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Testing Scots Pine for Resistance to Lophodermium Needle Cast

Prövning av tallens resistens mot tallskyttesvampen

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Abstract

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A method for assessing resistance to Lophodermium needle cast has been developed and applied for analysis of genetically dependent resistance in Scots pine. Determination of the decrease in height growth caused by the fungal attack was a satisfactory method for assessing the resistance. The relative height growth of the most susceptible progenies was reduced to about 30 % of that of the most resistant progenies.

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Preface

A cooperative venture concerned with screening for resistance to pathogenic fungi of progenies from Swedish pine seed orchards was initiated in 1971 between the Swedish College of Forestry and the Institute for Forest Improvement.

This paper reports on resistance of Scots pine (Pinus sylvestris L.) to Lophodermium pinastri (Schrad.) Chev.

1 Introduction

Breeding to improve resistance to plant diseases is based on the same principles as those generally followed in plant breeding, i.e. selection and combination of characters. The selection is made after resistance testing and implies a measuring of the resistance characters of individuals and populations. The breeding of agricultural crop plants has for a long time included breeding for resistance to important diseases (Borlaug, 1966). In several cases it has been possible to identify individual genes of resistance and their inheritance (Flor, 1955; Noronha-Wagner and Bettencourt, 1967; Mac Key & Mattson, 1972).

The experience of breeding disease-resistant forest trees is greatest in the USA, where breeding for resistance to *Cronartium fusiforme* of the southern pines, *Pinus taeda* and *Pinus eliotti* (Kinloch, 1972; Stonecypher et al., 1973; Dinus, 1972), and breeding of the white pines, *Pinus strobus* and *Pinus monticola* for resistance to *Cronartium ribicola* (Bingham, 1966; Hoff and McDonald, 1972; Patton and Riker, 1966) has been performed for many years. In Sweden, breeding for disease resistance in forest trees started in 1971 (Björkman, 1972).

Research into resistance to *Lophodermium pinastri* (Schrad.) Chev. of Scots pine (*Pinus sylvestris* L.) has been carried out in Germany (Tubeuf, 1901; Langner, 1933; Schütt, 1957b). In Sweden, the disease caused by *L. pinastri* has been investigated by Lagerberg (1913) and Johnsson (1975).

Breeding for disease resistance was started in Germany in the early 1950's when *Lophodermium* needle cast was epidemic in both young and old pine stands, some of which were situated in Schleswig-Holstein (Langner, 1951/52a; Schütt 1957b). During these severe epidemics, some of the trees in the seriously affected stands sustained only insignificant attacks. These individuals were vegetatively propagated and planted in special infection blocks where they were subjected to hard infection pressure for several years (Schütt, 1957a). It was found that none of the selected individuals was completely resistant to *Lophodermium*, although clear differences could be distinguished. Moreover, it was noticed that the degree of resistance could change with time.

In several provenance trials in Germany, the Netherlands, France and Poland, it was found that there were also clear differences in resistance between pine populations (Hattemer, 1966; Squillace et al, 1975; Lanier, 1968; Siwecki et al, 1975). In some of these trials an interaction between the susceptibility to *Lophodermium* and the environment was observed.

Lophodermium needle cast is one of the most intensively studied needle diseases of Scots pine (*Pinus sylvestris*) (Stephan, 1975b). Yet there is still much uncertainty as regards the biology of the pine and the inheritance of resistance characters. The production of hybrids of trees selected for resistance to *Lophodermium* has therefore not yet been possible. Purposeful research within these fields is therefore essential.

1.1 Objectives of the investigation

Breeding for disease resistance requires methods which can measure different degrees of resistance. Such methods should provide a correlation between the measured value of each attacked seedling and the prospects for the seedling developing after the attack. The methods should also enable the collected information to be analysed statistically.

It should be possible to measure the resistance by direct or indirect methods. If

the resistance of the seedlings is to be measured by a direct method, the environmental conditions must be the same for all test seedlings and the pathogen must have the same opportunity of attacking the seedlings.

If the resistance of the seedlings is to be measured by an indirect method, some known relation must exist between one or several measurable characters in the unaffected host seedling and its resistance to the pathogen. This relation must be of universal application.

Resistance test on progenies should make it possible to select for genetically dependent resistance either among the parents of the progenies, or among the progenies, or both. The selection must be made in such a way that the selected stock can be expected to have a higher degree of resistance than that of the initial population (Griffing, 1956; Falconer, 1964).

Against this background the following questions have been investigated in this work:

1. How should resistance to *Lophodermium* be measured?
2. Is resistance to *Lophodermium* genetically dependent and how can a selection for resistance to *Lophodermium* in a given population of Scots pine be made on a genetic basis?
3. Can some method of indirect selection be used?

2 Lophodermium needle cast, its causes and development

2.1 Biology of the pathogen

Taxonomy and distribution

Lophodermium pinastri (Schrad.) Chev. is ascomycetous, belonging to the order *Phacidiales* and the family *Hypodermataceae*. *Leptostroma pinastri* Desm. is the conidial stage of the fungus (Gäumann, 1963). Related fungi of importance in Swedish forestry are *Phacidium infestans* Karst., *Lophodermella sulcigena* Rostr. and *Lophophacidium hyperboreum* Lagerb. There are at least 17 different genera of the family *Hypodermataceae* with one or several species growing on conifers (Darker, 1968). In the genus Lophodermium alone, at least ten species are described which are more or less pathogenic to different conifers (Boyce, 1961).

In addition to *Pinus silvestris* L., *Lophodermium pinastri* has also been found growing on 26 other species of pine in Europe, Asia, Africa, Oceania and North America (Boyce, 1951).

Life cycle and anatomy

The development of the fungus is very irregular and depends on environmental factors (page 8). The life cycle of the pathogen—the commonest in central Europe—has been described by Rack (1963).

In damp weather during late summer and autumn ascospores spread from the needles on the ground. The spores germinate on the surface of living needles. From the beginning of August small brown infection spots can be observed. When the temperature increases in April—May, a rapid change occurs. Within a few days a whole stand of pine can change its colour from green to reddish-brown.

Depending on the strength of the wind, the needles then fall off fairly rapidly. As a rule, severely affected seedlings stand completely devoid of needles for some time, until the new shoots have had time to develop new needles.

The conidial stage of the fungus is developed first and can sometimes be observed before the needles are shed. After the needles have fallen, the apothecia of the fungus develop during late summer and autumn. They occur as dark elliptic spots on the surface of the needle. Later they develop from the tissue of the needle into gatherings, slitting longitudinally in damp weather and discharging the spores.

The fungus can also have a longer life cycle lasting for several years (Lagerberg, 1913; Hagem, 1926; Rack, 1963; Shevchenko, 1968).

The filiform ascospores have a length of 100—160 μ, a width of 2—2.5 μ and are enclosed by a mucilaginous envelope. A variety which is slightly shorter can occur on cones (Butin, 1975). The vegetatively formed conidia are smaller and rod-shaped, and, according to the same source, about 7×0.7 μ. These conidial spores are unable to germinate and their significance to the fungus is unknown (Jones, 1935). When the ascospores germinate on the surface of the needle, at least three different types of spores can be distinguished with regard to the cell division of the germ hyphae, the number of nuclei and growth (Stephan, 1969). There are relatively few stomata on one infection spot which are not penetrated by germ hyphae. On a double needle 200 individual infection spots can exist (Rack, 1963). The anatomy of the fungus inside the needle has been described in detail by Jones (1935).

After the fungus has penetrated the endodermis of the needle, the hyphae grow intra-

cellularly. Since the stomata of the needle have been destroyed by the fungus, the hyphae, after having penetrated the endodermis, cause an uncontrollable transpiration of water through the hyphae of the fungus from the conducting tissue of the needle to its surface. This initiates a reaction in the pine seedling, which leads to the shedding of the whole needle. The ripe apothecium is elliptic when viewed towards the surface of the needle, and has a length of 1—2 mm and a width of about half that size. It splits longitudinally in damp weather and closes again when the humidity level drops.

According to Rack (1963) each apothecium discharges about 2000 spores.

Ecology and physiology

The fungus has a parasitic and a saprophytic stage. The parasitic stage constitutes the part of the life cycle in which the fungus lives on and in the live needle. The saprophytic stage constitutes the other part of the life cycle.

The development of the apothecia is most rapid in those needles that have been shed in June—August. The development is accelerated by high humidity. The optimum temperature for an apothecia formation seems to be 13 to 14°C, which is lower than the optimum temperature for the vegetative growth of the mycelium, which is about 18°C (Rack, 1963).

From comparative studies on infections made in the open air and in greenhouses, it can be concluded that varying temperature increases the degree of fungal attack (Schütt, 1967).

During its saprophytic stage the fungus is highly dependent on the environmental factors influencing the moisture on the ground level. This is considered a contributory cause to the fact that the damage is especially frequent in dense plantations in grass-covered habitats and in nurseries (Lagerberg, 1913).

There are also examples of stands considerably exposed to the wind being heavily attacked by *Lophodermium* (Hagem, 1928). In such cases, however, it seems to be the parasitic stage which is favourably affected. The uncontrolled transpiration in the tree is of decisive importance.

Lophodermium needle cast seems able to exist on all types of forest soil and in nurseries. The physiological investigations performed have mostly been made *in vitro*. The fungus can be cultivated on artificial media. Growth is stimulated by the addition of pine needle extract (Schütt, 1964b). So far nobody has been able to make fructifications with germinable spores develop on artificial media (Melchior, 1975).

The pH-value of the needle tissue as well as its osmotic pressure have not proved to affect the development of the fungus (Hattemer, 1964). In different isolated cultures the optimum pH-value for vegetative growth in *Lophodermium* cultures can vary between 4 and 6. The fungus, however, will grow within the pH-interval of 3—9 (Stephan, 1973).

Fries (1938) and Stephan (l.c.) investigated the vitamin requirements of the fungus and found that in most *Lophodermium* strains the vegetative growth was favoured by biotin, thiamin and inositol. As a rule the fungus is able to hydrolyze starch (Stephan, l.c.).

Genetic variation

Biological race specialization is found within a great number of pathogenic fungi. Within *Puccinia graminis tritici,* the fungus which causes stem rust in wheat, there are, for instance, more than 200 known races and within each race several biotypes (Agrios, 1972).

Because of the difficulties of making the fungus reproduce under controlled conditions, it has not yet been possible to make artificial inoculations with specified biological strains of *Lophodermium pinastri.*

However, it has long since been known that several measurable characters of the species can vary widely. Mayr (1902) thought he could observe pathological differences between *Lophodermium* in nurseries and in older trees.

In Scotland Millar and Watson (1971) found two main types of *Lophodermium*. The two types possibly have pathological differences and present different morphological characters on the affected pine needles.

Stephan (1973) and Scholz and Stephan (1974, 1975a) analysed a great number of *Lophodermium* isolates in respect of morphological characters, optimum temperatures for vegetative growth, vitamin requirements and enzyme production. A large number of isolates have also been investigated with regard to the iso-enzymatic pattern, the number of nuclei per cell and their ability to change the hydrogen ion concentration in the culture medium. These investigations have shown that all the enumerated traits are subject to variation.

Thus the characters of the fungus can vary considerably. So long as controlled tests of the resistance are impracticable it will not be possible to establish whether or not this variability is of importance to the pathogenicity of the fungus.

2.2 The diseased pine

Pine seedlings with secondary needles usually survive single attacks by the pathogen. Even if all the needles have been affected and fall off in spring, the seedling is usually able to develop a new shoot with healthy needles out of the apical bud by means of the stored nutrients in the stem, branches and roots (Lagerberg, 1913). Seedlings with primary needles only, however, have less chance of surviving the attack. These small seedlings have less nutrients stored and the primary needles do not fall off as easily as do the secondary needles; thus, the fungus grows into the shoot as well.

In seedlings with secondary needles the fungus does not usually have time to reach the dwarf shoot, since the needle is shed before that. This needle shedding is a defence mechanism initiated by the pathogen. The infection starts the processes that cause the needle to fall off (Tubeuf, 1913; Langner, 1933).

The growth of a pine seedling depends upon the photosynthesis of the green biomass. If a large number of the needles formed during or before the preceding year are lost, root as well as stem and shoot growth is affected both directly and indirectly. The earlier during the year the needles are lost the more serious will be the negative influence (Rack, 1963). The nitrogen and carbohydrate supply in the seedling are built up in the previous year's needles during the spring. These nutrient reserves are largest immediatly before the buds open (Kreuger, 1967), i.e. usually during the season in which the pine seedling can be deprived of its needles to a greater or lesser degree by *Lophodermium*.

The number of needles on the growing shoot is predestined in the bud, which is formed as early as the year before the disease breaks out. The elongation of the shoot, however, is dependent upon the available nutrient supply and assimilated material formed in the older needles (Neish, 1958; Kreuger, 1967). If older needles are missing completely or partially, this implies a considerable loss of nutrient supply. A shorter shoot grows out, and the amount of nutrients in the roots, stem and branches is reduced.

The seedling has regulating mechanisms which endeavour to adjust the proportion of the dry weight in shoots and roots to a specific value; however, this value is dependent on environmental factors (Waering, 1970). If a seedling is deprived of some of its needles, thereby receiving a reduced supply of assimilated material, an unbalance arises temporarily between shoot and root. Relatively speaking, the growth of the root will suffer more from the shortage of carbohydrate than will the growth of the shoot. Due to reduced water and nutrient absorption in the root, growth of the shoot and bud is impaired. If the attack does not cease, the seedling will finally die of nutrient deficiency (Lyr et al, 1967).

The upper parts of a pine will be attacked less than the lower parts. The reasons may be that the infection spreads from the ground and that the microclimate higher up prevents fungus growth (Rack, 1963; Blair,

1970). When the pine has reached a certain height, the upper parts usually escape attack completely. A young pine stand at a height of two metres has therefore usually passed the limit below which the trees are susceptible to severe attack by *Lophodermium* needle cast. Thus, the growth ability of the pine and, consequently, its provenance and site quality class, indirectly affect the resistance of pine to *Lophodermium*.

2.3 What is resistance?

According to Gäumann (1951), genetically dependent resistance can be divided into two main types: either the seedling already has a defence against the pathogen, or it has the ability to develop a defence after the attack, i.e. a defence induced by the pathogen.

Both the pathogen and the host seedling—and thus the resistance—are dependent on the environmental factors. The supply of water, light and nutrients, for instance, can displace the defensive preparedness against diseases in a seedling in one direction or another. The pathogen on the other hand, depends on moisture, temperature and adequate plant substratum. Therefore, the resistance is also environmentally dependent.

Resistance testing should make it possible to perform a selection of that part of a population which possesses superior resistance characters. The resistance must therefore be measurable. Resistance to *Lophodermium* has been assessed by means of a visual inspection of the experimental material after a *Lophodermium* attack. The degree of attack has, however, been determined subjectively (Langner, 1951/52a; Schütt, 1957; Johnsson, 1975).

If a measurable character of the healthy host seedling can be correlated with genotypic resistance to the pathogen, this character enables an indirect selection for resistance to be made in a population with unknown resistance characters. This method of selection has been developed for several plant—pathogen systems (Nelson and Birkeland, 1929; Weissenberg, 1973, 1976; Sinclair et al, 1975). A number of such potential characters were investigated in pine for correlation to *Lophodermium* resistance. Hattemer (1964) examined the pH-value and the osmotic pressure in the sap of cells in pine needles. None of these two characters was found to be related to resistance to *Lophodermium*. The differences in the chemical composition of the cuticle wax of pine needles were investigated by Schütt (1971) and Schuck (1972). However, this character could not be related to resistance. On the other hand, measuring the growth of the fungus on agar containing sap from pine needles of different genetic origin gave promising results (Schütt, 1964b).

The pH-optimum for growth of the fungus varies between 4 and 6 (Stephan, 1973). The pH-values of the pine needles varies during the year (see page 35), but during the time of infection and establishment of the fungus in the needle, it is considerably lower than the optimum value for growth of the fungus (Wille, 1927; Hattemer, 1964). The ability of the fungus to change the pH-value of the needle tissues is counteracted by the buffer capacity of the tissues. Scholz and Stephan (1974, 1975a, b) found that this buffer capacity was positively correlated with resistance to *Lophodermium*.

The complex interaction, host—parasite—environment, makes it difficult to study how many and which mechanisms influence the resistance to *Lophodermium*. There is a hypothesis according to which the resistance is inherited polygenetically (Hattemer, 1966). This hypothesis is supported by the fact that no complete resistance seems to exist; on the contrary, numerous intermediate forms are found between highly and slightly susceptible individuals. Langner (1951/52b) assumed that inheritance of resistance to *Lophodermium* is more closely connected with the maternal genes than with the paternal genes. Johnsson (1975) stated the hypothesis that resistance to *Lophodermium* needle cast might depend on only two loci with a dominant and a recessive gene in each locus and equal and additional effects of the recessive genes. The great variation within the full-sib populations, however, invalidated the hypothesis.

3 Experimental material and bases for assessment in tests for resistance to Lophodermium at Tönnersjöheden

3.1 Experimental material

The experimental material contained four provenances of Scots pine, viz. Onsala, Boxholm, Röskär and Kårböle (Figure 1).

Each provenance was represented by progenies from six parent trees, three fathers and three mothers. 74 full-sib combinations were obtained as well as progenies after open pollination of the 12 mother trees (see Figure 2).

The seed was sown in the spring of 1971 at the Röskär experimental station, north of Stockholm. In October, 1972, about 25 seedlings of each progeny were planted in a nursery at the Tönnersjöheden experimental station. The nursery, with an area of about one-third of a hectare, has a shady location. Pine was no longer grown in 1972, since *Lophodermium* had always caused damage. The experimental design consisted of randomized blocks with five replications; i.e. each family was represented by five seedlings per block. The seedling spacing was 40 cm. The plots were arranged in rows, each row consisting of four plots.

Parallel to the experiment at Tönnersjöheden an experiment was started at Lång-

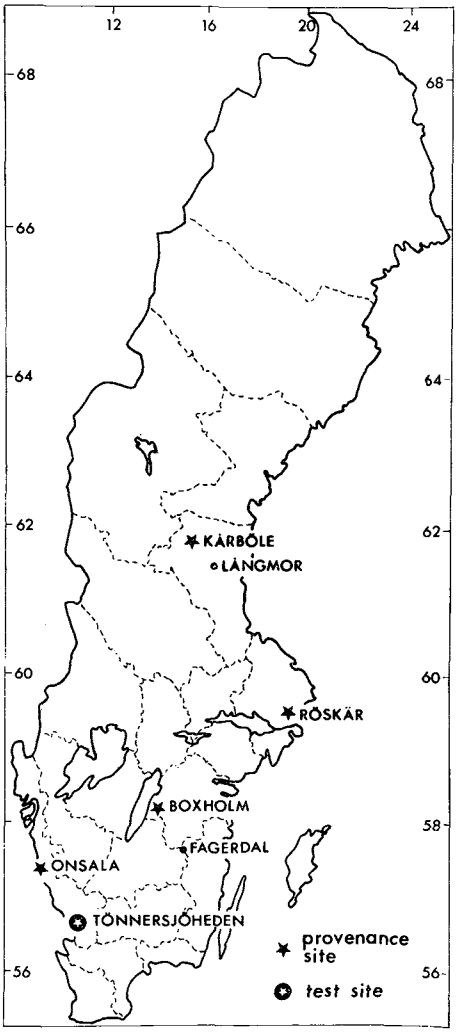


Figure 1. Geographical position of the original stands of the parent trees, the experimental site and the two parallel experiments.

	Lat.	Long.	Alt.
Kårböle	N61°55′	E15°13′	400 m above see level
Röskär	N59°24′	E18°12′	30 m above see level
Onsala	N57°30′	E12°00′	10 m above see level
Boxholm	N58°09′	E15°00′	160 m above see level
Långmor	N62°00′	E16°12′	160 m above see level
Fagerdal	N57°44′	E15°35′	135 m above see level

Crossing chart for the experimental material

♀ \ ♂	KÄRBÖLE			RÖSKÄR			ONSALA			BOXHOLM			open pollination
	X 18	X 26	X 52	B 101	B 102	B 103	N 4	N 201	N 202	E 4008	E 4011	E 4015	
KÄRBÖLE X 9	93*		94*	87		88	91			89		90	134
KÄRBÖLE X 10	101	103									98		135
KÄRBÖLE X 27		110			104			108	109		106	107*	136
RÖSKÄR B 1	7		8	1*		2	5		6	3		4	124
RÖSKÄR B 19	15*			9	10		13	14*		11	12		125
RÖSKÄR B 24		25	26		18*	19*		22	23		20	21	126
ONSALA N 6	67*			61		62	65*		66*			64*	131
ONSALA N 12	75	77					73*	74*					132
ONSALA N 28		85	86		78*	79			83			81*	133
BOXHOLM E 5055		58				53		56	57			55	130
BOXHOLM E 5024	41	42		35	36		39	40		37	38*		128
BOXHOLM E 5053			51			45*	48		49	46		47	129

Figure 2. Crossing chart for the experiment. Each crossing (family) included in the experiment has a family number. The hatched squares are crossings within provenances. Crossings with * are not completely represented in the experiment.

mor and the Department of Forest Genetics started a progeny trial of the same progenies in Fagerdal (Figure 1).

3.2 Bases for assessment in tests for resistance

In order to secure a homogeneous and strong infection, inoculum was collected in May, 1973, in an adjacent young stand at Tönnersjöheden. This stand was about 12 years old and had been severely attacked by *Lophodermium*. The dead needles, collected and transported in bags to the experimental site, had become brown-coloured in the same spring as that in which the collection took place. The needles were spread out on the ground amongst the seedlings in as even a layer as possible.

In April the following year the colour of the needles of the pine seedlings in a large part of the experimental material within a few days turned reddish-brown in a way that is typical of an attack by *Lophodermium* (Lagerberg, 1913). During the autumn, it was confirmed that the attacks had been caused by *Lophodermium pinastri,* by which time numerous fructifications had developed on the shed needles.

On the 22nd and 23rd of May, 1974, an inventory was made of the damage to each seedling. A subjective scale was used for the assessment of the damage which was designed after a close inspection had been made of different types of damage. The variable (ANG 1) corresponded to an assessment of the proportion of needles that had changed colour and turned brown (Table 1). Apart from the needles that were just about to grow out on this occasion, the needles

Table 1. Scale of assessment for degree of attack by needle cast after the first infection (ANG1).

Degree of attack	Share of attacked needles of current year – 2	Share of attacked needles of current year – 1
0	Less than 50 % attacked	Not attacked
1	More than 50 % attacked	Minor attack
2	More than 50 % attacked	Less than 30 % attacked
3	More than 50 % attacked	More than 30 % attacked
4	More than 50 % attacked	More than 60 % attacked

had been formed in 1972 and 1973. The seedlings had only one well-developed whorl of branches, namely, that formed in the summer of 1973.

At the same time the height of the seedlings under the developing leading shoot was measured, i.e. the height reached at the time of the first attack ($Height_1$).

The needles which turned brown in May, 1974, were shed within a week. It was later observed that more needles had been affected and had fallen off. In the spring of 1975, only in exceptional cases were needles from 1973 left on those seedlings that had sustained the least severe attacks.

In the spring of 1975 a severe attack by *Lophodermium* culminated again on the one-year-old needles. On the 12th and 13th of May an inventory of the damage was made by a slightly different method of assessment. The change was necessitated partly by the fact that there were practically no needles older than one year, and partly because each seedling had two well-developed whorls of branches. The damage to the needles from 1974 on the two whorls of branches was assessed individually according to the description in Table 2. In order to make a more objective calculation of the loss of needles, which would correspond to the five degrees of attack, 70 pairs of individuals were selected in the autumn of 1975. These pairs were chosen such that the individuals within the pairs were assessed to have sustained attacks of equal severity in 1974 and 1975. Furthermore they were of the same family and of about the same height; each class of attack was also equally represented, i.e. about 15 pairs to each degree of attack. One individual in each pair was removed in October, 1975, and that year's needles were counted.

In the spring of 1976 the experimental material was attacked for the third year running. On this occasion it was found for the first time that a number of seedlings had died from the disease.

An estimation of the damage was made according to the same principles as those used in the previous year (Table 2). Only the two upper whorls of branches were considered. The branch whorl at the bottom was completely devoid of green needles in the major part of the material. Then the second individual in each of the 70 selected pairs were removed. Loose needles were

Table 2. Scale of assessment for degree of attack by needle cast after the second and third infections, (ANG2) and (ANG3A). The two branch whorls are assessed separately and added together. The scale of assessment for an individual is therefore 0—8.

Degree of attack	Share of attacked needles of current year – 1
0	No brown needles, brown spots on green needles may appear
1	Minor attack
2	At least 30 % of the needles are brown
3	At least 70 % of the needles are brown
4	No green needles

Table 3. Calculation of needle loss during Oct.—May after attack by *L. pinastri* on current year needles. The attack was the third.

Degree of attack ANG3A	Needle loss, % (BARRF · 100), mean	Standard error of the mean	No. of seedling pairs
0	50.9	3.72	9
1	54.7	3.65	11
2	67.2	4.42	9
3	71.0	2.70	5
4	77.3	4.53	7
5	77.8	3.57	4
6	83.4	4.56	5
7	90.8	2.13	10
8	96.2	1.03	10

shaken off and the remaining needles counted. By comparison within the pairs, the number of needles lost from October, 1975, to May, 1976, could be estimated. These needle losses within 70 seedling pairs were grouped into the attack classes resulting from the assessment of May, 1976. The needle loss after the attack of 1976 was calculated as a percentage of the quantity of needles in 1975 (Table 3).

In the case of the individuals that were removed in the autumn of 1975, it was assumed that had they not been removed they would have undergone the same development as those which were in fact left. This assumption was based on the fact that the attacks during the two previous years had been assessed to be alike within the pairs. The needle loss within the nine classes of (ANG3A) was indicated (BARRF) (Table 3). This variable was thus a correction of the variable (ANG3A). The fact that the values of (BARRF) were not directly proportional to the values of (ANG3A) was not surprising.

The values of the needle loss within the nine classes of assessed degree of attack were analysed according to the Q-method of

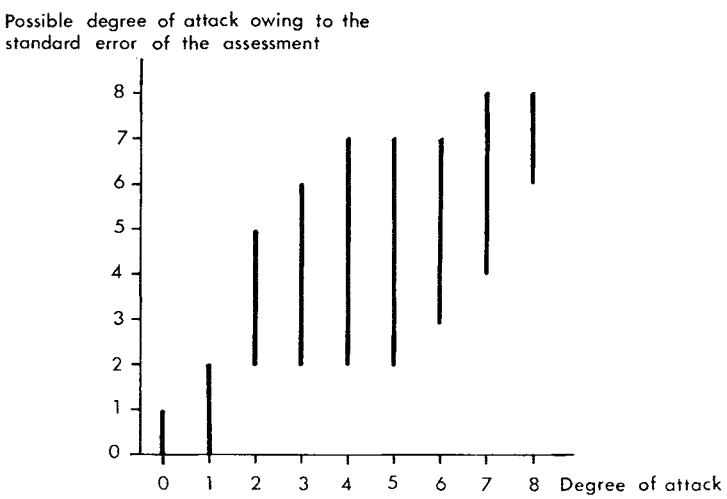


Figure 3. Analysis of means of calculated needle loss for nine degrees of attack (ANG3A) by needle cast.

Table 4. The deviations from the normal distribution of three variables (X^2) and the homogeneity of the variances (Bartlett-Box F).

$H_0: X^2 = 0$, Bartlett-Box F = 1 * = $p \leq 0.05$ ** = $p \leq 0.01$ *** = $p \leq 0.005$		
Variable	X^2	Bartlett-Box F
ASINRB	28.926***	1.744***
BARRF	24.608***	1.980***
LNRHTV	12.950*	1.142*

Newman & Keul on a significance level of 5 % (Snedecor and Cochran, 1976) (Figure 3).

The results of this analysis showed that the means of several of the classes cannot be separated from each other on a level of 5 % significance. Therefore, a special calculation was made of the degree of attack, as estimated for the third year, with division into three classes only. This variable, called (ANG3B) can assume the values 1, 2 or 3 according to the following:

(ANG3A) class 0—1 = (ANG3B) class 1
(ANG3A) class 2—7 = (ANG3B) class 2
(ANG3A) class 8 = (ANG3B) class 3

The values of variables obtained by the five variables described so far proved to have very skewed frequency distributions. The variable (BARRF) was therefore transformed into another variable (ASINRB) according to the following function:

$$(\text{ASINRB}) = \arcsin \sqrt{(\text{BARRF})}$$

The distribution of all six variables, however, could not be considered normal (Figure 4).

Late in July, 1976, i.e. in the summer after the third attack, the height of the experimental seedlings was measured (Height_2). Then the height growth for the three growing seasons during which the seedlings had been affected by *Lophodermium* was calculated. A quotient was obtained for each experimental individual $(\text{Height}_2 - \text{Height}_1)/\text{Height}_1$, which corresponded to the change in growth. The change in growth reflects the effect of the attack on the growth (page 32). The natural logarithm of these quotients was calculated, according to the following function:

$$\text{LNRHTV} = \ln [(\text{Height}_2 - \text{Height}_1)/\text{Height}_1]$$

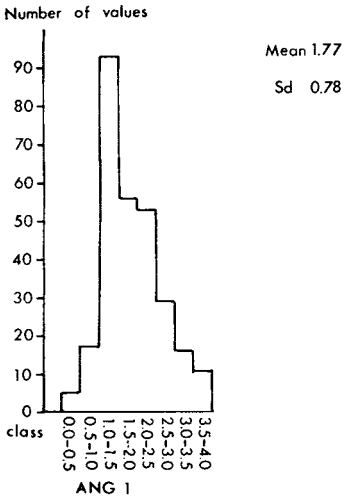
This variable was also used to obtain values with a more normal frequency distribution than that of any of the other six variables (Figure 4, Table 4).

3.3 Calculation of buffer capacity

On the 25th and 26th of July, 1975, current years' needles from eight full-sib families in the growth experiment at Fagerdal were collected. Four of the selected families, 53, 55, 56 and 58, had proved highly susceptible to *Lophodermium* at Tönnersjöheden and four others, 73, 74, 75 and 77, very resistant. From the same families, with the exception of Nos. 73 and 74, samples of needles were taken at the end of July in the experiment at Långmor. Similarly on the 2nd and 3rd of October of the same year, samples of needles from the same families in the experimental areas at Fagerdal and Långmor were taken. From each family needles were taken from the leader of 3—5 individuals.

Since the season in which the needles were collected proved to be of great importance to the analyses (page 25), new collections of needles were made in August and September the following year. This time the current year's needles were taken from different parts of the seedlings. Analyses were made of the four families, 55, 56, 75 and 77. A larger number of individuals were analysed than in the preceding year (Appendix 3). The needle samples were transported in a refrigerated box, by train or air, direct to the laboratory in Umeå, where they were kept frozen until the analyses were made. Also in 1977 needles of the same four families at Långmor were collected and analyzed for the third year running. These needles were collected on the 1st of September, 1977 and were transported to the laboratory with maintained turgescence.

The analyses of the buffer capacity employed a method described by Scholz and Stephan (1974).


Number of values
Mean 1.77
Sd 0.78
class
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0.5-1.0
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1.5-2.0
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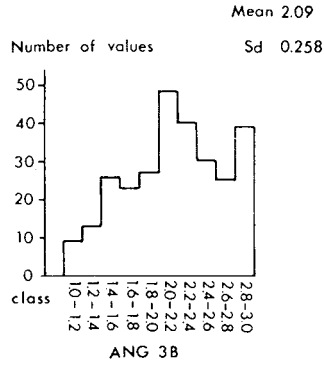
2.5-3.0
3.0-3.5
3.5-4.0
ANG 1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Number of values
Sd 0.258
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1.4-1.6
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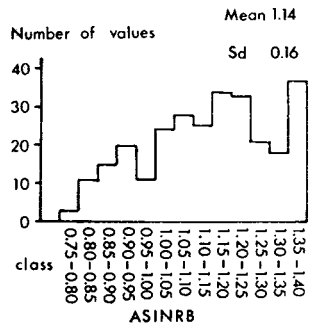
2.2-2.4
2.4-2.6
2.6-2.8
2.8-3.0
ANG 3B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Number of values
Sd 0.16
class
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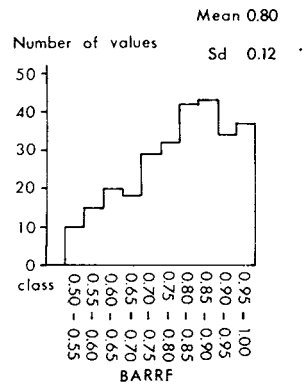
0.95-1.00
1.00-1.05
1.05-1.10
1.10-1.15
1.15-1.20
1.20-1.25
1.25-1.30
1.30-1.35
1.35-1.40
ASINRB


Mean 0.80
Number of values
Sd 0.12
class
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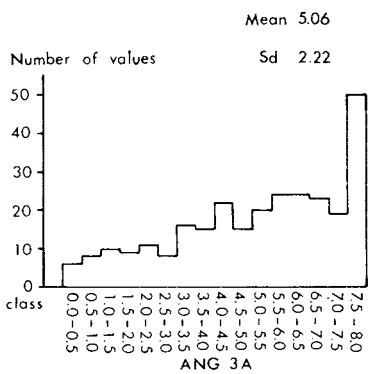
0.55-0.60
0.60-0.65
0.65-0.70
0.70-0.75
0.75-0.80
0.80-0.85
0.85-0.90
0.90-0.95
0.95-1.00
BARRF


Mean 5.06
Number of values
Sd 2.22
class
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0.5-1.0
1.0-1.5
1.5-2.0
2.0-2.5
2.5-3.0
3.0-3.5
3.5-4.0
4.0-4.5
4.5-5.0
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7.0-7.5
7.5-8.0
ANG 3A

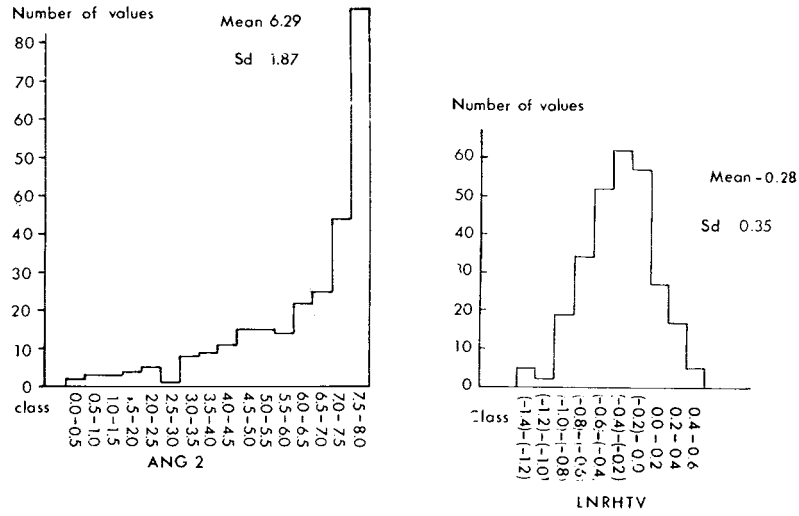
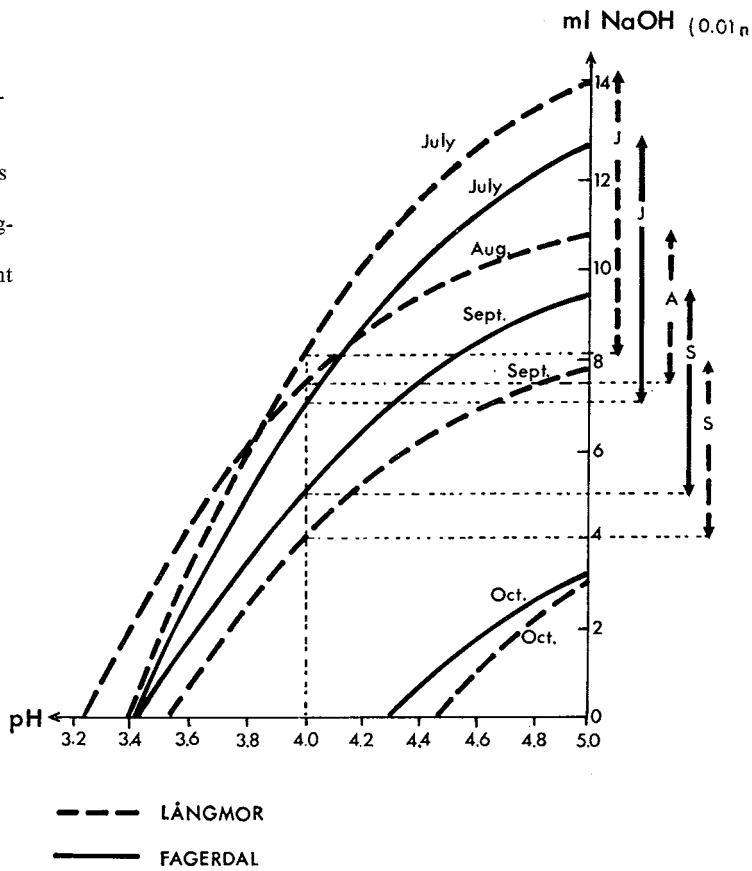


Figure 4. Distribution of the experimental material within the seven variables. Each figure describes the distribution of 280 plot means.

Figure 5. Examples of buffer capacity measurements. Titrations of needles of individuals of family 75. The needles were collected on two occasions in Fagerdal and on three occasions in Långmor. Each graph is the result of a measurement of an individual.



Two grammes of frozen needles was taken from each individual. 50 ml of distilled water was added, after which the mixture was homogenized in a mixer (Sorvall, Omnimixer 230) for 6 minutes and in ultrasound (Varian areograph) for 15 minutes. In both operations cooling by water was used. Then the mixture was filtered through a millipore filter (Satorius Membrane filter GmbH, size of pores 0.1 μ). 5 ml of destilled water was added to 25 ml of the filtrate, after which titration, using a solution of 0.01 N NaOH, was carried out to bring the pH-value of the solution up to 5.0. The titration was carried out by means of a potentiograph (Potentiograph E 536, Metrom Herisan).

The area under the titration graph was calculated. The area, enclosed by the titration graph, was abscissa and the ordinate at pH 5.0, was taken as a measure of the buffer capacity of the homogenized needles (Figure 5.) This measure is called buffer capacity I. The area was expressed as the number of integration units (IU).

For samples of needles with a pH-value below 4.0 the buffer capacity was also calculated according to another definition, called buffer capacity II. According to Henderson and McDonald (1962) this measure reflects the consumption of the base when titration between two fixed pH-values is performed. In this investigation the pH interval of 4.0—5.0 was chosen. The measure was expressed in ml of NaOH, in this case equal to milliequivalents $\times 10^{-2}$ (Figure 5).

3.4 **Methods of assessment and statistical analyses**

Of the seven variables used for measurements or assessments of the degree of attack, only three are parametric. The four non-parametric variables, (ANG1), (ANG2), (ANG3A) and (ANG3B), are not adequate for use in variance or regression analysis (Siegel, 1956; Conover, 1971). Only the medians of the families have therefore been calculated for these variables (Appendix 1). The other three variables are parametric, but if the values are to be used for regression analysis or analysis of variance, they should also have normal distributions and homogeneous variances (Snedecor and Cochran, 1967). None of the variables completely meet both of these requirements (Table 4). The variable based on change of growth (LNRHTV), however, better meets these requirements than do the other two. This variable should be considered the most suitable for the statistical calculations.

4 Results

4.1 Testing for resistance to Lophodermium, 1974—1976

The observations and measurements of the degree of attack to each seedling imply an assessment of a value influenced by three groups of factors: environmental factors at the experimental site, genetic factors in the host seedling, genetic factors in the pathogen, and any possible interactions.

In Appendix 1 the means and medians of the seven variables were grouped in a falling scale according to the values of variable (LNRHTV). The means of the families, parents and provenances for (LNRHTV) were arranged in Figure 6. These abridged results reveal considerable differences in the degree of attack within the experimental material. When attack is measured by needle loss, family N12×X26 has the lowest mean, 0.51. Family N6×N4, however, had the lowest reduction in height growth (Appendix 1). The family with the greatest needle loss was E5055×X26, while family E5055×N201 was affected most as regards height growth. In Figure 7 the separate dots represent plot means. This figure indicates that there is a strong correlation between vari-

♀ \ ♂		KÄRBÖLE X 18	KÄRBÖLE X 26	KÄRBÖLE X 52	RÖSKÄR B 101	RÖSKÄR B 102	RÖSKÄR B 103	ÖNSALA N 4	ÖNSALA N 201	ÖNSALA N 202	BOXHOLM E 4008	BOXHOLM E 4011	BOXHOLM E 4015			Open pollination	
KÄRBÖLE	X 9	-0.49*		-0.29*	-0.27		-0.49	-0.20			-0.15		-0.37	-0.32	-0.30	-0.52	-0.34
	X 10	-0.22	-0.21									-0.42		-0.28		-0.13	
	X 27		-0.43			-0.24			-0.14	-0.23		-0.42	-0.27*	-0.29		-0.37	
RÖSKÄR	B 1	-0.36		-0.23	-0.37*		-0.58	-0.12		-0.36	-0.24		-0.36	-0.32	-0.22	-0.39	-0.24
	B 19	-0.20*			-0.41	-0.11		-0.23	-0.24*		-0.24	-0.71		-0.31		-0.40	
	B 24		0.14	-0.21		0.24*	-0.04*		0.10	-0.12		-0.18	-0.10	-0.04		0.07	
ÖNSALA	N 6	0.14*			0.27		-0.10	0.49*		0.26*			0.05*	0.17	0.16	-0.04	0.09
	N 12	0.12	0.40					0.33*	0.40*					0.30		0.24	
	N 28		-0.06	0.08		0.20*	-0.07			0.02			0.18*	0.05		0.07	
BOXHOLM	E 5055		-0.46				-0.64		-0.73	-0.34			-0.65	-0.57	-0.42	-0.61	-0.52
	E 5024	-0.56	-0.55		-0.34	-0.09		-0.23	-0.22		-0.42	-0.42*		-0.34		-0.42	
	E 5053			-0.68			-0.24*	-0.27		-0.52	-0.24		-0.32	-0.39		-0.54	
		-0.24	-0.17	-0.26	-0.22	-0.03	-0.32	-0.07	-0.16	-0.21	-0.26	-0.43	-0.27	-0.22		-0.25	
		-0.22			-0.21			-0.15			-0.31						

Figure 6. Average change of growth (LNRHTV) caused by the attack of *Lophodermium*.
(LNRHTV) = ln [(Height$_2$ – Height$_1$)/Height$_1$].
Height$_1$ = height of seedlings before the attacks.

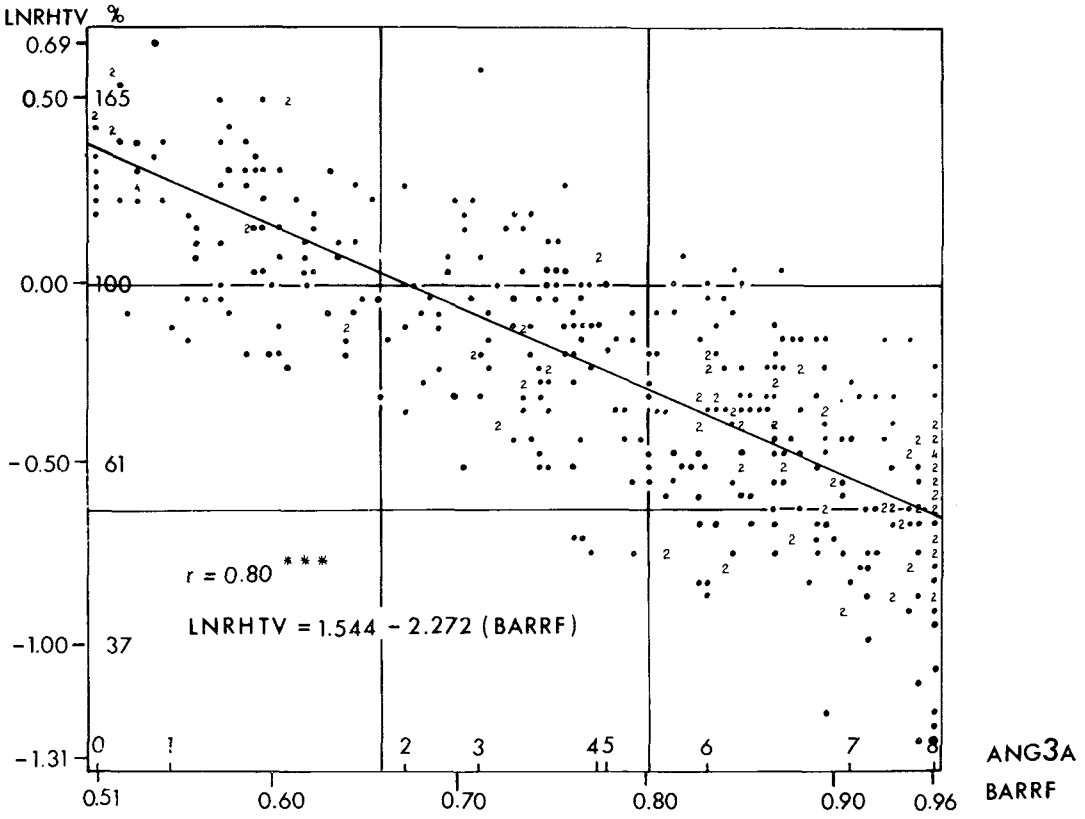


Figure 7. Relation between logarithmic values of change of growth (LNRHTV) and needle loss (BARRF). (ANG3A) = needle loss at different degrees of attack after the third infection (see Table 3). % = relative height growth.

ables (LNRHTV) and (BARRF). The needle loss corresponding to the nine classes of estimated degree of attack, (ANG3A), has also been indicated on the abscissa in Figure 7.

If the experimental material is divided into crossings between the four provenances, 16 subpopulations are obtained. Comparable with the subpopulations are the four populations representing the progeny of the provenances after open pollination. In Figure 8, the distribution of degree of attack within these 20 populations is shown. The figure refers to the assessment values after the third attack (ANG3A). From this figure it appears that the five populations which are progenies of mother N also show the highest resistance. In the group, N×N, 52 % of all observations are in the degree 0 class. The group, E×X, has the highest number of degree 8 observations, and other groups with E as a mother were seriously affected.

In general, the effects on the progenies of the mother trees seem to have been of greater importance to the resistance than the effects of the father trees, regardless of whether these effects are favourable or otherwise. If the two populations, N×E and E×N, are compared, we find that, in the former group, the degree of attack is relatively evenly distributed across the whole range from 0 to 8, while most of the individuals in group E×N belong to degree 7 and 8. In group N×N, the distribution of the attack provides an almost inverted image of the distribution within group E×E.

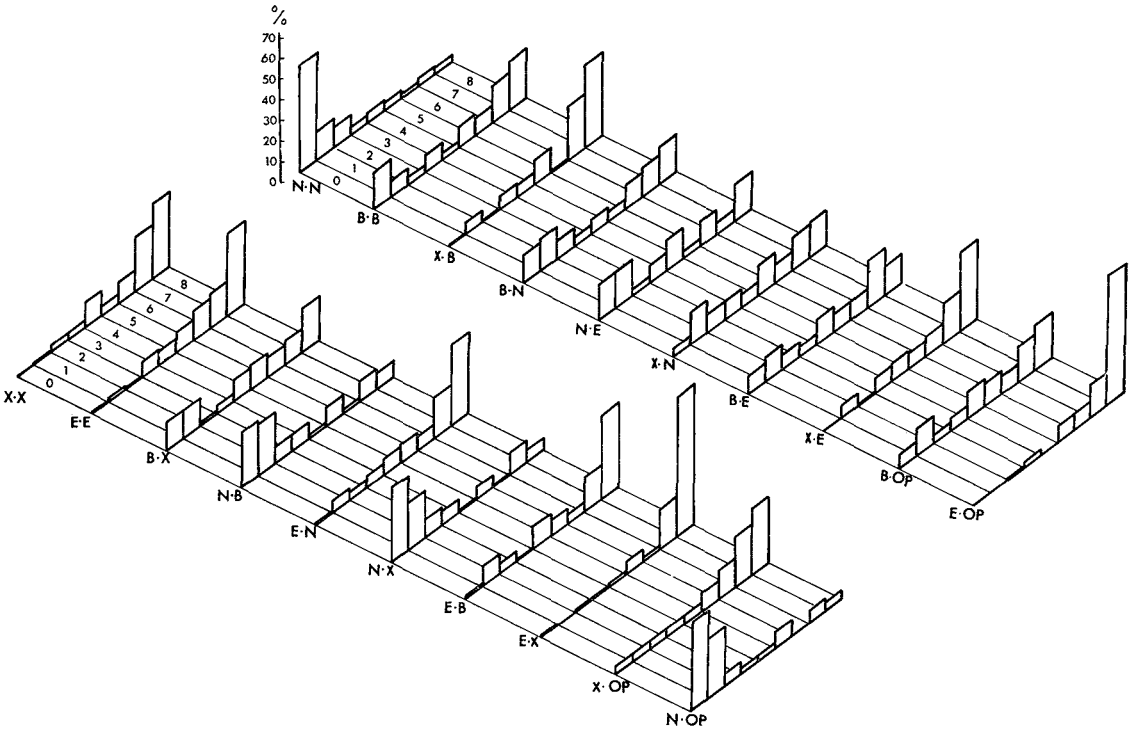


Figure 8. Percentage distribution of attack in 1976 (ANG3A) in 20 sub-populations of the experimental stock. The sub-populations consist of progenies after crossings within and between provenances, and progenies after open pollination.

The effects of the parent trees

The analyses of variance and regression analyses are based on plot means. The twelve half-sib populations as well as the 18 full-sib populations with less than five replications in the test have been omitted in these calculations. The 18 full-sib populations are indicated by asterisks in Figure 6 and by brackets in Appendix 1. The 56 full-sib populations completely represented in the experiment together consitute 280 plots. The following analyses start from the 280 plot means.

The statistical analyses were made by means of a statistical standard program at the Data Processing Centre, Umeå. The processing mainly follows the routine of an incomplete diallelic crossing design, worked out by Matern (1976).

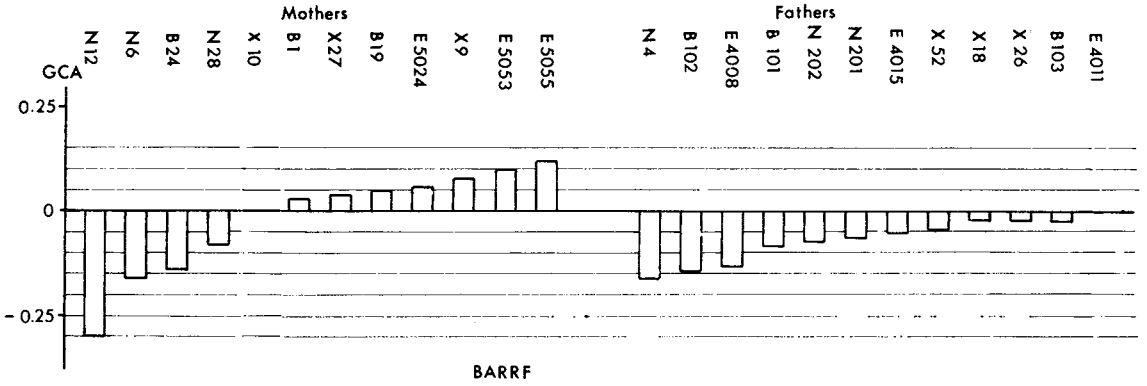
The genetic effects were analysed by analysis of variance (Table 5). As can be seen from the table, the component of variance of mothers is considerably larger than that of fathers. It should be noticed, however, that no given tree was both mother and father.

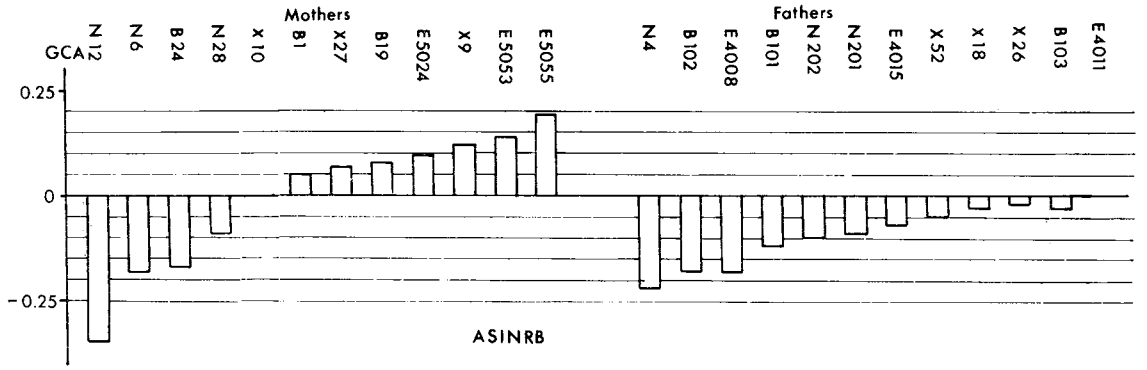
The average contribution of the individual parent trees to measured values of variables of the offspring were calculated by means of regression analysis. The following function was used:

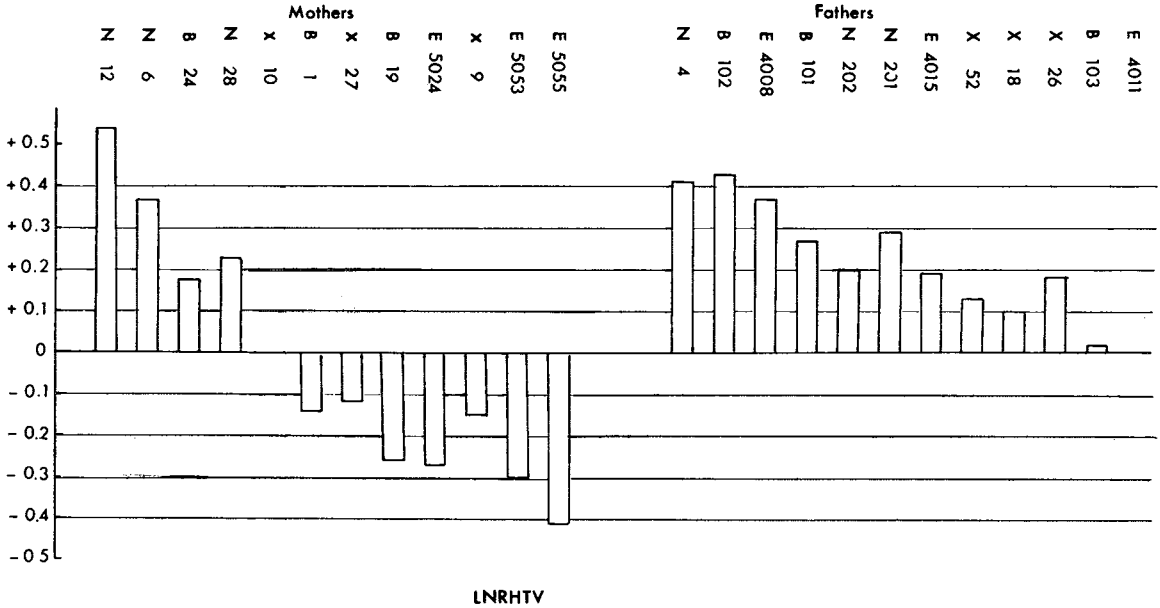
$$y_{ijk} = \beta_i + \gamma_j + s_{ij} + \varrho_k + \varepsilon_{ijk}$$

where

y_{ijk} = the plot mean of crossing ij in block k
β_i = the general combining ability of mother i
γ_j = the general combining ability of father j
s_{ij} = the specific combining ability in crossing ij
ϱ_k = the effect of block k







Figures 9. General combining ability (GCA) in respect of susceptibility to *Lophodermium* needle cast of 24 parent trees. The mothers are compared with mother X10, and the fathers with father E4011. The calculations are based on three different variables.

Table 5. Analysis of variance of the degree of attack in 56 progenies attacked by *Lophodermium pinastri*.

Source of variation	DF	BARRF	ASINRB	LNRHTV	Expected values of mean squares
Blocks	4	0.0720	0.1323	1.617***	$\sigma_\varepsilon^2 + 56\ \sigma_\varrho^2$
Mothers + (Mothers × Fathers)	44	0.0629	0.1039	0.368***	$\sigma_\varepsilon^2 + 5\ (\sigma_\beta^2 + \sigma_s^2)$
Fathers + (Mothers × Fathers)	44	0.0176	0.0326	0.142*	$\sigma_\varepsilon^2 + 5\ (\sigma_\gamma^2 + \sigma_s^2)$
Mothers × Fathers	33	0.0086	0.0152	0.077*	$\sigma_\varepsilon^2 + 5\ \sigma_s^2$
Error	220	0.0049	0.0085	0.048	σ_ε^2
Total	279				

Estimated components of variance after three different assessments of degree of attack

Comp. of var.	ASINRB (%)	BARRF (%)	LNRHTV (%)
$\hat{\sigma}_s^2$	0.00134 (4)	0.0007 (4)	0.0058 (4)
$\hat{\sigma}_\beta^2$	0.01773 (53)	0.0109 (56)	0.0582 (38)
$\hat{\sigma}_\gamma^2$	0.00349 (10)	0.0018 (9)	0.0130 (9)
$\hat{\sigma}_\varepsilon^2$	0.00851 (26)	0.0049 (25)	0.0480 (31)
$\hat{\sigma}_\varrho^2$	0.00221 (7)	0.0012 (6)	0.0280 (18)

$\hat{\sigma}_s^2$ = variance for specific combination ability (SCA)
$\hat{\sigma}_\beta^2$ = variance for general combining ability of mothers (GCA_M)
$\hat{\sigma}_\gamma^2$ = variance for general combining ability of fathers (GCA_F)
$\hat{\sigma}_\varrho^2$ = variance for effect of replication
$\hat{\sigma}_\varepsilon^2$ = variance for error

ε_{ijk} = the error of crossing ij in the kth replication

A detailed description of the calculations is given in Appendix 2.

The values of the general combining abilities (GCA) are reproduced graphically in Figure 9. Since the GCA of all parents cannot be calculated (owing to lack of degree of freedom), a father and a mother are reference values of the eleven calculated values of fathers or mothers. These parents are father E4011 and mother X10.

As can be seen from Table 5, the relative size of $\hat{\sigma}^2_\beta$ was larger than $\hat{\sigma}^2_\gamma$. The effect of interaction between parents was significant but only on the level of 5 %. The same applies to the effect of fathers. The effects of mothers, however, were highly significant.

The reproduced general combining ability (GCA) in Figure 9 should, in all cases, be related to the two reference values of mother X10 and father E4011. As can be seen from the calculations of arithmetical means of parents (Figure 6), although not based on exactly the same values, the means of the eleven other fathers were in most cases larger than the mean of E4011.

As regards the GCA of the fathers, smaller relative differences than among those of the mothers were found in all of the calculations. The rank of the reproduced values (GCA) was much the same, whatever variable was used in the calculation.

With the analyses of variance based on plot means, the variance of error (Table 5) did not show the variation between individuals of the same family within plots. As shown in Table 6, such variation occurred in the material, especially within certain families (page 34).

Table 6. Analysis of variance of individual values within families and within plots.

Source of variation	DF	Mean squares
		LNRHTV
Between families	85	1.63
Within families	1849	0.15
Between plots within families	321	0.07
Within plots within families	1528	0.16
Total	1934	

Analyses of the attack within and between provenances.

Table 7 shows an abridgement of analysis of variance. The material was divided into three hierarchic levels.

Level 1: Groups of provenance crossings formed either after crossings within provenances or between provenances

Level 2: Groups of families formed within the same provenance crossings

Level 3: Families within provenance crossings

At level 2 there were two types of provenances crossing, viz. crossings between provenances and crossings within provenances. No distinction was made between reciprocal provenance crossings; thus crossing E×X was considered equivalent to X×E, for instance. Therefore, there are only six such groups of families. In Table 7 these six groups of crossings are called B-crossings. The four groups of families formed by crossings within the provenances, X×X, B×B, N×N and E×E, are called

Table 7. Analysis of variance of degree of attack in 56 progenies (families) of pine, *Pinus sylvestris,* attacked by *Lophodermium pinastri.* The analysis is made with regard to the division of families into provenances. W-crossings = families within provenances, B-crossings = families within crossings of provenances. The degree of attack is assessed on three different principles, (BARRF), (ASINRB) and (LNRHTV) (see text).

Source of variation	DF		Mean squares		
			BARRF	ASINRB	LNRHTV
a Block		4	0.072	0.132	1.617
W-crossings					
b Between provenances	3		0.040	0.063	0.200
c Between families within provenances	7	10	0.020	0.041	0.180
B-crossings					
d Between provenances	5		0.247	0.413	1.492
e Between families within provenances	39	44	0.036	0.059	0.220
f Difference between W- and B-crossings	1	55	0.068	0.106	0.116
g Error		220	0.005	0.009	0.048
Total		279			
	Fe/c		1.80	1.44	1.222*
	Fd/b		6.18	6.56	7.460*
	Fd/e		6.86	7.00	6.782***
	Fb/c		2.00	1.54	1.111*

W-crossings in the table.

The calculated quotients of variance in Table 7 imply a comparison within level three between families formed in two different ways, i.e. the quotient between the mean squares in line c and e, and also a comparison within level two, i.e. the quotient between the mean squares in line b and d—a comparison between two different types of population: within provenances or between crossings of provenances.

The quotient Fe/c is not significant at $p = 0.05$, which means that there is no certain reason to suspect less variation between families formed within provenances than between families formed between provenances. Neither does the quotient Fd/b indicate greater differences between the six groups, comprising of the provenance crossings, than between the four groups originating from crossings within the four provenances. It should be noticed, however, that W-crossings indicated by an asterisk in Figure 2 were not included in these analyses.

The only quotient of variance that is clearly significant is Fd/e, i.e. the quotient between B-crossings and families within these provenance crossings. By disregarding the probable differences between reciprocal crossings, the calculations probably lead to an underestimation of the mean square in line d and an overestimation of the mean square in line e.

4.2 Analyses of the buffer capacity of the needles

The first year's analyses of needles collected on the 25th and 26th of July and on the 2nd and 3rd of October showed that the time of sampling was of great importance to the buffer capacity. The July needles had a pH-values about 1 unit lower than did those from October. Therefore, the titration to a pH of 5.0 of the July needles resulted in a value of buffer capacity I that was about 10 times larger than the corresponding value of the October needles.

The material collected at the end of July was relatively limited in extent (samples of three individuals of each family at Långmor and Fagerdal, Appendix 3a).

Measurements of these needles showed, however, that the habitat of the pine seedlings was of great importance to the buffer capacity. Needles from seedlings growing at Långmor showed a significantly higher buffer capacity than did those from Fagerdal, when the same families were compared (Figure 10 and Table 8). The mutual relation between the means of the eight families was generally similar at both sites.

In the following year, 1976, samples of needles were taken on the 4th of August at Långmor and on the 2nd and 3rd of September at Långmor and Fagerdal. To increase the reliability of the measurements, samples were taken from a greater number of seedlings (15—24 per family) but only from two susceptible and two resistant families (Nos. 55, 56 and 75, 77, respectively). In the two resistant families at Långmor, buffer capacity I at the beginning of August was 76 and 72 IU, respectively, as against 78 and 79 IU in July. The corresponding values for the two susceptible families were 72 and 89 IU in August, and 80 and 94 IU in July (Table 8). On the 1st of September 1977 needles were collected from the same four families at Långmor. The analysis of those needles showed a similar result as did that of the previous year (Figure 10).

It appeared from the September samples of 1976 that buffer capacity I had decreased to a value of between 33 and 48 IU, and that the needles collected at Fagerdal showed lower values than did those collected at Långmor when compared with the same families. The means of families showed the same relative magnitude within the two groups at both sites, i.e. family 56 had a higher buffer capacity than family 55, and family 75 a higher buffer capacity than family 77. Between the two groups no significant diferences could be found at any of the sites. From the analysis of September needles of 1977 the same order of magnitude was showed, however with a bigger buffer capacity in the susceptible than in the resistant families (Figure 10). The measurements made in September, 1976, are probably the most representative of the whole

Table 8. Means of buffer capacity I in eight selected families.

	Resistant				Susceptible			
Family no.	75	77	73	74	55	56	53	58
(LNRHTV)	0.13	0.40	0.33	0.41	−0.65	−0.73	−0.64	−0.47
Site and month								
Långmor July 75	78.40 ± 3.93	79.00 ± 0.47			80.00 ± 6.44	94.00 ± 3.41	66.20 ± 5.12	77.90 ± 7.17
Långmor Aug. 76	75.64 ± 1.59	71.69 ± 2.59			72.45 ± 1.91	88.78 ± 3.20		
Långmor Sept. 76	36.94 ± 1.67	33.24 ± 1.18			44.02 ± 1.79	47.82 ± 2.20		
Långmor Sept. 77	26.39 ± 1.40	22.06 ± 1.87			39.65 ± 1.69	42.95 ± 2.79		
Långmor Oct. 75	6.30 ± 1.13	5.60 ± 0.50			7.10 ± 0.80	6.90 ± 1.92	6.60 ± 0.93	4.50 ± 1.05
Fagerdal July 75	65.10 ± 4.94	67.80 ± 5.07	54.90 ± 4.38	59.70 ± 1.71	58.30 ± 3.64	70.60 ± 0.70	79.20 ± 3.77	72.50 ± 1.07
Fagerdal Sept. 76	35.23 ± 1.96	32.71 ± 1.72			34.60 ± 1.54	42.00 ± 2.52		
Fagerdal Oct. 75	6.40 ± 0.32	4.70 ± 0.53	6.10 ± 0.81	6.50 ± 0.32	4.20 ± 1.02	10.20 ± 2.46	6.40 ± 2.23	4.80 ± 1.05

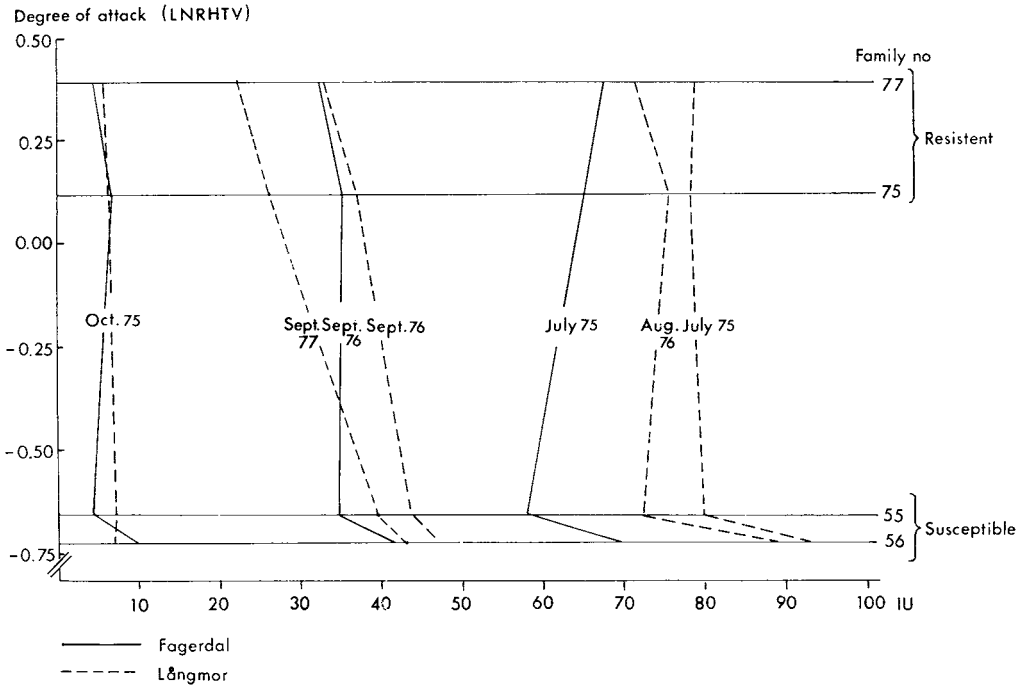


Figure 10. Buffer capacity I in needles of four full-sib populations.

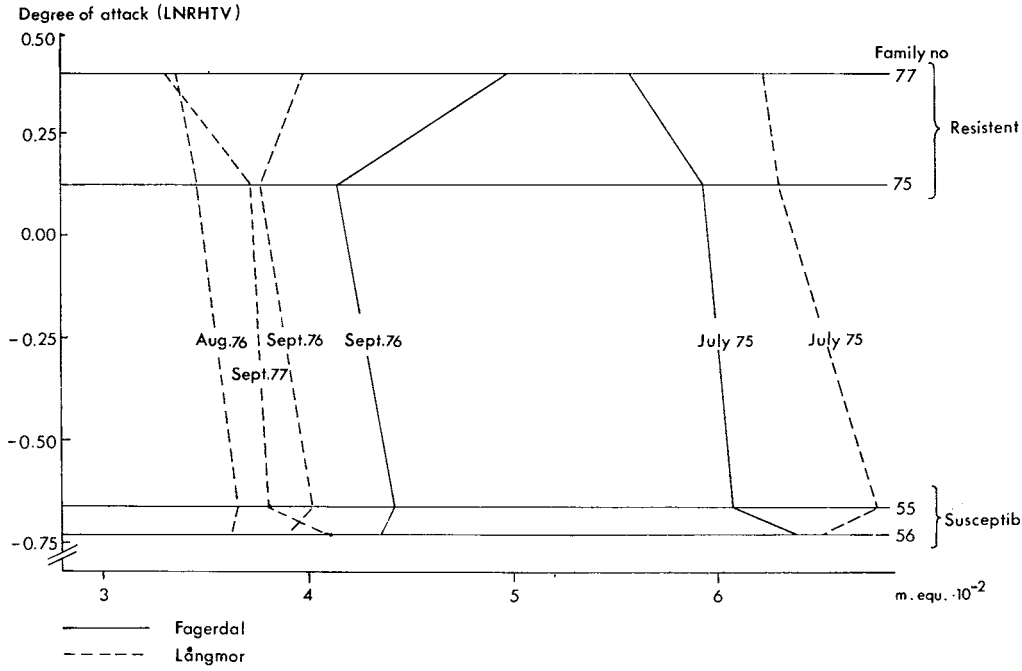


Figure 11. Buffer capacity II in needles of four full-sib populations.

Table 9. Analysis of variance of buffer capacity I and buffer capacity II in pine needles of four progenies (fam. 55, 56, 7, and 76 during July 75, Aug. 76 and Sept. 76.

Source of variation	DF	Mean squares		
		Buffer capacity I	Buffer capacity II	
Between mothers	1	3300.32	0.171	a
Within mothers	237	463.29	0.827	b
Between families within mothers	2	3270.29 440.10	1.305 0.823	c d
Within families	235			
Total	238			
	F^{c}/d	7.431***	1.59°	
	F^{a}/c	1.009°		
	F^{a}/b		0.21°	

Table 10. Analysis of variance of buffer capacity I and buffer capacity II in pine needles of four progenies (families) at two sites, Långmor and Fagerdal, in September 1976.

Source of variation	DF	Mean squares		
		Buffer capacity I	Buffer capacity II	
Between places	1	968.55	10.27	a
Within places	145	79.73	0.25	b
Between mothers within places	2	1174.07	1.52	c
Within mothers	143	64.42	0.23	d
Between families within mothers	4	197.12	1.90	e
Within families	139	60.60	0.18	f
Total	146			
	F^{e}/f	3.25*	10.59***	
	F^{c}/e	5.96°	0.80°	
	F^{a}/c	4.91°	5.42°	

investigation, since the resistant and the susceptible groups are each represented by measurements of about 35 individuals both at Långmor and Fagerdal. The results reveal, however, that no significant differences can be shown between the groups, i.e. between progenies of mother trees (Table 10). The differences found earlier between families within groups with different degrees of resistance also existed in the September needles (Table 10).

Tables 9 and 10 show that the differences between families within mother trees, i.e. between families within the resistant and the susceptible groups, are significant. On the other hand there is no significant difference between the two groups.

In order to test the accuracy of the analysis, two lots of measurements of the buffer capacity in the needles were made on six individuals. The percentage divergencies of the six remeasurements are shown in

Table 11. Means of buffer capacity II in eight selected families.

	Resistant				Susceptible			
Family no.	75	77	73	74	55	56	53	58
LNRHTV	0.13	0.40	0.33	0.41	−0.66	−0.73	−0.64	−0.47
Site and month								
Långmor July 75	6.30 ± 0.06	6.23 ± 0.12			6.77 ± 0.34	6.50 ± 0.06	6.10 ± 0.00	6.57 ± 0.07
Långmor Aug. 76	3.46 ± 0.06	3.36 ± 0.05			3.66 ± 0.10	3.64 ± 0.07		
Långmor Sept. 76	3.78 ± 0.10	3.99 ± 0.07			4.02 ± 0.06	3.89 ± 0.05		
Långmor Sept. 77	3.74 ± 0.06	3.27 ± 0.12			3.78 ± 0.07	4.10 ± 0.06		
Fagerdal July	5.93 ± 0.12	6.57 ± 0.41	4.93 ± 1.08	6.50 ± 0.36	6.07 ± 0.09	6.40 ± 0.12	6.37 ± 0.20	6.63 ± 0.18
Fagerdal Sept.	4.14 ± 0.13	4.97 ± 0.13			4.42 ± 0.07	4.36 ± 0.14		

Appendix 3 (page 61). These remeasurements were made on needles collected in July. The six divergencies have a variance of 3.40, which means that a mean based on 20 separate measurements has a standard error due to incorrect measurements, that can be estimated to less than ± 0.5 %.

Buffer capacity II (according to definition, page 18) could not be calculated for needle samples taken in October, since the pH-value of the needles exceeded 4.0. In all other samples buffer capacity II was calculated between pH 4.0 and pH 5.0 (Appendix 3b). It was found that during the period when the measurements were made the buffer capacity was highest in July and lowest in August, after which it rose again in September (Table 11, Figure 11). This seasonal rhythm was the same for all families at both sites.

If the whole material is viewed without regard to time and place of collection, no certain differences in buffer capacity II were found between groups with different degrees of resistance (Table 9). However, in the needles collected in September, 1977, there was a highly significant difference between families *within* the two groups of maternal offspring (Table 10).

In conclusion, with the definitions of buffer capacity used, these traits in the needles vary widely in different families. In the material analysed, no correlation between the buffering properties of homogenized needles and resistance to *Lophodermium* is apparent. Between those sib populations where significant differences in buffer capacity occurred, the difference in the degree of resistance was so small that it is impossible to express an opinion as to whether or not there is a positive or negative correlation. The results indicate, however, that the buffer capacity is a trait characteristic of every full-sib population.

5 Discussion

5.1 Methods of measurement

The assessments of the degree of attack, made during May in 1974, 1975 and 1976, were estimations of the proportion of needles on each seedling that had been discoloured in a way typical of attacks by *Lophodermium* (Lagerberg, 1913; Langner, 1933). In making these assessments it was not merely a matter of measuring objectively the resistance to *Lophodermium* of each pine. Nor could the grading of the damage be said to be directly proportional to a measurable character coupled with resistance. The degree of attack after the second infection, (ANG2), was apparently higher in most cases than the corresponding values after the third infection, (ANG3A). There could be several reasons for this. At the revision in May, 1976, (ANG3A), there were three whorls of branches on each seedling. Since, without exception, the whorl at the bottom lacked needles, the assessment in May, 1976, (ANG3A), was based on the sum of the degrees of attack sustained by the two upper branch whorls. In 1975, however, there were only two whorls of branches to assess. The sum of these two whorls was (ANG2).

On a comparison of the observations from the first and last years, it was found that the plots assessed as having sustained moderate attacks in the first year had been assessed with considerable variation two years later. It was also noticed that plots attacked severely in the last year, had been assessed with much greater variation two years earlier than those with a low degree of attack in the last year. According to Figure 12 it seems as though, in a large part of the material, the degree of attack had been changed from lower to higher values on a comparison of the first and the last assessment occasions. This can partly be explained by the inability of some seedlings to grow out of the danger zone while under the influence of the pathogen.

The increasing degree of attack during the three years could also be due to the increase of the infection pressure on the experimental site, caused by the production of spores from a steadily increasing quantity of needls. The change of attack was not noticed in seedlings that had already sustained maximum-intensity attacks at the time of the first revision, while the effect manifested itself in other parts of the test material.

The scale of degree of attack, divided into nine degrees, was transferred at the last year's revision to an objectively determined needle loss, (BARRF), for each degree of attack, by the method described in Table 3. It appeared in this calculation that even the seedlings assessed as the least affected, i.e. showing degree 0 of attack on two upper branch whorls, had lost on the average 50 % of the needles between October and May. The calculations showed that the needle losses were considerably larger throughout than was evident in the ocular assessment of the proportion of discoloured needles (ANG3A). Except for those attributable purely to errors of judgement, the possible causes include the fact that some of the discoloured needles could have been shed before the revision in May, 1976, or that some of the affected needles had not had time to be stained at the revision, and were so loosely attached to the dwarf shoots that they fell off, while still green, when the branches were collected. Moreover, the calculations of needle losses showed that some of the nine classes of (ANG3A) could not be distinguished from each other. Therefore, several adjacent classes were grouped

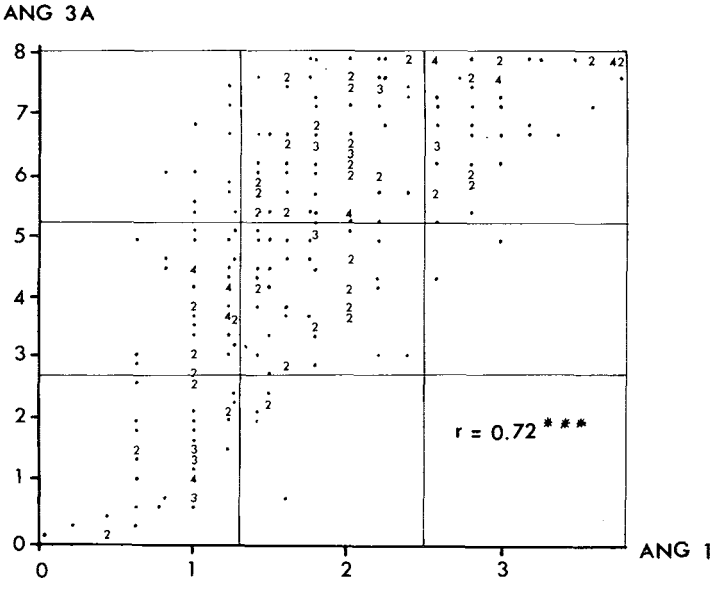


Figure 12. Comparison between the degree of attack in the first year (ANG1) and the last year (ANG3A).

together, with the result that only three classes were obtained (page 14). The variable that was formed (ANG3B) proved to be unsuitable for statistical analysis (page 18).

Further reasons for the change in the degree of attack during the three years were the different degrees of exposure of the branches to the pathogen and the change in the microclimate in the upper parts of the pine seedlings during the period (Blair, 1970). The needles on the lower branches were probably exposed to a higher infection pressure, resulting in a more severe attack. Besides, the microclimate nearest the ground was probably more conducive to attacks. The seedlings which were able to grow out rapidly of the danger zone close to the ground therefore partly escaped the attacks by the pathogen. This growing ability could, however, be caused by a real resistance in the seedlings at the first time of attack when they were all at a susceptible height. If a seedling was affected then, its chance of growing out of the danger zone was reduced. The effect of an attack was therefore cumulative from one year to the other.

At the time of the first attack the differences in height between all of the seedlings in the sample were relatively small. The mean height was 56.7 cm with a standard deviation of 9.4. If the seedlings were divided into two groups, one group consisting of progenies of the five mothers which later proved to provide the highest degree of resistance, and the other consisting of progenies of the seven mothers which later proved to provide the lowest degree of resistance, the mean height before the first attack would be 55.9 ± 0.4 cm in the former group and 57.1 ± 0.4 cm in the latter. Thus, the proportion of the sample with seedlings whose height growth was most affected by the *Lophodermium* attack had an initial position superior to that of the resistant seedlings in the sample. When the experiment was finished the mean height of the entire sample was 98.9 cm, with a standard deviation of 37.3.

Schütt (1966) found that it can be very difficult to determine the resistance from visual inspections of the attack, since sometimes there is no correlation between the assessed degree of attack, and the decrease in growth or seedling mortality. In an experiment he found that there was no correlation between assessed degree of attack and ability to recover, when the ability to

recover was arranged in three classes, great, minor and medium, the seedlings most severely affected proved to belong to either the great or minor classes, with the class of medium recovery ability consisting of slightly affected seedlings.

It is clear from the above that there is a great need for an objective method of measurement to determine the total effect of the *Lophodermium* attack in pine seedlings.

From a practical point of view, there is scarcely any reason to distinguish the resistance in a certain part of the seedling (for instance, at a certain height above the ground) from the resistance depending on rapid growth out of the danger zone. In the regions where selection for resistance to *Lophodermium* is of interest, the selection criterion should be the total effect of the *Lophodermium* attack on the chances that the pine seedling has to survive and grow.

The relative height growth, $(Height_2 - Height_1)/Height_1$ expresses the effect of the *Lophodermium* attack on the growth of the seedlings. The relative height growth reflects the change in growth and is probably a measure of the genetic traits of the pine seedlings in respect of their ability to resist the effect of the attack on growth.

According to the laws formulated by Mitscherlich (1909), the biological growth development follows a function of products which include the environmental factors influencing growth and having multiplicative effects. The height growth of a tree can thus be equated to the products of the effects of a great number of different factors. The *Lophodermium* attack is one such factor affecting the growth of the pine seedling.

f(b) indicates the effect on the height growth of all biological factors, genetic as well as ecological, except for the factor which is influenced by the *Lophodermium* attack. The effect of this factor is indicated g(l). The following function of the growth (HTV) on a given occasion after the first instance of attack can then be formulated

$$HTV = f(b) \times g(l) \times \varepsilon$$

where ε indicates a random variable with $\ln(\varepsilon)$ having a normal distribution with the mean = 0.

Through conversion of the function of products into a function of logarithms, the multiplicative effects become additional. So the height growth on a given occasion can be written (cf. Sundberg, 1970):

$$\ln(HVT) = \ln[f(b)] + \ln[g(l)] + \ln(\varepsilon)$$

Since the *Lophodermium* attacks have not affected the growth at all at the time of the first measurement of the height, a measure of the effect of this factor is obtained by calculating the logarithm of the quotient, $\ln\left[\frac{HTV_2}{HTV_1}\right]$ This expression is indicated (LNRHTV), the natural logarithm of the relative height growth. The antilogarithm of the quotient corresponds to the effect of the *Lophodermium* attack on the height growth of a pine seedling. The average effect on a group of individuals, for instance, a family, is obtained by calculating the antilogarithm of the arithmetical mean of the (LNRHTV) of the individuals (Table 12). Table 12 also shows that the effect on the relative height growth largely agrees with the assessed degree of attack. There are a few exceptions, however; for instance, family 81, which grew 120 % of the height before the attack, in spite of the fact that the degree of attack, (ANG3A), has as high a median as 6. Family 20, on the other hand, grew only 84 % despite the degree of attack being assessed as minor.

5.2 Differences in the degree of resistance between provenances

In experiments for assessment of genetically dependent resistance to *Lophodermium* conducted in Germany, Poland and France on provenance material of *Pinus sylvestris*, the high resistance to *Lophodermium* of the northern provenances has often been pointed out (Schütt, 1957a; Hattemer, 1966; Siwecki et al, 1975; Lanier, 1968). Provenances with the lowest resistance in these

Table 12. Relative height growth as a percentage of the seedling height of the families before the Lophodermium-attack and their medians of attack (ANG3A).

Family no.	ANG3A	RHTV · 100
65	0	163
74	0	149
77	0	149
73	0	139
61	1	131
66	1	130
132	0	127
18	0	127
78	0	122
81	6	120
25	1	115
67	2	115
75	1	113
22	1	111
86	2	108
133	1	107
126	2	107
64	1	105
83	3	102
131	1	96
19	3	96
85	5	94
79	5	93
36	4	91
62	1	90
21	2	90
10	4	90
23	1	89
5	4	89
135	6	88
108	5	87
89	6	86
20	2	84
15	6	82
26	5	82
91	4	82
103	6	81
101	5	80
40	7	80
39	3	79
8	6	79
13	5	79
109	5	79
3	4	79
46	5	79
104	7	79
45	8	79
11	5	79
14	6	79
87	7	76
48	6	76
107	6	76
94	7	75
47	7	73

Table 12 continued

Family no.	ANG3A	RHTV · 100
57	7	71
35	5	71
6	7	70
7	7	70
4	7	70
136	7	70
1	7	70
90	8	70
124	7	68
125	6	67
9	6	66
37	6	66
106	8	66
98	7	66
128	7	66
38	8	66
110	8	65
58	8	63
93	8	61
88	7	61
49	7	59
134	7	59
129	8	58
42	8	58
41	7	57
2	8	56
130	8	54
53	8	53
55	8	52
51	8	51
12	7	49
56	8	48

tests were often German or French, while Scandinavian, Scottish and Finnish provenances as a rule evinced high resistance. Deviation with the provenances also occurred.

Kalela (1937) suspected a connection between the climate in the home region of the provenance and susceptibility to *Lophodermium*; the more maritime the climate, the greater the susceptibility. However, in the 20 provenances tested by Schütt (1964a), this was not always the case. Among the 20 provenances tested at 10 sites, the provenance of Bergen, for instance, proved to be very resistant at all of the sites.

Hagem (1926) found in an experiment in the south of Norway that of several Norwegian and Scottish provenances, the local

provenance best resisted attacks by *Lophodermium*.

The material tested in the present work consisted of full-sib populations after crossings within and between four provenances. All provenances originated from Sweden but their sites of origin differed widely; Kårböle in the north being situated at latitude 62° and Onsala in the south at latitude 57°. Other environmental factors also differed considerably at the four sites. Onsala and Röskär represent low-altitude sites while Kårböle has a relatively high altitude (Figure 1). As regards the maritime influence, Kårböle should be considered to be the least and Onsala the most maritime of the sites. The environmental factors characteristic of Onsala are also considered to correspond best to those of the experimental site, Tönnersjöheden. It should be noticed, however, that the provenances have been moved southwards, which, according to several authors, has been found to lead to decreased susceptibility, in contrast to the movement of provenances from south to north (Dengler, 1955; Schütt, 1964a; Siwecki et al, 1975; Przybylski, 1972). The original environments of the provenances dealt with can therefore be said to represent a relatively wide spectrum of site factors. On the basis of the results, however, it is impossible to trace a continuous increase or decrease in the degree of attack, relative to the geographical or maritime position. But there is a clear connection between degree of attack and provenance. The families which are progeny, completely or partly, of the provenance Onsala were attacked least, while those which were completely or partly progeny of the provenance Boxholm were most affected. The provenances Kårböle and Röskär occupied an intermediate position.

The ability to survive shows the same trend as do the other results (Table 13). The mother trees from Boxholm gave the poorest progeny. Schütt (1964a) and Hattemer (1966) have shown that the degree of attack is affected by the interaction between provenance and experimental site. This may be explained by the fact that the general vitality of the host plant and, consequently, the resistance of *Lophodermium* depends on the site conditions (Gäumann, 1951; Schütt, 1958). Therefore, it is very likely that the material originating from Kårböle has a different seasonal rhythm as regards wintering and sprouting, for instance, than the material from Onsala (Hagner, 1970). It is therefore probably essential that the test for resistance be made within the same climatic zone as that in which the seedling stock will be used in practical reforestation.

Table 13. Share of individuals killed by *Lophodermium* needle cast after three years of attack.

	X	B	N	E	OP
X	4 %	5 %	0 %	2 %	4 %
B	2 %	0 %	3 %	1 %	2 %
N	1 %	2 %	1 %	6 %	0 %
E	9 %	8 %	5 %	3 %	11 %

Another factor, the importance of which could not be tested in this experiment, is the variation within the species *Lophodermium pinastri*. The inoculum was collected in a young stand at Tönnersjöheden. What the result would have been if the inoculum had been collected in another place is not clear. Variation within the fungus species exists (Stephan, 1973; Staley, 1975). The pathological importance of this variation has not been proved, however (Mayr, 1902; Millar and Watson, 1971).

5.3 Differences in the degree of resistance between sib populations and between individuals

In the 1950's Schütt (1957a) established orchards of single individuals of Scots pine, selected with respect to their resistance to *Lophodermium*. Clear differences could be found in these orchards between individuals selected from the same stand.

In the test material at Tönnersjöheden, it was found that great individual differences in the degree of attack can occur within

certain full-sib population. In 1976, for instance, both very slightly affected and very strongly affected individuals occurred within family 23, with the frequency distribution shown in Figure 13. However, as a rule the degree of attack within the families varied less, (e.g. families 77 and 58 both progeny of the same father).

Viewed at the family level, the results show pronounced differences within the same provenance crossings (Figure 6). The differences were largest within crossings of different provenances, which can also be seen in Table 7. Within group B×N, family 22 was best with (LNRHTV) = 0.10, while the corresponding value for family 6 is −0.36 (Figure 6). Within group B×E, the family means of (LNRHTV) varied between −0.10 and 0.71 for the mean degree of attack of the families. In the families where B24 was a mother, relatively high family means could be observed. Concerning the overall mean and the means of parents, B24 and the three mother trees from Onsala contributed to the increase of the means of all twelve fathers. The principal parent trees providing progenies of low resistance were the mother trees from Boxholm and Kårböle, as is also evident in Figure 9.

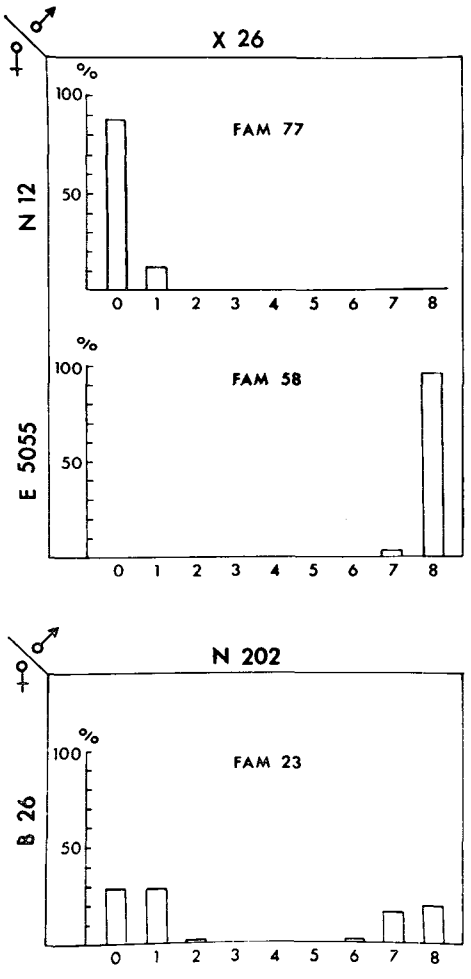


Figure 13. The distribution of degree of attack (ANG3A) within three full-sib populations.

5.4 The buffer capacity of the needles

According to Hägg (1969) the buffering effect is defined as the derivative of the titration graph. Therefore, the buffering effect varies according to the pH-value. In this investigation, "buffer capacity" has been defined in two different ways. According to Scholz and Stephan (1974), the buffer capacity is equal to the area between the titration graph, the abscissa and the ordinate at pH 5.0 (Figure 5). Thus, this buffer capacity, buffer capacity I, depends on the buffering effect throughout the titration, and the shape of the whole titration graph is considered. But in the cases where the investigated samples of needles have different pH-values, the buffer capacity will also depend on the acidity of the needles. It is therefore possible that the titration of two needle samples will provide curves of the same shape on the titration graph, but with a different buffer capacity (Figure 5). When buffer capacity II is measured the consumption of base (or acid) in titration between two fixed pH-values is determined (McDonald and Henderson, 1962). Thus, buffer capacity II is a measure of the buffering effect between two pH-values, irrespective of the acidity of the needles at the beginning of the titration.

Differences in buffer capacity between seasons and sampling sites (Figure 10) are no doubt the result of different pH-values in the homogenized needles. The acidity of the needles together with other chemical

Table 14. Change of pH-value in the needles in the experimental sample at Långmor and Fagerdal during July—October.

	Långmor	Fagerdal
July (1975)	3.36 ± 0.02	3.42 ± 0.01
Aug. (1976)	3.12 ± 0.02	
Sept. (1976)	3.46 ± 0.02	3.60 ± 0.01
Oct. (1975)	4.37 ± 0.02	4.30 ± 0.02

characters are correlated with physiological processes, and therefore have a genetically and climatically settled seasonal rhythm (Rudloff, 1972; Juvonen, 1970). When investigating the change of pH-values in pine needles during the year, Wille (1927) found in one test that this value rose during autumn and winter from a minimum value of 3.6 in July, reaching a maximum value of 4.8 in May. The seasonal rhythm of pH-values of seedlings, genetically adapted to a climate with given light and temperature conditions will therefore vary from site to site. In the north of Sweden the photoperiod is longer than in the south during July, August and most of September. From this it follows that during this season, photoperiodically dependent processes in the south of Sweden will precede similar processes occurring in the north of Sweden, in the case of a given pine provenance. This phenomenon is supported by the measurements made on needles from Fagerdal and Långmor during July—October in respect of buffer capacity (Figure 10) and pH-value (Table 14).

Even if a comparison of buffer capacity I of the needles is made between seedlings from a given site and time, the comparison will only be purposeful if the pH-values of the needles are equal, since the buffer capacity, defined in this way, will depend on both the concentration of the acids and their buffering properties.

When buffer capacity II is measured, only the amount of base required to increase the pH-value from one level to another is considered. This value does not tell us anything about the shape of the titration graph within the pH-interval. In the analyses made in this investigation, the concentration of the acids in the homogenized needles was highest in August (Table 14). In spite of this, buffer capacity II of all four families was lowest in this season (Figure 11). Since buffer capacity I was still relatively high in August (Figures 5 and 10), other acids with lower buffer capacity may be the reason for the low pH-value at this time, rather than the effect of the acids on the pH-values of the needles in July and September (Barton, 1967; Minimakava, 1976).

A possible reason why the results of this investigation did not demonstrate the same good correlation between buffer capacity and resistance as those recorded by Scholz and Stephan (1974, 1975a) might be that the variation in the degree of resistance of the investigated families may have been larger than the variation in the degree of resistance between the clones in the Scholz and Stephan study. In the latter case the clones were grafts of trees believed to be resistant from the beginning. Nonetheless these clones proved to have different levels of resistance after exposure during a number of years to strong infection pressure.

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Sammanfattning

Inledning

En förutsättning för resistensförädling är, att det finns riktiga mätmetoder för resistens och att det finns resistens, som är genetiskt betingad. Målsättningen med detta arbete är att undersöka:

- hur resistens mot *Lophodermium* bör mätas,
- om resistens mot *Lophodermium* är genetiskt betingad och hur ett genetiskt urval för resistens ur en given tallpopulation kan utföras,
- om någon indirekt urvalsmetod kan användas.

Sjukdomen tallskytte

Tallskytte orsakas av en svamp, *Lophodermium pinastri*. Svampangreppet leder till en för tidig barrfällning. Tallplantor infekteras vanligtvis under sensommaren—hösten, då svampens sporer utvecklas på tidigare avfallna barr på marken. Angreppet utvecklas under våren då barren torkar och avstöts från tallplantan. Svampen har således ett parasitiskt och ett saprofytiskt utvecklingsskede. Sjukdomen orsakar ekonomiskt betydande skador i plantskolor och i kulturbestånd i södra Sverige.

Tallskytteangrepp medför större eller mindre förlust av barr för tallplantor, vilket innebär nedsatt kolsyreassimilation och förlust av upplagsnäring.

Resistensmätning bör ske på ett sådant sätt, att mätvärdena ger uttryck för svampangreppets effekt på tallplantors förmåga att överleva och tillväxa i sådana miljöer där svampen vanligen angriper.

Material och metoder

Resistens mot tallskyttesvampen *Lophodermium pinastri* testades hos 74 helsyskongrupper och 12 halvsyskongrupper av fyra olika provenienser av *Pinus sylvestris* i en plantskola på Tönnersjöhedens försökspark i södra Sverige. Varje syskongrupp bestod av ca 25 individer. De 86 grupperna var avkommor efter 12 moderträd och 12 faderträd.

Plantorna infekterades artificiellt med *Lophodermium* vid tre års ålder. Infektionsmaterialet hade insamlats i ett angripet tallbestånd på Tönnersjöheden.

Plantornas angreppsgrad bedömdes tre år i följd enligt en subjektiv bedömningsskala. Det tredje årets bedömning jämfördes med en objektiv mätning av barrförlusten inom varje klass av den subjektiva bedömningsskalan. Barrförlusterna jämfördes med förändring av höjdtillväxt hos plantorna.

En metod för indirekt mätning av resistens testades också. Denna metod innebar att jämföra buffertkapaciteten i barr från oskadade plantor som växte på två andra försöksplatser med resistens hos angripna plantor inom samma helsyskongrupper på Tönnersjöheden.

Resultat

Av de sju variabler (jfr 3.2) som användes som mått på resistens, visade sig den tillväxtförändring angreppet förorsakade vara den mest användbara. Tillväxtförändringen var dock nära korrelerad med både uppskattad och beräknad barrförlust.

Syskonpopulationernas och provenienserna genomsnittliga resistens beräknades liksom föräldraträdens allmänna kombinationsförmåga. Högst resistens uppmättes på den halländska proveniensen Onsala. Proveniens

Boxholm i Östergötland visade sig ha lägst resistens medan de två övriga, Röskär i södra Uppland och Kårböle i norra Hälsingland intog en mellanställning. Skillnader mellan helsyskonpopulationer förekom också, speciellt då populationerna bildats efter korsningar mellan olika provenienser.

Barrens buffertkapacitet hos friska plantor visade sig hos de åtta undersökta helsyskonpopulationerna inte vara korrelerad med resistens hos angripna syskonplantor.

Diskussion

Sjukdomens effekt på tallplantorna är svår att bedöma vid en enda uppskattning av angreppsgraden. Plantornas förmåga att växa upp ur den mest utsatta nivån närmast marken är beroende dels av resistensen mot *Lophodermium* hos de utsatta delarna av plantan, dels av tillväxtförmågan före och under sjukdomens inverkan. Dessutom blir effekten av sjukdomen beroende av plantornas mer eller mindre goda anpassning till ståndortsmiljön eftersom den allmänna vitaliteten påverkas därav.

Eftersom svampen är en miljöfaktor som påverkar tillväxten, bör det vara logiskt att uttrycka effekten av svampangreppet genom att mäta den tillväxtförändring svampen givit upphov till. Tillväxten under de tre första vegetationsperioderna, då svampen ännu inte angripit, jämförs med tillväxten då plantan utsätts för tre års upprepade angrepp. För varje planta har kvoten mellan tillväxten under de två treårsperioderna beräknats. Logaritmen för denna kvot har använts som mått på resistensen.

Moderträden har genomgående visat sig ha större betydelse för avkommornas resistens än faderträden. Alla fyra provenienserna är förflyttade söderut till försökslokalen. Proveniens Onsala har dock utsatts för minst förändringar i klimatiskt avseende. Försöksresultaten tyder inte på att det finns något samband mellan resistens och ursprungsortens latitud eller maritimitet. Den brist på korrelation mellan resistens och buffertkapacitet som har konstaterats, skulle eventuellt kunna bero på alltför stora skillnader i resistens hos de undersökta familjerna.

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Appendix 1

The experimental material grouped into families and ranked according to change of growth (LNRHTV).

$LNRHTV = \ln((Height_2 - Height_1)/Height_1)$

$Height_1$ = Height before the attacks

$Height_2$ = Height after the attacks

$ASINRB = \text{arc sin } \sqrt{(BARRF)}$

BARRF = Share of lost needle biomass after the third attack

ANG1 = Degree of attack after the first infection

ANG2 = Degree of attack after the second infection

ANG3A = Degree of attack after the third infection

ANG3B = Degree of attack after the third infection (see page 13)

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
(65)	N6	N4	LNRHTV	0.49	0.27	18	1
			ASINRB	0.89	0.17	18	
			BARRF	0.59	0.14	18	
			ANG1	0		20	
			ANG2	1		20	
			ANG3A	0		18	
			ANG3B	1		18	
(74)	N12	N201	LNRHTV	0.40	0.31	18	2
			ASINRB	0.82	0.05	18	
			BARRF	0.53	0.05	18	
			ANG1	1		20	
			ANG2	1		20	
			ANG3A	0		18	
			ANG3B	1		18	
77	N12	X26	LNRHTV	0.40	0.24	24	3
			ASINRB	0.80	0.01	24	
			BARRF	0.51	0.01	24	
			ANG1	0		25	
			ANG2	0		25	
			ANG3A	0		24	
			ANG3B	1		24	
(73)	N12	N4	LNRHTV	0.33	0.27	18	4
			ASINRB	0.85	0.15	18	
			BARRF	0.56	0.12	18	
			ANG1	1		20	
			ANG2	1		20	
			ANG3A	0		18	
			ANG3B	1		18	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
61	N6	B101	LNRHTV	0.27	0.33	23	5
			ASINRB	0.91	0.14	23	
			BARRF	0.62	0.12	23	
			ANG1	1		25	
			ANG2	4		25	
			ANG3A	1		23	
			ANG3B	1		23	
(66)	N6	N202	LNRHTV	0.26	0.26	14	6
			ASINRB	0.93	0.15	14	
			BARRF	0.63	0.13	14	
			ANG1	1		15	
			ANG2	2		15	
			ANG3A	1		14	
			ANG3B	1		14	
132	N12	O.P.	LNRHTV	0.24	0.42	25	7
			ASINRB	0.86	0.15	25	
			BARRF	0.57	0.12	25	
			ANG1	1		25	
			ANG2	2		25	
			ANG3A	0		25	
			ANG3B	1		25	
(18)	B24	B102	LNRHTV	0.24	0.27	14	8
			ASINRB	0.83	0.13	14	
			BARRF	0.54	0.11	14	
			ANG1	1		15	
			ANG2	0		15	
			ANG3A	0		14	
			ANG3B	1		14	
(78)	N28	B102	LNRHTV	0.20	0.24	19	9
			ASINRB	0.86	0.12	19	
			BARRF	0.57	0.11	19	
			ANG1	1		20	
			ANG2	3		20	
			ANG3A	0		19	
			ANG3B	1		19	
(81)	N28	E4015	LNRHTV	0.18	0.41	15	10
			ASINRB	1.18	0.19	15	
			BARRF	0.83	0.13	15	
			ANG1	1		15	
			ANG2	7		15	
			ANG3A	6		15	
			ANG3B	2		15	
25	B24	X26	LNRHTV	0.14	0.25	23	11
			ASINRB	0.91	0.16	23	
			BARRF	0.62	0.14	23	
			ANG1	1		25	
			ANG2	2		24	
			ANG3A	1		23	
			ANG3B	1		23	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
(67)	N6	X18	LNRHTV	0.14	0.27	15	12
			ASINRB	0.97	0.16	15	
			BARRF	0.67	0.14	15	
			ANG1	1		15	
			ANG2	5		15	
			ANG3A	2		15	
			ANG3B	2		15	
75	N12	X18	LNRHTV	0.12	0.31	24	13
			ASINRB	0.85	0.08	24	
			BARRF	0.56	0.07	24	
			ANG1	1		25	
			ANG2	3		25	
			ANG3A	1		24	
			ANG3B	1		24	
22	B24	N201	LNRHTV	0.10	0.33	24	14
			ASINRB	0.91	0.14	24	
			BARRF	0.61	0.13	24	
			ANG1	1		25	
			ANG2	4		25	
			ANG3A	1		24	
			ANG3B	1		24	
86	N28	X52	LNRHTV	0.08	0.43	25	15
			ASINRB	1.01	0.20	25	
			BARRF	0.70	0.16	25	
			ANG1	1		25	
			ANG2	5		25	
			ANG3A	2		25	
			ANG3B	2		25	
133	N28	O.P.	LNRHTV	0.07	0.39	25	16
			ASINRB	0.93	0.19	25	
			BARRF	0.63	0.15	25	
			ANG1	1		25	
			ANG2	4		25	
			ANG3A	1		25	
			ANG3B	1		25	
126	B24	O.P.	LNRHTV	0.07	0.39	25	17
			ASINRB	0.98	0.18	25	
			BARRF	0.68	0.15	25	
			ANG1	1		25	
			ANG2	5		25	
			ANG3A	2		25	
			ANG3B	2		25	
(64)	N6	E4015	LNRHTV	0.05	0.55	19	18
			ASINRB	0.94	0.16	19	
			BARRF	0.64	0.15	19	
			ANG1	1		20	
			ANG2	4		20	
			ANG3A	1		19	
			ANG3B	1		19	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
83	N28	N202	LNRHTV	0.02	0.39	24	19
			ASINRB	0.99	0.17	24	
			BARRF	0.69	0.14	24	
			ANG1	1		25	
			ANG2	4		25	
			ANG3A	3		24	
			ANG3B	2		24	
131	N6	O.P.	LNRHTV	−0.04	0.30	23	20
			ASINRB	0.91	0.15	23	
			BARRF	0.62	0.14	23	
			ANG1	1		24	
			ANG2	4		24	
			ANG3A	1		23	
			ANG3B	1		23	
(19)	B24	B103	LNRHTV	−0.04	0.38	19	21
			ASINRB	1.05	0.18	25	
			BARRF	0.74	0.15	25	
			ANG1	1		20	
			ANG2	6		20	
			ANG3A	3		25	
			ANG3B	2		19	
85	N28	X26	LNRHTV	−0.06	0.43	23	22
			ASINRB	1.10	0.21	23	
			BARRF	0.77	0.17	23	
			ANG1	1		25	
			ANG2	7		24	
			ANG3A	5		23	
			ANG3B	2		23	
79	N28	B103	LNRHTV	−0.07	0.30	22	23
			ASINRB	1.10	0.20	22	
			BARRF	0.78	0.16	22	
			ANG1	1		25	
			ANG2	7		24	
			ANG3A	5		22	
			ANG3B	2		22	
36	E5024	B102	LNRHTV	−0.09	0.28	23	24
			ASINRB	1.08	0.20	23	
			BARRF	0.76	0.15	23	
			ANG1	2		25	
			ANG2	7		25	
			ANG3A	4		23	
			ANG3B	2		23	
62	N6	B103	LNRHTV	−0.10	0.46	24	25
			ASINRB	0.98	0.21	24	
			BARRF	0.67	0.17	24	
			ANG1	1		25	
			ANG2	3		25	
			ANG3A	1		24	
			ANG3B	1		24	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
21	B24	E4015	LNRHTV	−0.10	0.32	21	26
			ASINRB	0.97	0.17	21	
			BARRF	0.67	0.14	21	
			ANG1	1		25	
			ANG2	6		25	
			ANG3A	2		21	
			ANG3B	2		21	
10	B19	B102	LNRHTV	−0.11	0.47	24	27
			ASINRB	1.02	0.20	24	
			BARRF	0.71	0.16	24	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	4		24	
			ANG3B	2		24	
23	B24	N202	LNRHTV	−0.12	0.54	25	28
			ASINRB	1.02	0.25	25	
			BARRF	0.69	0.20	25	
			ANG1	1		25	
			ANG2	5		25	
			ANG3A	1		25	
			ANG3B	1		25	
5	B1	N4	LNRHTV	−0.12	0.52	25	29
			ASINRB	1.05	0.18	25	
			BARRF	0.73	0.14	25	
			ANG1	2		25	
			ANG2	6		25	
			ANG3A	4		25	
			ANG3B	2		25	
135	X10	O.P.	LNRHTV	−0.13	0.49	25	30
			ASINRB	1.13	0.21	25	
			BARRF	0.79	0.16	25	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	6		25	
			ANG3B	2		25	
108	X27	N201	LNRHTV	−0.14	0.40	24	31
			ASINRB	1.12	0.18	24	
			BARRF	0.79	0.14	24	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	5		24	
			ANG3B	2		24	
89	X9	E4008	LNRHTV	−0.15	0.37	24	32
			ASINRB	1.14	0.16	24	
			BARRF	0.81	0.13	24	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	6		24	
			ANG3B	2		24	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
20	B24	E4011	LNRHTV	−0.18	0.30	25	33
			ASINRB	0.98	0.17	25	
			BARRF	0.68	0.15	25	
			ANG1	1		25	
			ANG2	5		25	
			ANG3A	2		25	
			ANG3B	2		25	
(15)	B19	X18	LNRHTV	−0.20	0.51	19	34
			ASINRB	1.15	0.18	19	
			BARRF	0.82	0.13	19	
			ANG1	2		20	
			ANG2	7		20	
			ANG3A	6		19	
			ANG3B	2		19	
26	B24	X52	LNRHTV	−0.20	0.45	25	35
			ASINRB	1.05	0.19	25	
			BARRF	0.74	0.16	25	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	5		25	
			ANG3B	2		25	
91	X9	N4	LNRHTV	−0.20	0.44	23	36
			ASINRB	1.09	0.20	23	
			BARRF	0.76	0.16	23	
			ANG1	1		25	
			ANG2	6		25	
			ANG3A	4		23	
			ANG3B	2		23	
103	X10	X26	LNRHTV	−0.21	0.23	23	37
			ASINRB	1.19	0.14	23	
			BARRF	0.85	0.10	23	
			ANG1	1		25	
			ANG2	8		24	
			ANG3A	6		23	
			ANG3B	2		23	
101	X10	X18	LNRHTV	−0.22	0.45	25	38
			ASINRB	1.16	0.15	25	
			BARRF	0.83	0.10	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	5		25	
			ANG3B	2		25	
40	E5024	N201	LNRHTV	−0.22	0.36	25	39
			ASINRB	1.25	0.12	25	
			BARRF	0.89	0.08	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		25	
			ANG3B	2		25	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
39	E5024	N4	LNRHTV	−0.23	0.35	24	40
			ASINRB	1.03	0.17	24	
			BARRF	0.72	0.14	24	
			ANG1	1		25	
			ANG2	5		25	
			ANG3A	3		24	
			ANG3B	2		24	
8	B1	X52	LNRHTV	−0.23	0.39	22	41
			ASINRB	1.14	0.21	22	
			BARRF	0.80	0.16	22	
			ANG1	2		25	
			ANG2	8		24	
			ANG3A	6		22	
			ANG3B	2		22	
13	B19	N4	LNRHTV	−0.23	0.39	23	42
			ASINRB	1.08	0.20	23	
			BARRF	0.76	0.16	23	
			ANG1	2		25	
			ANG2	6		25	
			ANG3A	5		23	
			ANG3B	2		23	
109	X27	N202	LNRHTV	−0.23	0.40	23	43
			ASINRB	1.09	0.19	23	
			BARRF	0.77	0.15	23	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	5		23	
			ANG3B	2		23	
3	B1	E4008	LNRHTV	−0.24	0.33	23	44
			ASINRB	1.02	0.17	23	
			BARRF	0.72	0.14	23	
			ANG1	1		24	
			ANG2	7		24	
			ANG3A	4		23	
			ANG3B	2		23	
46	E5053	E4008	LNRHTV	−0.24	0.27	22	45
			ASINRB	1.14	0.11	22	
			BARRF	0.82	0.08	22	
			ANG1	1		25	
			ANG2	7		23	
			ANG3A	5		22	
			ANG3B	2		22	
104	X27	B102	LNRHTV	−0.24	0.29	24	46
			ASINRB	1.18	0.19	24	
			BARRF	0.83	0.14	24	
			ANG1	2		25	
			ANG2	8		24	
			ANG3A	7		24	
			ANG3B	2		24	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
(45)	E5053	B103	LNRHTV	−0.24	0.51	13	47
			ASINRB	1.25	0.18	13	
			BARRF	0.88	0.13	13	
			ANG1	2		15	
			ANG2	8		15	
			ANG3A	8		13	
			ANG3B	3		13	
11	B19	E4008	LNRHTV	−0.24	0.32	24	48
			ASINRB	1.13	0.17	24	
			BARRF	0.80	0.13	24	
			ANG1	1		25	
			ANG2	7		25	
			ANG3A	5		24	
			ANG3B	2		24	
(14)	B19	N201	LNRHTV	−0.24	0.37	18	49
			ASINRB	1.22	0.16	18	
			BARRF	0.87	0.12	18	
			ANG1	2		20	
			ANG2	8		20	
			ANG3A	6		18	
			ANG3B	2		18	
87	X9	B101	LNRHTV	−0.27	0.35	25	50
			ASINRB	1.22	0.18	25	
			BARRF	0.86	0.13	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		25	
			ANG3B	2		25	
48	E5053	N4	LNRHTV	−0.27	0.41	25	51
			ASINRB	1.16	0.18	25	
			BARRF	0.82	0.13	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	6		25	
			ANG3B	2		25	
(107)	X27	E4015	LNRHTV	−0.27	0.28	19	52
			ASINRB	1.15	0.16	19	
			BARRF	0.81	0.12	19	
			ANG1	1		20	
			ANG2	7		20	
			ANG3A	6		19	
			ANG3B	2		19	
(94)	X9	X52	LNRHTV	−0.29	0.36	19	53
			ASINRB	1.26	0.10	19	
			BARRF	0.90	0.06	19	
			ANG1	2		20	
			ANG2	8		20	
			ANG3A	7		19	
			ANG3B	3		19	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
47	E5053	E4015	LNRHTV	−0.32	0.31	24	54
			ASINRB	1.21	0.17	24	
			BARRF	0.86	0.12	24	
			ANG1	2		25	
			ANG2	8		24	
			ANG3A	7		24	
			ANG3B	2		24	
57	E5055	N202	LNRHTV	−0.34	0.42	24	55
			ASINRB	1.22	0.12	24	
			BARRF	0.87	0.08	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
35	E5024	B101	LNRHTV	−0.34	0.33	23	56
			ASINRB	1.13	0.18	23	
			BARRF	0.80	0.14	23	
			ANG1	2		25	
			ANG2	7		24	
			ANG3A	5		23	
			ANG3B	2		23	
6	B1	N202	LNRHTV	−0.36	0.45	25	57
			ASINRB	1.21	0.14	25	
			BARRF	0.86	0.09	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		25	
			ANG3B	2		25	
7	B1	X18	LNRHTV	−0.36	0.40	24	58
			ASINRB	1.22	0.18	24	
			BARRF	0.86	0.13	24	
			ANG1	2		25	
			ANG2	8		24	
			ANG3A	7		24	
			ANG3B	2		24	
4	B1	E4015	LNRHTV	−0.36	0.36	25	59
			ASINRB	1.19	0.14	25	
			BARRF	0.85	0.10	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		25	
			ANG3B	2		25	
136	X27	O.P.	LNRHTV	−0.37	0.37	22	60
			ASINRB	1.24	0.14	22	
			BARRF	0.88	0.10	22	
			ANG1	1		23	
			ANG2	8		23	
			ANG3A	7		22	
			ANG3B	2		22	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
(1)	B1	B101	LNRHTV	−0.37	0.28	18	61
			ASINRB	1.21	0.17	18	
			BARRF	0.86	0.12	18	
			ANG1	2		18	
			ANG2	8		18	
			ANG3A	7		18	
			ANG3B	2		18	
90	X9	E4015	LNRHTV	−0.37	0.34	24	62
			ASINRB	1.27	0.15	24	
			BARRF	0.90	0.11	24	
			ANG1	1		25	
			ANG2	8		25	
			ANG3A	8		24	
			ANG3B	3		24	
124	B1	O.P.	LNRHTV	−0.39	0.38	24	63
			ASINRB	1.21	0.18	24	
			BARRF	0.85	0.13	24	
			ANG1	3		25	
			ANG2	8		24	
			ANG3A	7		24	
			ANG3B	2		24	
125	B19	O.P.	LNRHTV	−0.40	0.43	23	64
			ASINRB	1.18	0.14	23	
			BARRF	0.84	0.09	23	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	6		23	
			ANG3B	2		23	
9	B19	B101	LNRHTV	−0.41	0.35	22	65
			ASINRB	1.19	0.16	22	
			BARRF	0.85	0.11	22	
			ANG1	2		25	
			ANG2	7		25	
			ANG3A	6		22	
			ANG3B	2		22	
37	E5024	E4008	LNRHTV	−0.42	0.37	24	65
			ASINRB	1.14	0.15	24	
			BARRF	0.81	0.11	24	
			ANG1	2		25	
			ANG2	7		25	
			ANG3A	6		24	
			ANG3B	2		24	
106	X27	E4011	LNRHTV	−0.42	0.43	23	67
			ASINRB	1.30	0.14	23	
			BARRF	0.92	0.10	23	
			ANG1	2		24	
			ANG2	8		24	
			ANG3A	8		23	
			ANG3B	3		23	

Family no.	Mother	Father		Mean or median	Sd	No. of ind.	Rank
98	X10	E4011	LNRHTV	−0.42	0.36	24	68
			ASINRB	1.20	0.15	24	
			BARRF	0.86	0.11	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
128	E5024	O.P.	LNRHTV	−0.42	0.40	24	69
			ASINRB	1.23	0.14	24	
			BARRF	0.88	0.09	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
(38)	E5024	E4011	LNRHTV	−0.42	0.32	20	70
			ASINRB	1.32	0.10	20	
			BARRF	0.93	0.06	20	
			ANG1	3		20	
			ANG2	8		20	
			ANG3A	8		20	
			ANG3B	3		20	
110	X27	X26	LNRHTV	−0.43	0.50	24	71
			ASINRB	1.27	0.14	24	
			BARRF	0.90	0.09	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	8		24	
			ANG3B	3		24	
58	E5055	X26	LNRHTV	−0.46	0.40	23	72
			ASINRB	1.37	0.02	23	
			BARRF	0.96	0.01	23	
			ANG1	4		25	
			ANG2	8		23	
			ANG3A	8		23	
			ANG3B	3		23	
(93)	X9	X18	LNRHTV	−0.49	0.28	19	73
			ASINRB	1.32	0.08	19	
			BARRF	0.94	0.05	19	
			ANG1	2		20	
			ANG2	8		20	
			ANG3A	8		19	
			ANG3B	3		19	
88	X9	B103	LNRHTV	−0.49	0.30	24	74
			ASINRB	1.26	0.13	24	
			BARRF	0.89	0.09	24	
			ANG1	1		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	

Family no.	Mother	Father		Mean or median	Sd	No of ind.	Rank
49	E5053	N202	LNRHTV	−0.52	0.55	24	75
			ASINRB	1.26	0.15	24	
			BARRF	0.89	0.10	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
134	X9	O.P.	LNRHTV	−0.52	0.33	24	76
			ASINRB	1.23	0.14	24	
			BARRF	0.87	0.10	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
129	E5053	O.P.	LNRHTV	−0.54	0.35	24	77
			ASINRB	1.33	0.08	24	
			BARRF	0.94	0.05	24	
			ANG1	3		25	
			ANG2	8		25	
			ANG3A	8		24	
			ANG3B	3		24	
42	E5024	X26	LNRHTV	−0.55	0.42	25	78
			ASINRB	1.32	0.09	25	
			BARRF	0.93	0.06	25	
			ANG1	3		25	
			ANG2	8		25	
			ANG3A	8		25	
			ANG3B	3		25	
41	E5024	X18	LNRHTV	−0.56	0.44	24	79
			ASINRB	1.23	0.17	24	
			BARRF	0.87	0.12	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
2	B1	B103	LNRHTV	−0.58	0.43	22	80
			ASINRB	1.25	0.18	22	
			BARRF	0.88	0.14	22	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	8		22	
			ANG3B	3		22	
130	E5055	O.P.	LNRHTV	−0.61	0.39	25	81
			ASINRB	1.33	0.10	25	
			BARRF	0.94	0.06	25	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	8		25	
			ANG3B	3		25	

Family no.	Mother	Father		Mean or median	Sd	No of ind.	Rank
53	E5055	B103	LNRHTV	−0.64	0.43	24	82
			ASINRB	1.35	0.07	24	
			BARRF	0.95	0.04	24	
			ANG1	3		25	
			ANG2	8		25	
			ANG3A	8		24	
			ANG3B	3		24	
55	E5055		LNRHTV	−0.65	0.30	25	83
			ASINRB	1.32	0.09	25	
			BARRF	0.93	0.05	25	
			ANG1	3		25	
			ANG2	8		25	
			ANG3A	8		25	
			ANG3B	3		25	
51	E5053	X52	LNRHTV	−0.68	0.47	24	84
			ASINRB	1.32	0.08	24	
			BARRF	0.93	0.05	24	
			ANG1	2		25	
			ANG2	8		24	
			ANG3A	8		24	
			ANG3B	3		24	
12	B19	E4011	LNRHTV	−0.71	0.45	24	85
			ASINRB	1.29	0.10	24	
			BARRF	0.91	0.06	24	
			ANG1	2		25	
			ANG2	8		25	
			ANG3A	7		24	
			ANG3B	2		24	
56	E5055	N201	LNRHTV	−0.73	0.47	24	86
			ASINRB	1.36	0.04	24	
			BARRF	0.95	0.02	24	
			ANG1	2		24	
			ANG2	8		24	
			ANG3A	8		24	
			ANG3B	3		24	

Appendix 2

Account of the calculation technique used to estimate general combining ability (Figure 9) and variances (Table 5) (mainly according to Matern, 1976)

In the experiment there are $n = 56$ families completely represented in $r = 5$ replications. These families are progenies of $g_1 = 12$ mother trees and $g_2 = 12$ father trees. Assume that the progeny of the crossing between the *i*:th mother and the *j*:th father is one of the 56 families in the test. Let y_{ijk} be the mean per seedling in a plot in the *k*:th block where this family is. Assume that this value is generated by a mechanism to be described by the formula

$$y_{ijk} = \mu + \varrho_k + \beta_i + \gamma_j + s_{ij} + \varepsilon_{ijk}$$

where

- μ expresses the general level of the y-values in the test
- ϱ_k indicates the effect of replication (in block No. *k*)
- β_i and γ_j respectively are expressions of general combining ability of mother No. *i* and father No. *j*
- s_{ij} is the specific combining ability and ε_{ijk} the error.

It is also assumed that ϱ, β, γ, s and ε are independent random variables, varying around 0 with the variances σ_ϱ^2, σ_β^2, σ_γ^2, σ_s^2 and σ_ε^2, respectively. In order to be able to estimate "components of variance" (Table 5), various sums of squares are calculated. The sums of squares of error with $(n-1)(r-1)$ degrees of freedom, of replication with $(r-1)$ degrees of freedom, and of families with $(n-1)$ degrees of freedom are calculated as in an experiment designed as randomized blocks with n treatments (Snedecor and Cochran, 1967).

In order to estimate σ_β^2, σ_γ^2 and σ_s^2, it is necessary to divide the sum of squares of families in different ways. A sum of squares is calculated by adjusting to the family means—by the method of least squares—an expression of the type $m + b_i + c_j$. Then are also obtained estimations (b_i) and (c_j) of the parameters (β_i) and (γ_j). These estimations are indefinite in the sense that the same adjustment is obtained by adding an additive constant to each b_i and an (other) additive constant to each c_j. The minimized sum of squares which has $n - g_1 - g_2 + 1$ degrees of freedom is found in the line "Mother + Father" in Table 5.

A sum of squares between mothers (*ss* (mothers)) and a sum of squares between fathers (*ss* (fathers)), with $g_1 - 1$ and $g_2 - 1$ degrees of freedom, respectively, are also calculated. By substracting (*ss* (mothers)) from (*ss* (families)) a sum of squares with $n - g_1$ degrees of freedom is obtained. Its expected value is a linear expression in σ_γ^2, σ_s^2 and σ_ε^2. The corresponding mean square is entered in the row "Fathers + (Mothers × Fathers)" in Table 5. The mean square "Mothers + (Mothers × Fathers)" was calculated in a similar manner.

The expected values of the mean squares are entered in Table 5. Estimates of the variances, σ_β^2, σ_γ^2, σ_s^2, etc., are obtained by equalizing the expected values and their mean squares. Those estimations are entered in Table 5.

The 24 values, b_1, b_2, b_3 ... b_{12} and c_1, c_2, c_3 ... c_{12}, are estimates of the general combining ability (GCA) of mother and father trees, respectively, and are shown in Figure 9. Because of the calculating technique the level of the values has been adjusted so that one b_i and one c_j are equal to 0, namely X10 and E4011. The variances and covariances of the estimates of (GCA) can be used to estimate the standard error

in each contrast between b_i values and each contrast between c_j values. The variances and covariances for each of the three variables have been filed by the author and are available to anyone who may be interested in them.

Appendix 3 a

Buffer capacity I of pine needles in a varying number of individuals of eight full-sib families.

Family 55

Långmor					Fagerdal		
July 1975	Aug. 1976	Sept. 1976	Sept. 1977	Oct. 1975	July 1975	Sept. 1976	Oct. 1975
87.6	64.9	48.8	43.5	10.0	65.4	23.9	4.0
85.6	70.6	33.5	37.9	7.2	56.0	40.0	4.2
67.2	73.3	41.1	44.5	6.7	53.4	21.7	6.7
	81.0	32.0	36.0	6.0		28.2	1.7
	77.9	41.4	44.2	5.4		42.6	
	85.1	43.9	43.6			30.6	
	79.8	34.0	33.8			32.4	
	77.6	35.3	33.7			40.8	
	80.1	31.4				29.6	
	87.7	41.0				29.9	
	60.3	51.2				31.8	
	70.5	51.6				26.4	
	69.0	51.8				37.0	
	62.2	59.3				30.4	
	59.5	45.2				41.0	
	64.0	43.3				27.2	
	69.4	42.7				41.2	
	76.3	52.9				31.3	
	67.4	52.9				35.9	
		47.2				54.9	
						35.7	
						46.0	
						29.3	
						33.5	
						43.6	
Mean: 80.00	72.45	44.02	39.65	7.10	58.30	34.60	4.20

Family 56

	Långmor					Fagerdal		
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	Oct. 1975	July 1975	Sept. 1976	Oct. 1975
	91.2	74.5	43.5	49.1	4.6	69.3	37.6	10.5
	97.5	71.5	34.9	33.8	12.5	71.7	36.9	13.9
	93.4	76.3	36.7	33.3	4.3	70.8	53.4	3.1
		100.5	50.6	51.0	6.1		27.5	13.2
		90.6	50.5	50.2			35.1	
		70.4	46.7	49.9			46.5	
		93.7	56.0	40.6			50.0	
		109.2	52.7	35.7			40.7	
		102.0	57.4				34.9	
		111.8	40.1				45.1	
		98.4	43.6				62.4	
		87.2	36.4				32.7	
		94.2	34.3				48.5	
		92.4	55.3				36.4	
		81.1	57.7					
		72.0	62.6					
		83.5	54.2					
Mean:	94.03	88.78	47.84	42.95	6.88	70.6	41.98	10.15

Family 77

	Långmor					Fagerdal		
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	Oct. 1975	July 1975	Sept. 1976	Oct. 1975
	78.3	83.6	32.3	25.6	4.8	57.7	38.6	1.5
	78.8	68.9	24.1	14.2	6.8	72.0	34.1	7.4
	79.9	81.2	32.0	18.3	4.7	73.7	38.0	8.0
		76.0	33.6	25.0	5.9		28.9	2.6
		75.0	29.8	27.0			46.2	4.0
		73.9	30.4	18.7			15.3	
		59.6	21.7	25.8			24.2	
		55.9	31.4				31.2	
		67.7	36.4				39.6	
		60.3	38.9				23.0	
		55.3	28.7				21.4	
		94.8	39.6				46.5	
		77.4	38.7				41.4	
		74.8	40.1				31.1	
		86.2	31.2				21.8	
		75.2	33.0				37.5	
		62.9	39.7				29.6	
		61.8	34.9				24.2	
			35.1				30.6	
							33.5	
							35.2	
							35.7	
							30.2	
							47.3	
Mean:	79.00	71.69	33.24	22.08	5.55	67.80	32.71	4.70

Family 53

	Långmor		Fagerdal	
	July 1975	Oct. 1975	July 1975	Oct. 1975
	63.9	6.3	73.7	10.8
	58.5	4.2	77.4	4.5
	76.1	7.0	86.4	3.8
		8.7		
Mean:	66.17	6.55	79.17	6.37

Family 58

	Långmor		Fagerdal	
	July 1975	Oct. 1975	July 1975	Oct. 1975
	85.6	5.8	70.4	3.6
	84.6	2.4	73.6	8.8
	63.6	5.2	73.6	2.8
				3.9
				4.7
Mean:	77.93	4.47	72.53	4.76

Family 75

	Långmor					Fagerdal		
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	Oct. 1975	July 1975	Sept. 1976	Oct. 1975
	85.3	71.4	45.9	30.6	4.5	57.6	45.6	7.0
	71.7	76.1	29.7	26.1	8.4	63.2	33.3	6.4
	78.3	76.0	42.2	30.8	6.1	74.4	29.1	5.9
		69.8	43.5	27.4			24.7	
		77.4	36.2	20.2			27.7	
		91.5	32.6	24.0			30.3	
		79.1	44.1	25.7			26.8	
		71.4	34.1				45.7	
		73.0	38.8				31.6	
		81.1	29.0				41.2	
		63.3	35.3				40.2	
		76.6	45.0				38.7	
		77.1	31.7				44.8	
		75.2	29.0				33.5	
Mean:	78.43	75.64	36.94	26.39	6.33	65.07	35.23	6.43

Family 73

	Fagerdal	
	July 1975	Oct. 1975
	58.1	6.2
	60.3	8.3
	46.2	4.5
		5.4
Mean:	54.86	6.10

Family 74

	Fagerdal	
	July 1975	Oct. 1975
	56.4	7.1
	62.1	6.5
	60.6	6.0
Mean:	59.70	6.53

Control measurements of six individuals

Used value	Control value	Deviation in %
69.3	69.9	– 0.87
71.7	71.2	0.56
70.8	73.1	– 3.24
85.6	89.2	– 4.21
84.6	86.7	– 2.48
63.6	63.8	– 0.31

for 20 values: $s_{\bar{x}} = \sqrt{\frac{3 \cdot 40}{20}} = 0.41\ \%$

Appendix 3b

Buffer capacity II between pH 4.0 and pH 5.0 in a varying number of individuals of eight full-sib families.

Family 55

	Långmor				Fagerdal	
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	July 1975	Sept. 1976
	7.2	4.5	4.2	3.9	6.1	3.7
	7.0	3.4	3.5	3.8	5.9	4.6
	6.1	3.5	3.8	4.0	6.2	4.3
		4.2	3.9	3.6		3.5
		4.0	4.3	3.8		4.4
		4.4	4.3	3.6		3.9
		3.3	3.8	3.5		3.8
		3.4	3.9	4.0		4.5
		3.6	3.6			4.1
		3.7	4.1			3.7
		3.4	3.7			4.1
		3.9	4.1			4.6
		3.9	3.7			3.7
		3.8	4.0			4.7
		3.0	4.0			4.7
		3.0	4.5			4.8
		3.4	3.9			4.8
		3.7	4.4			4.6
		3.4	4.3			4.3
			4.4			4.1
						4.4
						4.0
						3.9
						4.0
						4.3
Means:	6.77	3.66	4.02	3.78	6.07	4.22

Family 56

	Långmor				Fagerdal	
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	July 1975	Sept. 1976
	6.4	3.5	3.8	3.8	6.6	4.4
	6.5	3.8	3.8	4.0	6.4	3.9
	6.6	3.6	3.6	4.3	6.2	4.3
		4.0	3.9	4.1		4.8
		3.6	3.7	4.2		4.6
		3.9	4.2	4.1		5.0
		4.1	3.6	4.0		5.2
		3.4	3.7	4.3		3.6
		3.3	3.8			3.7
		3.7	4.1			4.2
		3.4	3.7			4.0
		3.1	4.3			5.1
		3.9	4.0			4.0
		3.9	4.0			4.3
		3.7	3.8			
		3.3	4.1			
		3.7	4.1			
Means:	6.50	3.64	3.89	4.10	6.40	4.36

Family 53

	Långmor	Fagerdal
	July 1975	July 1975
	6.1	6.7
	6.1	6.4
	6.1	6.0
Means:	6.10	6.37

Family 58

	Långmor	Fagerdal
	July 1975	July 1975
	6.7	6.3
	6.5	6.7
	6.5	6.9
Means:	6.57	6.63

Family 75

	Långmor				Fagerdal	
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	July 1975	Sept. 1976
	6.4	3.7	3.6	3.9	6.0	4.0
	6.2	3.5	4.4	3.8	5.7	3.9
	6.3	3.6	3.6	3.5	6.1	3.7
		3.4	3.9	3.7		3.7
		3.7	4.2	3.6		4.2
		3.8	3.9	3.8		4.2
		3.0	3.9	3.9		4.7
		3.1	4.2			3.9
		3.3	3.4			5.3
		3.2	3.3			4.0
		3.5	3.4			4.2
		3.6	3.6			4.7
		3.6	3.4			3.5
		3.4	4.1			4.0
Means:	6.30	3.46	3.78	3.74	5.93	4.14

Family 77

	Långmor				Fagerdal	
	July 1975	Aug. 1976	Sept. 1976	Sept. 1977	July 1975	Sept. 1976
	6.3	3.5	4.1	3.3	6.7	5.1
	6.4	3.7	4.4	3.0	7.2	5.4
	6.0	3.3	4.1	3.0	5.8	5.7
		3.3	3.7	3.7		5.7
		3.2	3.8	3.4		5.5
		3.3	4.0	2.9		5.4
		3.1	4.0	3.6		4.7
		3.2	4.1			5.3
		3.3	3.6			5.3
		3.4	4.0			4.4
		3.3	4.0			4.6
		3.0	3.2			4.8
		3.2	4.1			4.9
		3.2	3.8			5.4
		3.5	3.9			3.4
		3.6	3.9			3.7
		3.6	4.3			4.6
		3.7	4.6			5.6
			4.3			4.8
						6.0
						5.2
						5.0
						4.4
						4.3
Means:	6.23	3.36	3.99	3.27	6.57	4.97

Family 73

	Fagerdal
	July 1975
	5.7
	6.3
	2.8
Mean:	4.93

Family 74

	Fagerdal
	July 1975
	7.2
	6.3
	6.0
Mean:	6.50