with at least 50% rust incidence, are expected to be the most successful strategies for the improvement of fusiform rust resistance in slash pine. These conclusions endorse the current CFGRP breeding strategy for slash pine.

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Buchbesprechungen

Forstliche Vegetationskunde. Pareys Studientexte 82. Von A. FISCHER. 1995. Blackwell Wissenschafts-Verlag, Berlin und Wien. ISBN 3-8263-3061-7. 315 Seiten mit Abbildungen, Tabellen und Übersichten. Broschiert DM 58,–/öS 429,–/sFr 58,–.

Die Vegetationskunde hat beständig an Bedeutung in der forstwissenschaftlichen Forschung und Praxis gewonnen. Neben der Ansprache des Bodens nutzen Forstleute die Vegetation als Zeiger der Standortbedingungen. Angesichts des großen Fächerspektrums während der forstlichen Ausbildung bleibt es nicht aus, daß man sich nicht mit allen Fächern in gleicher Intensität auseinandersetzt. In der forstlichen Praxis und in landschaftsbezogenen Berufsfeldern werden jedoch fundierte Kenntnisse der allgemeinen Zusammenhänge aller Disziplinen benötigt.

Der vorliegende Leitfaden führt das 1949 von RUBNER verfaßte Buch *„Die Waldgesellschaften in Bayern"* fort und akzentuiert die Vegetation als Ausdruck und Indikator der Umweltbedingungen im forstlichen Bereich stärker. Der Autor listet keine Einzelfaktoren auf, so werden beispielsweise weder sämtliche geobotanischen Arbeitsmethoden erklärt, noch sämtliche Waldgesellschaften Deutschlands behandelt oder die Verbreitungsgebiete aller Baumarten vorgestellt. Vielmehr steht das Grundverständnis für geobotanische Zusammenhänge als Basis für forstliches Handeln im Vordergrund. Einer knappen Einführung folgt im 2. Kapitel ein breit angelegter Grundlagenteil, in dem für einen Forstmann wesentliche allgemeine geobotanische Konzepte, die wichtigsten Grundbegriffe und die gängigsten methodischen Ansätze vorgestellt werden. Das 3. Kapitel umfaßt eine Übersicht der flächenmäßig bedeu-

tenden Waldgesellschaften Mitteleuropas einschließlich ihrer standörtlichen Kennzeichnung und benennt diagnostisch die wichtigen Arten. Dem Anwendungsbereich der forstlichen Vegetationskunde gilt das letzte Kapitel, in dem exemplarisch Nutzungsmöglichkeiten und Auswertungswege aufgezeichnet werden. Ein 16seitiges Literaturverzeichnis erleichtert dem Interessierten den Einstieg in die weiterführende Fachliteratur. In 2 Anhängen werden hochwüchsige Gehölze in der Bundesrepublik Deutschland und wichtigste Charakter-, Differential- und Zeigerarten mitteleuropäischer Waldgesellschaften aufgelistet. Ein 12seitiges Register beschließt den Leitfaden.

Das Buch zeichnet sich durch sein beispielhaftes didaktisches Konzept und seine systematische Gliederung aus. Allen Studierenden an Hoch- und Fachhochschulen sowie den im praktischen Waldbau Tätigen bietet die „Forstliche Vegetationskunde“ einen umfassenden Überblick über den gesamten Themenkomplex. Darüber hinaus kann das Buch auch allen mit Landnutzung und Landschaftsgestaltung beschäftigten Personen uneingeschränkt empfohlen werden.

M. LIESEBACH (Grosshansdorf)

Wälder der Schweiz. Von Lindengrün zu Lärchengold – Vielfalt der Waldbilder und Waldgesellschaften in der Schweiz. Von P. STEIGER. 1994. Ott Verlag, Thun. ISBN 3-7225-6200-7. 360 Seiten mit 763 Farbabbildungen sowie rund 250 s/w-Zeichnungen, Karten und Skizzen. Gebunden DM 79,–.

Die Waldfläche der Schweiz beträgt rund 12 000 km^2 und bedeckt etwa 27 % der Landesfläche. In der Schweiz mit stark ausgeprägtem Relief und fehlenden Tiefländern sind höhenbedingte Wechsel der Vegetation, oft auf kürzester Distanz, weitverbreitet. Die Waldbilder wandeln sich auch von Region zu Region. In Abhängigkeit von Boden, Klima, Lage und weiteren Faktoren bilden Bäume, Sträucher und Kräuter Waldgesellschaften.

Im 80seitigen Einführungsteil werden die einzelnen Faktoren, die einen Waldstandort bestimmen und ihre Bedeutung für die Ausbildung unterschiedlicher Pflanzengemeinschaften erläutert. Der Leser erfährt etwas über die Waldgeschichte der Schweiz, die Waldvegetation, den forstlichen Standort (Klima, Boden, Höhenstufen), die Konkurrenz der Arten und die Wirtschaftsformen im Wald. Weiterhin informiert der Autor über Wälder als Lebensraum für Tiere und als Erholungsraum sowie über Waldschäden.

Im 2. Teil des Buches werden auf 225 Seiten die Waldgesellschaften der Schweiz vorgestellt. Die Beschreibung der 116 Waldgesellschaften erfolgt in 6 Kapiteln: Buchenwaldstufe, wärmeliebende Eichenmischwälder der Hügelstufe, Tannen-Buchen- und Tannenwaldstufe, Fichtenstufe der Randalpen, Nadelwälder der Inneralpen, Föhrenwälder. Je ein idealisiertes Bestandesprofil und eine Verbreitungskarte ergänzen die Beschreibungen der einzelnen Waldgesellschaften. Am Ende einer Beschreibung werden Name und Nummer der Waldgesellschaft des verbreiteten, z. Z. vergriffenen Standardwerks „Waldgesellschaften und Waldstandorte der Schweiz“ von ELLENBERG und KLÖTZLI (1972) aufgeführt.

Abgerundet wird die umfassende Dokumentation durch einen 55seitigen Anhang. Dieser enthält neben einer Systematik der Waldgesellschaften, eine vorläufige Übersicht über Verbreitung, Seltenheit und Schutzstatus der Waldgesellschaften der Schweiz und 9 Ökogramme, die für die Alpennordseite, Inneralpen und Alpensüdseite sowie Höhenstufen die Verknüpfung der Waldgesellschaften verdeutlichen. 25 Seiten Register der Waldgesellschaften, Pflanzennamen (deutsch und wissenschaftlich), ökologischen Zeigerwerte der beschriebenen Pflanzen, Orte und Sachworte sowie ein 440 Titel umfassendes Literaturverzeichnis beschließen das Werk.

Durch eine allgemeinverständliche Sprache ist das Buch nicht nur für Fachleute, sondern auch für interessierte Laien leicht verständlich. Der breite Einführungsteil, die umfassende und reich bebilderte Beschreibung der Waldgesellschaften sowie der umfangreiche Anhang machen „Wälder der Schweiz“ zu einem unentbehrlichen Nachschlagewerk für alle an Waldgesellschaften Interessierte.

M. LIESEBACH (Grosshansdorf)

Waldrandpflege: Grundlagen und Konzepte. Reihe: Praktischer Naturschutz. Von T. COCH unter Mitwirkung von H. HONDONG. 1995. Neumann Verlag, Radebeul. ISBN 3-7402-0150-9. 240 Seiten mit 40 Farbfotos, 67 schwarz/weiß Abbildungen und 21 Tabellen. Gebunden DM 78,–.

Waldränder, insbesondere Waldaußenränder als Übergang zwischen Offenflur und Wald, sind prägende Elemente in unserer Kulturlandschaft. Die Waldaußen- und die Waldinnenränder, letztere sind die Grenzbereiche zwischen Waldbeständen, die u. a. durch Schneisen oder Wege getrennt sind, erfüllen Schutzfunktionen für die dahinter liegenden Bestände gegen Wind, Sturm, Sonne sowie Feuer und haben besondere Bedeutung für den Biotop- und Artenschutz. Der Autor zeigt in den ersten beiden Abschnitten die Besonderheiten des Landschaftselementes Waldrand auf. Dabei mißt er im 1. Abschnitt der landschaftsgeschichtlichen Entwicklung von Waldrandstrukturen besondere Bedeutung bei. Im 2. Abschnitt erfolgt zum einen auf der Ebene von Pflanzengesellschaften eine Vorstellung der waldrandtypischen Flora, zum anderen werden die Lebensbedingungen für die Fauna an Hand einer Vielzahl ausgewählter Tierarten beschrieben. Der letzte Abschnitt stellt Methoden der Erfassung und der Bewertung von Vegetation und Fauna vor und führt im abschließenden Kapitel „Naturschutz an Waldrändern“ die zuvor behandelten Aspekte zusammen. Dabei verdeutlicht der Autor, daß einerseits die aktuelle Ausprägung der Vegetation oder der Fauna ohne landschaftsgeschichtlichen Bezug kaum interpretiert werden kann, andererseits die Landschaftsgeschichte ihrerseits ohne einen strukturellen Bezug aussagelos im Hinblick auf mögliche Biotopqualitäten vergangener Landschaftssituationen bleibt. Den Abschluß des Buches bilden einige Entwicklungskonzepte und Pflegemaßnahmen, die auch die Förderung und den Erhalt seltener und gefährdeter Baum- und Straucharten berücksichtigen. Der Autor verliert sich nicht in pragmatischen Äußerungen, sondern hat an Hand naturschutzfachlicher Untersuchungen eine wertvolle Monographie des Lebensraums Waldrand verfaßt.

M. LIESEBACH (Grosshansdorf)

The vegetative multiplication of forest trees. (In Rumanian). By V. ENESCU, L. IONITA and M. PALADA. 1995. Ed. Ceres, Bucharest. 334 pages.

The book deals with the vegetative propagation of forest trees and its implications on practical forestry. Also, it covers the concept of clonal forestry. At the beginning, the authors point out the scientific basis of vegetative propagation, paying special attention to the cellular level and the importance of meristems. The relationship between propagation and developmental phase, e.g. juvenile, adult, and scenescent phase are treated as well as the rejuvenilisation by way of apex cultures.

The second part of the book presents the conventional methods of vegetative propagation; grafting, micro grafting, and by way of cuttings. Serial and large scale propagation are described for Norway spruce, pines, oaks, beech, ash tree, and sycamore maple.

Propagation by means of organs, tissues and cells In vitro are demonstrated in the third part of the book. Different theoretical and technical aspects of both, micro propagation by organogenesis and embryogenesis are covered.

The fourth chapter of the book gives the latest applications of in vitro cultures: different ploidy levels, zygotic embryo, somatic crossbreeding, genetic transformation, somaclonal reproduction, nitrogen fixation, and possibilities to receive secondary products.

The fifth part of the book introduces plant breeding strategies based on vegetative propagation beginning with the preservation of the inherent genetic diversity and ending with examples of plant breeding strategies based on clonal selection.

The last chapter of the book covers clonal forestry. It includes theoretical aspects like clone cultures and clone genetics as well as practical implications for forestry as regards to modernisation, advantages and disadvantages, its principles and possibilities for use including forest policy and legislation.

The subjects covered are well reviewed which is shown by the comprehensive bibliography consisting of 670 titles. The significance of the volume is that it shows the large scientific progress in this field and the strong implications on practical application to forestry and commercial use. New strategies and national forest policies are necessary to include the novel techniques as well as theories and concepts reaching from micro propagation to genetic engineering.

The book addresses forest administrators and scientists specialised in forestry, horticulture, and related fields. Written in a clear and lapidary style, the book is easy to read for anybody in control of the Romanian language.

I. SMÂNTANÂ (Bucharest)

Waldökologie. UTB für Wissenschaft: Große Reihe. Von H.-J. OTTO. 1994. Verlag Eugen Ulmer, Stuttgart. ISBN 3-8001-2665-6. 391 Seiten mit 140 Zeichnungen und 19 Tabellen. Gebunden DM 78,–.

Der Verfasser, Professor Dr. HANS-JÜRGEN OTTO, hat auf der Grundlage seiner forstlichen Praxis und seiner universitären Lehre eine zusammenfassende Abhandlung der Waldökologie vorgelegt. Anhand zahlreicher praktischer Beispiele aus bestehenden Wäldern sowie zahlreichen Zeichnungen werden Modelle und allgemeine ökologische Konzepte dargestellt. Einführend wird im 1. Kapitel auf die Stellung der Waldökologie im Feld der Naturwissenschaften eingegangen. Nachfolgend werden die Faktoren der unbelebten Umwelt, die Wirkung der Umwelt auf den Wald, die Anpassung des Waldes an die Umwelt und die ökologischen Rückwirkungen des Waldes auf die Umwelt eingehend behandelt. Im dritten Kapitel geht der Autor auf die Besonderheiten des Waldökosystems im Vergleich mit anderen Ökosystemen ein und setzt sich mit dem räumlichen Auftreten sowie der inneren Organisation von Waldökosystemen auseinander. Die zeitliche Entwicklung von Waldökosystemen wird im letzten Kapitel beschrieben. Nach der Abhandlung der Antriebskräfte der Walddynamik wird ein Einblick in die kurz-, mittel- und langfristige Veränderung des Waldes gegeben. Dabei wird nicht nur auf das intakte ökologische Wirkungsgefüge im Wald eingegangen, sondern auch die Wirkung von Störungen und Unregelmäßigkeiten dargestellt. Verzeichnisse mit der verwendeten und der weiterführenden Literatur folgen jeweils am Ende der einzelnen Kapitel. Ein umfangreiches Sachregister beschließt dieses ausgezeichnete Buch, das als Informationsquelle und Nachschlagewerk allen ökologisch Interessierten uneingeschränkt zu empfehlen ist.

M. LIESEBACH (Grosshansdorf)

Danksagung (Note of Thanks)

Die unerwartet große Zahl von Glückwünschen anläßlich meines 90. Geburtstages und ein mich behinderndes Augenleiden müssen zu erheblichen Verzögerungen bei der beabsichtigten individuellen Beantwortung führen. Ich möchte daher vorsorglich auf diesem Wege meinen Dank aussprechen.

Besonders liegt es mir jedoch am Herzen, in aller Öffentlichkeit meinem ehemaligen Mitarbeiter und Freund, dem Ordinarius der Universität Göttingen, Professor HATTEMER für die von ihm bei der Feierstunde im Schmalenbecker Institut gegebene Beurteilung meiner Person und Forschungstätigkeit zu danken. Die von ihm gegebene Nachzeichnung der Institutsentwicklung nach Kriegsende bis zu meiner Pensionierung und die Würdigung auch meiner nachfolgenden ebenso viele Jahre währenden Tätigkeit, waren für mich ein Rückblick besonderer Art. Sie brachten mir, ohne daß HATTEMERS Ausführungen dies anklingen ließen, nicht nur die Erfolge in Erinnerung, sondern auch meine immer wieder wach werdenden Zweifel an gelegentlich gefällten Entscheidungen und für richtig gehaltenen Meinungen.

Zu danken habe ich auch allen denen, die meinen Werdegang in der Sache kritisch begleitet haben. Ohne sie wäre die Zahl möglicher Fehlentscheidungen sicherlich sehr viel größer.

W. LANGNER

I would like to personally express my gratitude to all the unexpectedly large number of good wishes received by me on my 90th birthday. But, unfortunately, due to an eye problem there would be a considerable delay in sending individual responses. Therefore, I would like to express my thanks through this note in the Silvae Genetica.

I would especially like to thank my former colleague and friend, Prof. HATTEMER, University of Göttingen, for presenting an overall assessment of my person and a review of my research accomplishments during my birthday celebration at the Schmalenbeck Institute. His retracing the evolution of the Institute after the War and upto my retirement, as well as the appreciation of the many years of my subsequent activities, was for me, in retrospect, very special. Even without HATTEMER's mentioning it, I was reminded, not only of the successes, but also my ever present doubts regarding certain decisions and those considered as correct opinions.

I would like to thank all who were my critics throughout my entire career. Without them, the number of possible wrong decisions made would have been certainly much more.

W. LANGNER

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