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LOPHODERMELLA SULCIGENA ON SCOTS PINE IN FINLAND

RISTO JALKANEN

SELOSTE

MÄNNYNHARMAAKARISTE SUOMESSA

HELSINKI 1986

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Cover (front & back): Scots pine (*Pinus sylvestris* L.) is the most important tree species in Finland. Pine dominated forest covers about 60 per cent of forest land and its total volume is nearly 700 mil. cu.m. The front cover shows a young Scots pine and the back cover a 30-metre-high, 140-year-old tree.

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RISTO JALKANEN

LOPHODERMELLA SULCIGENA ON SCOTS PINE IN FINLAND

To be presented, with the permission of the Faculty of Agriculture and Forestry of the University of Helsinki, for public criticism in Auditorium XII, Aleksanterinkatu 5, November 14, 1986, at 12 o'clock noon.

SELOSTE

MÄNNYNHARMAAKARISTE SUOMESSA

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The effects of the *Lophodermella* needle cast on Scots pine (*Pinus sylvestris* L.) and factors affecting susceptibility as well the infection and life cycle of *Lophodermella sulcigena* (Rostrup) Höhnel were studied during the years 1978—1985 in southern, central and northern Finland. A total of 210 healthy or slightly, moderately or heavily infected trees were felled for growth measurements.

The first ascocarps of *L. sulcigena* matured mainly in June, nearly to the day at the beginning of the needle flush. The pathogen sporulated in June—July 1—3 hours after the start of rain or on nights when a relative humidity of at least 90 % was reached. The needles were infected normally during the first half of the needle elongation, in June in southern Finland, in June—July in central Finland and in July in northern Finland. The initial symptoms appeared a month after the infection; after three months needle shedding started. The majority of needles shed in two peaks, in June and in August of the year following the infection, leading to premature needle death 2—3 years before normal shedding of the oldest needles.

Within a stand the epidemic started in the dense and lowest parts of the plantation, wherefrom the disease spread in a few years to the upper parts; the lowest parts of the stand began to recover at the same time. The epidemic lasted 5 years in the stands. Suppressed trees were not as susceptible as trees above medium size. However, the greater the distance (up to 30 metres) from the edge of the field, the more heavily the trees were infected by *L. sulcigena*. The infections occurring during the second half of the needle elongation did not affect needle length significantly.

The bigger the annual increment had been before the epidemic and the more heavily the trees were diseased, the more the disease retarded the growth of Scots pine. The growth of heavily infected trees dropped as early as in the second year of the epidemic, whereas the disease had no effect on the growth of slightly infected trees. Height, radial and volume growth of heavily infected trees all fell significantly below the level of healthy trees. The height, radial and volume growth losses of heavily infected trees were 14.5 %, 26.0 % and 28.8 % respectively during the epidemic, and 5.2 %, 6.6 % and 16.4 % in 20 years.

Tämän työn tarkoituksena oli tutkia harmaakaristeen aiheuttajasienen (*Lophodermella sulcigena* (Rostrup) Höhnel) elämänkiertoa ja harmaakariste-epidemian vaikutuksia männyn neulasiin ja puuston kehitykseen. Tutkimukset tehtiin Etelä-, Keski- ja Pohjois-Suomessa vuosina 1978—1985.

Kasvutappioiden määrittämiseksi Halkivahan metsiköstä, jonka kaikkien puiden harmaakaristeisuus määritettiin vuosittain epidemian aikana, hakattiin 210 kasvukoepuuta (5.0 % metsikön puumäärästä) syyskesällä 1982. Koepuut jakaantuivat neljään eri tavoin sairastaneiden puiden ryhmään, joissa kussakin oli noin 50 mäntyä: terveet puut, lievästi sairaat (uusimmasta neulaskerrasta tuhoutunut v. 1978—80 alle 34 %), keskinkertaisesti sairaat (34—66 %) ja ankarasti sairaat (yli 66 %) puut.

Ensimmäiset taudinaiheuttajan itiöemät kypsyivät pääosin kesäkuussa lähes päivälleen neulasten pituuskasvun alkaessa. *L. sulcigena* itiöi kesä—heinäkuussa 1—3 tunnin kuluttua sateen alkamisesta tai aamuyöllä kosteuden noustua vähintäin 90 %:iin. Uudet neulaset saastuivat pituuskasvunsa ensimmäisellä puoliskolla keskimäärin kesäkuussa Etelä-Suomessa, kesä—heinäkuun vaihteessa Keski-Suomessa ja heinäkuussa Pohjois-Suomessa. Taudinoireet ilmestyivät noin kuukauden kuluttua infektiosta, ja vajaan kolmen kuukauden kuluttua ensimmäiset kääpiöversot karisivat. Harmaakaristeiset neulaset karisivat kuitenkin pääosin kesä- ja elokuussa vuoden kuluttua infektiosta. Neulasten pituuskehityksen jälkimmäisellä puoliskolla tapahtunut harmaakaristeinfektio ei vaikuttanut neulasten pituuksiin merkittävästi.

Harmaakariste-epidemia puhkesi metsikön tiheissä alaosissa, mistä tauti laajeni ylempiin osiin samalla, kun alimpien osien aiemmin ankarasti sairaat puut alkoivat tervehtyä. Metsikössä epidemia kesti 5 vuotta. Alistetut puut eivät olleet yhtä sairaita kuin keskimääräistä kookkaammat puut. Toisaalta harmaakaristeisuus lisääntyi pellon laidasta metsikön sisälle päin aina jopa 30 metriin asti.

Harmaakariste hidasti puiden kasvua sitä enemmän, mitä ankarammin puut olivat sairastuneet. Ankarimmin sairastuivat ennen epidemiaa parhaiten kasvaneet männyt. Sädekasvu taantui selvästi enemmän ja nopeammin kuin pituuskasvu, molemmat kuitenkin tilastollisesti merkitsevästi. Ankarasti sairaat puut toipuivat 2 vuodessa terveiden puiden kasvun tasolle. Lievästi sairaiden puiden kasvu ei taantunut merkitsevästi.

5-vuotisen epidemian seurauksena ankarasti sairaat puut menettivät pituuskasvustaan 14.5 %, sädekasvustaan 26.0 % ja tilavuuskasvustaan 28.8 %. Koko metsikön 20-vuotisen elinajan aikaisesta kasvusta ne menettivät vastaavasti 5.2 %, 6.6 % ja 16.4 %.

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PREFACE

The present investigations were conducted at the Department of Forest Protection, Section Forest Pathology, the Finnish Forest Research Institute.

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Last but not least I wish to express my sincere and warm thanks to my wife, Marja, and our daughters, Aino and Emma, to whom I dedicate my thesis.

Rovaniemi, April 1986

Risto Jalkanen

1. INTRODUCTION

Lophodermella sulcigena (Rostrup) Höhnel causes the premature death of current-year needles of two-needle pines (Millar 1984). The pathogen is widespread in the boreal forest zone and south of it in Eurasia (Rostrup 1883, Lagerberg 1910, Vanin 1925, Darker 1932, Kalandra 1938, Terrier 1944, Moriondo 1963, Hanso 1970, Millar 1970, Lazarev 1983), but does not occur in North America, where related fungi exist (Millar 1984). In Finland only *Pinus sylvestris* L. has been susceptible to *L. sulcigena* (Jalkanen 1985).

L. sulcigena has a one-year life cycle, during which the fungus, at first infecting growing semi-mature needles only, makes its ascocarps on both sides of the needles (Campbell and Syrop 1975). The hysterothecia have a stroma-like structure (Campbell and Syrop 1975), and the ascospores are released singly from the asci (Campbell 1973, Minter and Cannon 1984) when the relative humidity rises above 90 % (Millar 1970). As early as one month after infection, the first disease symptoms appear on needles (Williamson et al. 1976), whose main autumnal colour is reddish brown (Terrier 1944). The diseased needles shed down mainly in the summer following the infection (Watson 1971). Also the mycofloral successions in needles infected by *L. sulcigena* have been examined (Mitchell and Millar 1978). Although the pathogen can infest trees even 15—18 m high (Lagerberg 1910), it nevertheless is a disease of young even-aged dense stands (Jørstad 1925), especially on fertile soils (Krutov 1979, Jalkanen 1985). When a tree is infected by *L. sulcigena* in successive years, its growth is retarded. However, because the *Lophodermella* needle cast is not known to have killed trees, it should be easy to follow an epidemic and to determine its effects on growth. Only one work on growth effects has been published (Mitchell et al. 1976a) despite the fact that shoots shortened by *L. sulcigena* were demonstrated as early as 1910 by Lagerberg. In the course of the *Lophodermella* needle cast epidemic a secondary coloniser of the needles infected primarily by *L. sulcigena* is found (Münch and von Tubeuf 1910). It is believed that this fungus, *Hendersonia acicola* Münch & Tubeuf, biologically prevents the pathogen from fruiting (Darker 1967, Mitchell et al. 1976b).

As can be concluded from the short introduction above on the disease, the biology of *L. sulcigena* is fairly well known. However, due to differences among various areas results are often inapplicable for use in other parts of the continent. In addition, a disease like *Lophodermella* needle cast, which occurs epidemically only irregularly makes it difficult to produce new research knowledge relevant for a local area. Since the beginning of the latest *Lophodermella* needle cast epidemic in Finland a review of the literature on the disease has been published for the forest professional. The review (Jalkanen 1981) is based mainly on foreign research.

2. STUDY OUTLINE

The *Lophodermella* needle cast is one of the most important needle diseases on Scots pine in Finland. Yet knowledge on the disease was very limited when the nearly ten-year long epidemic broke out in the mid 1970's. The disease was exceptionally conspicuous particularly soon after the first disease symptoms. A need arose to review the knowledge on the *Lophodermella* needle cast (Jalkanen 1981). In addition, the lack of Finnish research knowledge led to a research program to produce information applicable especially in Finland. This program exploited the *Lophodermella* needle cast epidemic which started in 1976 and apparently ceased in the summer of 1985 (Jalkanen 1986).

The main aims of the present study are the following:

1. To study the life cycle of the pathogen and its influence on the needles of Scots pine,
2. to study the dynamics of the *Lophodermella* epidemic at the stand level, and
3. to assess the effects of the disease on the growth of Scots pine.

3. MATERIALS AND METHODS

31. Experimental stands

The experiments were carried out in six Scots pine stands. Halkivaha and Kaksoiskivi in Loppi represented southern Finland, Kekälinen in Multia central Finland, and Sieväkari, Kaihuavaara and Juotas in Rovaniemi rural commune, hereinafter referred to as Rovaniemi northern Finland (Fig. 1). The experimental stands were 15—40 years old and naturally infected by *L. sulcigena*. These, according to Jalkanen (1985), could be considered as typical stands with heavy occurrence of the *Lophodermella* needle cast for the years 1976—1984 on a fresh or better forest site.

Loppi. The stands of Halkivaha (Grid 27°E 672:35) and Kaksoiskivi (673:36) were planted by the Foundation for Forest Tree Breeding in spring 1963 in the field with a spacing of 1 or 2 metres. The pines were 14 years old when the *Lophodermella* epidemic started. The forest sites, mineral soil at Halkivaha and peatland at Kaksoiskivi, were very fertile. The peat layer at Kaksoiskivi thickened from zero to one metre in the direction of the slope, which was 0.6 %. The soil surface sloped 5.8 % at Halkivaha. The 3-hectare plantation of Halkivaha consisted of 4515 pines and the 7-hectare plantation of Kaksoiskivi of 8670 pines in the spring of 1978. Because of lower mortality Kaksoiskivi had been overdense and clearly denser than Halkivaha for many years. When the *Lophodermella* epidemic began at Halkivaha in the spring of 1977, a country road was built through the stand. The road is drawn in Fig. 13 F. The fertility of the soil was reflected as lush growth in both stands: long and thick branches growing through neighbouring tree rows, slow natural pruning of branches, breakage of leader shoots, leader changes and growth cessations prematurely during the growth period (growth disturbances) and bending of trees.

Multia, Kekälinen (693:38). The plantation, which was established in an abandoned field by the Department of Forest Genetics, the Finnish Forest Research Institute in 1970, consisted of open-pollinated clonal offspring. The trial trees (no 422/2) were subjected to the disease in the summer of 1978.

Rovaniemi, Sieväkari (736:48). The naturally born stand, aged 10—40 years, is situated in the Kivalo experimental area of the Forest Research Institute. Some pines were infected by *L. sulcigena* in summer 1979. New diseased trees appeared in 1983.

Rovaniemi, Kaihuavaara (736:49). This stand, planted in the year 1957, is situated on the NW-slope of the hill Kaihuavaara in the Kivalo experimental area. The quality of the trees is not good, and they have suffered to an exceptional extent from resin-top disease (*Peridermium pini* (Link.) Chev.). The *Lophodermella* epidemic began in the summer of 1979.

Rovaniemi, Juotas (736:49). This stand consisted of 20—40-year-old pines, which were born naturally or sown by the private owner. The site was a spruce (*Picea abies* (L.) H. Karsten) habitat. The epidemic began in the year 1979. All three Rovaniemi research stands are situated within 5 km of one other.

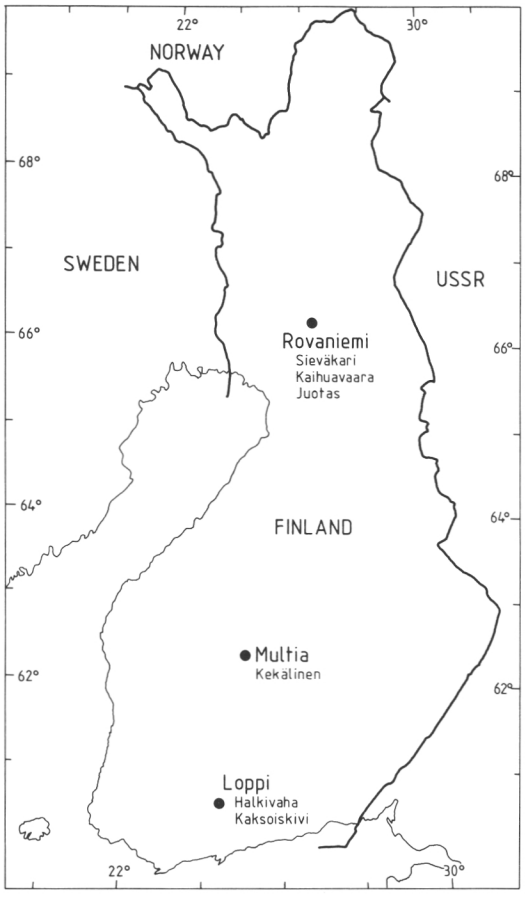


Fig. 1. The location of the experimental stands.

32. Disease definitions and surveys

The following disease terms are used in the thesis:

Disease incidence (DI). The relative amount of trees infected by *L. sulcigena* per area. A tree is infected if it belongs to one of the disease classes 2, 3 or 4.

Disease severity (DS). The relative amount of current-year short shoots infected by *L. sulcigena* per tree or shoot. The DS is always based on the count of removed short shoots, and can attain a value between 0 and 100 %. In the stand surveys, the DS is estimated after which trees are placed in one of the 4 disease classes.

Disease class (DC). In disease surveys, the trees are divided into 4 disease classes according to DS-estimates. Disease class 1 (DC 1) is composed of healthy trees, in disease classes 2, 3 and 4 (DC 2, 3 and 4) *L. sulcigena* has infected 1—33 % (slightly), 34—66 % (moderately) and 67—100 % (heavily) of the current-year short shoots, respectively (Table 1). This classification is used in the stand surveys only.

Disease degree (DD). This is always used for a group larger than one tree and calculated using the means of disease classes (see Table 1). For this reason, the DD could not attain a value greater than 83.5 %. If the DS has not been counted by removing the needles, the DD is used only.

Table 1. The scoring of *Lophodermella* needle cast for single trees and the class means for the assessment of disease degree.

Disease class	No. of diseased short shoots Range %	Class mean %	Description of the disease class
DC 1	0	0	healthy
DC 2	1—33	16.5	slightly infected
DC 3	34—66	49.0	moderately infected
DC 4	67—100	83.5	heavily infected

Disease index (DX). The ratio of the DDs of a sub-population and the whole population multiplied by 100.

When surveying the *Lophodermella* needle cast it was presumed that all partly or totally dead current-year needles were infected by *L. sulcigena*. A short shoot was considered diseased if one or both of its needles were necrotic. To maintain accuracy in estimating the proportion of diseased short shoots, the visual estimates were compared with figures obtained by removing the needles from shoots and by counting the total number and scars of short shoots, and diseased ones. The accuracy was tested especially before the surveys but also during them.

Disease surveys were carried out only at Halkivaha and at Kaksoiskivi, where DCs were estimated for all trees on the basis of the infestations of the summers 1977—1981 (Halkivaha) and in 1977—78 (Kaksoiskivi) either in the autumn of the year of infection or in the spring following the infection. Possible infections as early as 1976 were checked during the first survey in the spring of 1978 at Halkivaha. The plantation was also checked in the years 1982—83.

The relative size of all trees in relation to the nearest neighbouring trees was classified both at Halkivaha and at Kaksoiskivi in spring 1978 as follows:

1 = a tree below medium size
2 = a tree of medium size
3 = a tree above medium size.

The shoot contact of a tree with respect to neighbouring trees was determined for all pines by estimating the amount of the lower canopy which had contacts with neighbouring trees according to the following classification:

a tree was in connection with branches of neighbouring trees
1 = no connections (0 %)
2 = in 25 % of shoots
3 = in 50 % of shoots
4 = in 75 % of shoots
5 = on every side (100 %).

Also the shoot contact was estimated only once (in the spring of 1978).

Halkivaha and Kaksoiskivi were plotted using x-y-coordinates. The value of x was the number of the row and the value of y was the tree's distance (in metres) from the x-axis. The y-axis was parallel to the tree row. The trees were individualized using the coordinates for drawing disease maps. For this purpose the stands were divided into rectangles of 10 by 20 metres (Halkivaha) and 20 by 20 metres (Kaksoiskivi), in which there would be a maximum of 100 pines at the original planting density. The DD was calculated for each rectangle. Thereafter the rectangle net was removed so that every rectangle got DDs (calculated according to the same

principles) in the middle, on all sides, and in the corners. Only the rectangles in which at least 10 pines were located were taken into account. In this way the stands got a DD value every five or ten metres. In addition, both stands were divided into 3 subareas according to the height of the terrain.

33. Studies on the life cycle of the pathogen

Phenology of Scots pine, stages of the life cycle of *L. sulcigena* and disease symptoms were followed in the years 1978—1979 at Halkivaha, Loppi in five trees, a total of 22 shoots (DC 4). The time of initial colour changes in green needles and the period of disappearance of green colour from needles were determined for 33 labelled needles.

A slight infection of *L. sulcigena* would cause only a small visible spot on the needle. *L. sulcigena* can invade only the recently flushed needle base, which, according to Millar (1970), is not more than 5 mm on *Pinus nigra* var. *maritima* (Aiton) Melville). Therefore the length of the needle at the time of infection was supposed (Watson 1971) to be equal to the infection distance (= the distance from infection point (spot) to the tip of needle, Fig. 2). The time of infection was determined with the help of the growth curve for needles and infection distance. In all the locations of 71 infection spots on 64 needles of six shoots (2 trees) were measured.

Shoots of heavily infested pines were placed into kraft paper (Kaksoiskivi in 1979) or terylene pollination bags (other experiments) in order to see the time of infection. Before the experiments began at the end of May or in early June, needles infected by *L. sulcigena* were removed from the shoots so that not a single hysterothecium was left in the bags. From any given tree, 10—12 shoots were chosen for bagging with various exposure periods for each and with 3—6 trees as replications in every experiment. Three different bagging methods, (1a), (1b) and (2), were employed:

(1) All shoots were bagged before the needles started to flush.

(1a) Bags were removed for the rest of the summer weekly.

(1b) Bags were removed for one week during the summer.

(2) At the beginning of the experiment shoots were free, but new shoots were bagged weekly for the rest of the summer.

Control shoots were kept either in the bags or free all the time. DS was counted from these. Experiment (1a) was carried out at Kaksoiskivi in 1979, (1b) at Multia in 1980, at Kaihuavaara in 1981 and (2) at Kaihuavaara in 1984. All three methods were used at Juotas in the summer of 1985. At the end of August or in early September experimental shoots were cut for assessing DSs in the laboratory.

Short shoots with ascocarps of the pathogen in both needles were collected from the trees used in bagging experiments. Weekly (Kaksoiskivi) or at 2—3-day intervals (Sieväkari, Juotas) ten short shoots were placed into a Petri dish with water. After 3 hours the opening of hysterothecia was determined (water test). Only the number of needles with open hysterothecia was checked, not the maturing of all hysterothecia in a needle. The test periods were the following: 25 May to 12 June 1979 at Kaksoiskivi, 1 June to 15 July 1981 and 1 June to 20 August 1984 at Sieväkari, and 25 June to 13 August 1985 at Juotas. The hysterothecia are defined as mature when they open with a longitudinal slit in humid conditions (water test of this report).

The needles of the years 1981 and 1984 from Sieväkari were stored in FAA (formalin-alcohol-acetic acid in test tubes). One half of the material was embedded in paraffin, sectioned and stained with

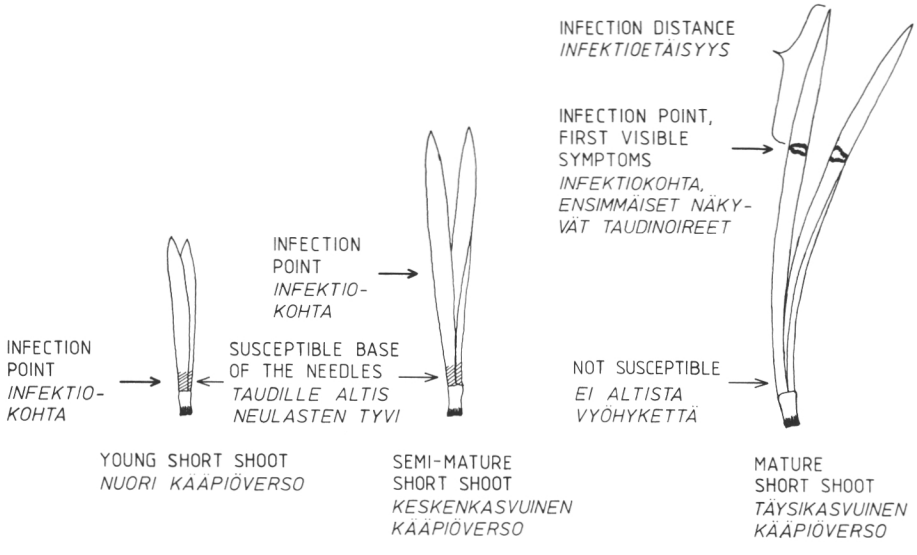


Fig. 2. A schematic presentation of the area on the base of the needle susceptible to *Lophodermella sulcigena* during the elongation of the short shoot and of the determination of infection distance (adapted from Watson 1971).

safranin-fastgreen in order to confirm the results of the water test and to ascertain the emptying of ascocarps.

Ascospores of *L. sulcigena* were trapped from 14 June to 16 July 1984 at Sieväkari. A Rotobar trap was surrounded by living branches of some moderately infected pines (the same as in the water test). In the vicinity a Burkard volumetric spore trap (e.g. Millar and Bird 1971) was surrounded by newly cut shoots infected by *L. sulcigena*. The shoots were kept in water pails. During the trapping precipitation, relative humidity and temperature were recorded with a thermohygrograph and a pluviograph.

34. Determinations of needle shedding

Firstly, the needle shedding was determined directly by counting the amount of needle scars at intervals of 1—4 weeks from May 1978 to September 1979 at Halkivaha. Secondly, the fall of short shoots to the ground was ascertained with eleven litter funnels placed under the heavily infected trees. The diameter of the opening of a funnel was 0.25 m. The litter samples were gathered in a cloth bag under the funnel (Fig. 3). Six funnels were situated at Halkivaha and five at Kaksoiskivi. The gathering of litter began on 23 May 1978 and ended on 24 December 1980. In May—August the funnels were emptied every week, otherwise at intervals of 2—4 weeks. The funnels were situated at a distance from the stem equal to the distance of shoots of 1977 from the stem at the middle height of the living crown. The location of the funnels was not changed during the collection period, which presumably has altered the litter composition.

The litter samples were brought in paper bags to the laboratory. When analyzing the short shoots they were divided into two groups:

1) shedding due to the *Lophodermella* needle cast, and
2) shedding due to another reason.

In addition, the needles infected by *L. sulcigena* were divided into current-year short shoots and into ones infected one year earlier. From the litter it was also checked whether one or both needles of the short shoot were infected by *L. sulcigena*. Shedding for another reason was considered as normal shedding of 3—5-year-old senescent needles.

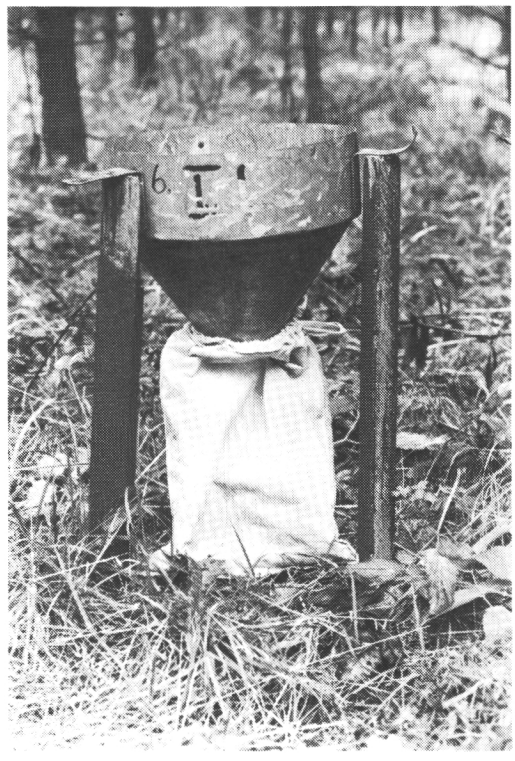


Fig. 3. A litter funnel for collecting needles of Scots pine.

35. Needle measurements

Needle elongation curve was based on the measurements of the lengths of 10 needles growing in the base, mid and top part of each shoot. Measurements were carried out weekly. The fascicular sheath was not measured.

The effect of the *Lophodermella* needle cast on needle elongation was examined at Juotas, Rovaniemi on the basis of the infection in the summer of 1985. Altogether 2448 needles (1224 short shoots) were collected and their lengths measured from 5 pines, taking 10 shoots from each tree. The short shoots of every shoot were divided into the following three types:

1) both needles healthy (H/H),
2) one needle healthy, the other diseased (H/D), and
3) both needles diseased (D/D).

The diseased needles which were approved for the material had to be necrotized over more than half of the length of the needle. The needles of spot type were thrown away. The length of the needles of the short shoots were measured to an accuracy of 0.01 mm using a growth ring microscope. A total of 50 observations was obtained for types H/H and H/D. However, for type D/D only 21 observations were obtained because of low DS. The material (1224 short shoots) showed a preponderance of types H/H (72.4 %) and H/D (24.9 %). Thus, the type D/D was represented only by 33 short shoots (2.7 %).

36. Determination of stand growth losses

361. Sample trees

The effect of the *Lophodermella* needle cast on the growth and development of a Scots pine stand was studied at Halkivaha. Every n:th tree of a class was determined for each DC. The size of n depended on the total number of trees in the class so that at least 50 sample trees for each DC would be obtained. The sampling was started from row one and ended in the last row (100) in the same order as the *Lophodermella* needle cast was surveyed.

The average DD of sample trees in three intermediate years during the 5-year epidemic had to be the same as that for the DC which the tree represented. However, the following conditions had to be met: The tree should not grow at the edge of the stand, nor

should its shoot contacts with neighbouring trees be zero or 100 %. The tree also had to be of medium size when compared to the neighbouring trees in the spring 1978. Noticeable leader changes or other injuries weakening growth were not allowed during the period of increment measurement. If the tree selected from the adp-list did not fulfill the requirements, the nearest tree from the same row fulfilling them was taken instead. Leader change was the most common reason for rejecting a tree in the field. Its general occurrence was calculated to be 38 % for the total number of trees in the stand at Halkivaha.

There was a total of 210 sample trees for growth calculations, which was 5.0 % of the total number of pines (4207) in the stand at the moment of felling (Table 2).

Table 2. The distribution of sample and all trees in to disease classes according to the average disease class of the years 1978—1980 at Halkivaha.

Average disease class	Disease classes in the years 1978—1980	Total number of trees in the stand	sample trees
1	1, 1, 1	512	49
1	2, 1, 1	447	—
2	2, 2, 1	527	—
2	3, 1, 1	3	—
2	2, 2, 2	1487	49
2	3, 2, 1	31	—
2	3, 2, 2	397	1
2	3, 3, 1	3	—
2	4, 2, 1	2	—
3	3, 3, 2	206	6
3	4, 2, 2	53	—
3	4, 3, 1	1	—
3	3, 3, 3	59	39
3	4, 3, 2	145	1
3	4, 4, 2	55	—
3	4, 3, 3	88	5
4	4, 4, 3	126	15
4	4, 4, 4	65	45
Together		4207	210

362. Felling and measuring of sample trees

The sample trees were felled in August of 1982. The breast height and north were marked on the sample trees before felling. The breast height diameter ($d_{1.3}$) was measured at the same time. The measurements were always made from the same point of the compass. The trees were cut to a 0.1 m stump. Tree height and the annual height increments for the years 1970—82 were measured after the felling. Still, possible leader changes were checked when measuring the heights.

Disks of 0.02 m in thickness were sawn from every sample tree at the height of 0.1, 0.5, 1.3 and 2.0 metres and after that at every even-numbered height in metres right up to the leader. The mean height of the sample trees in 1982 was 7.68 m (the shortest tree being 5.65 m, the tallest 9.81 m), mean diameter 10.0 cm and mean volume 42.2 dm^3. The age was 20 years. Sample tree number, sawing height and an arrow indicating north were marked on the upper surface of the disks. All disks from one tree were placed in a paper bag with the number of the sample tree. The bags were placed in cold storage in order to prevent the disks from molding until conducting of measurements.

In the laboratory the lower surfaces of the disks were planed smooth in order to make it easier to distinguish annual growth rings. The radius was measured with and without bark to an accuracy of one millimetre. The annual growth rings were defined only from one radius which pointed east in the stand and which was the same as in the measurement of the radius without bark. The annual rings were measured with a stereo microscope with the scale in the ocular. All growth rings from the cambium till the pith were measured. The smallest unit on the ocular measure represented 0.17 mm, which was the accuracy of the annual growth ring measurement.

363. Growth calculations

Volumes of the trees were calculated as sections by using the equation of a cone of frustrum (1) (Snellman 1984).

$$(1) \qquad Vol = 0.26177994h\,(d_0d_0 + d_0d_1 + d_1d_1),$$

where Vol = volume of the bolt, d_0 = the lower diameter of the bolt, d_1 = the upper diameter of the bolt, and h = the height of the bolt.

In the growth loss calculations, two 5-year periods were used. The former comprised the years 1972—1976 representing the time before the *Lophodermella* epidemic ('healthy period', HP), the latter the years 1978—1982, representing the time of the epidemic ('epidemical period', EP). Although the epidemic started in 1977, this year, judging from the growth curves, could not be assigned reliably to either period. By omitting the year 1977 these two periods could be clearly distinguished as periods without and with the disease.

The so-called *growth ratio* (GR) was calculated at first for all the years of the HP and for every DC by comparing the growths of trees in DCs separately to the growths of the healthy tree class (DC 1). The GRs were determined for each growth type, height, radial and volume increments. Finally, because of the non-significant variation within a growth type between years, the average GRs were calculated for height, radial and volume growths of the HP.

Using the GRs and the measured growth increments of trees in DC 1 during the EP the growth estimates $i_{y(est)}$ were calculated for DC 2—4 for each year of the epidemic. The growth estimates were thought to correspond to the real growth increments had there not been a *Lophodermella* epidemic in the stand, and the GRs between healthy and diseased trees would not have been changed without the disease. Equation (2) was used for calculating growth estimates.

$$(2) \qquad i_{y(est)} = r_{t(d)}\, i_{1(y)},$$

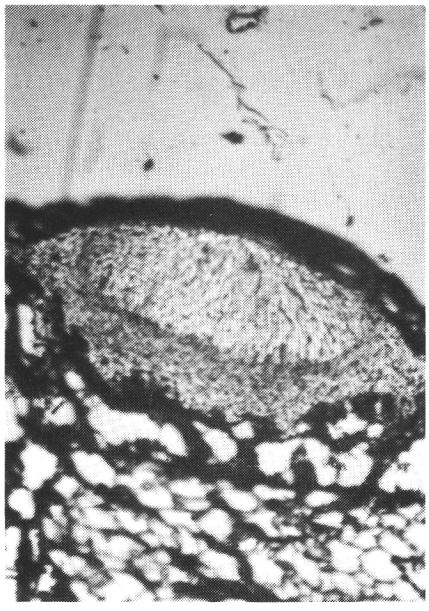
where y = a year of the epidemical period, d = disease class, $i_{1(y)}$ = mean increment of DC 1 in the year y, and $r_{t(d)}$ = the growth ratio of the growth type t (height, radial, volume) in the disease class d. Estimates for DC 2—4 for the whole epidemical period $i_{d(est)}$ were calculated by equation (3).

$$(3) \qquad i_{d(est)} = \sum_{78}^{82} i_{y(est)}$$

Relative growth losses $i_{d(loss)}$ were calculated by equation (4).

$$(4) \qquad i_{d(loss)} = 100 - 100 \frac{i_{d(est)}}{i_d},$$

where d = disease class, $i_{d(est)}$ = the estimate for the growth of disease class d in the epidemical period, and i_d = the real measured growth of the disease class d in the epidemical period.



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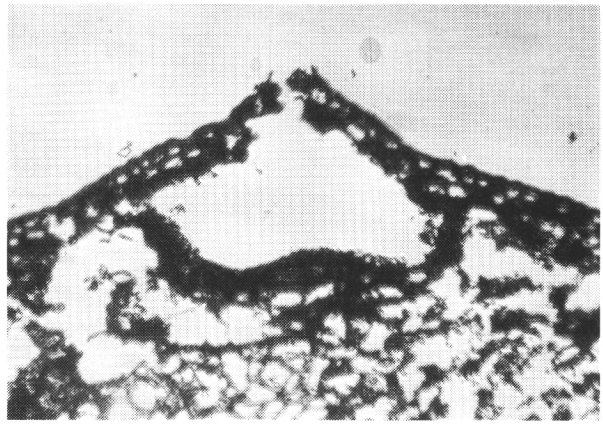

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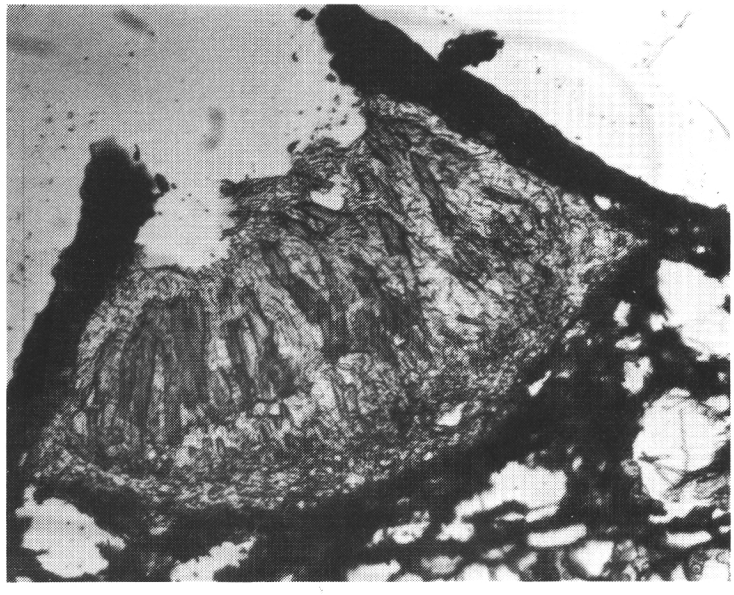

Figs. 4—6. Different stages of a hysterothecium of *Lophodermella sulcigena* at Rovaniemi. 4) Developing in February, magnification 250x. 5) Mature, opened hysterothecium with asci and ascospores in July, 500x. 6) Empty hysterothecium in September, 200x.

4. RESULTS

41. The life cycle of the pathogen and disease symptoms

411. Ascocarp development

The first signs of developing ascocarps on needles were seen as longitudinal striations in November, about five months after infection. In February 1981, needles from Rovaniemi already bore ascocarps in their final shape and structure, and the needles had swollen at the point of the ascocarps (Fig. 4). In the spring the ascocarps swelled further, and asci developed in them.

The maturation of ascocarps (Fig. 5) depended on the timing of growth season and the warmth of early summer. The first ascocarps opened as early as the end of May in southern Finland (Loppi in 1979), if early summer was warmer than average. In northern Finland ascocarps opened in mid-June at the earliest when the warm early summer of 1984 occurred, or, at the latest, during the last week of June in the exceptionally cold early summer of 1981, or when the growth of shoots started many weeks later than normal (1985).

The period from the first open ascocarps to the presence of open ascocarps in all needles was 2 weeks in a warm season and 3 weeks in a cold one. Further ascocarps opened within 7 weeks; some did not open at all. The first mature ascocarps were seen yearly almost on the day the needle flush commenced. Thus, the maturation peak coincided with 2—3 first weeks' growth of needles.

412. Sporulation

Ascocarps generally bore spores throughout July to early August in northern Finland. The matter was not checked in southern Finland, but the maturing period should start earlier than in northern Finland

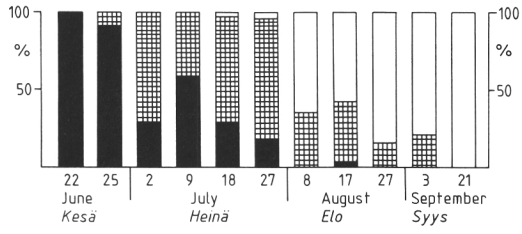

Fig. 7. Relative amount of immature (black columns), mature with ascospores (columns with squares), and empty hysterothecia (white) in summer 1984 at Rovaniemi. Data is based on ten needles from each date and determined from microtome sections by light microscope.

although it is equally long (6—7 weeks). The first empty ascocarps in summer 1984 were found in late July, at the time when developing ascocarps still numbered about 20—30 % of the total (Fig. 7). The span from the first open ascocarps to first empty ones lasted about 5 weeks. In August 1984 more than half of the ascocarps were empty. In September no open hysterothecia had ascospores or asci (Fig. 6).

The first ascospores were trapped with a Rotobar in Rovaniemi on 14 June 1984, a day after the first ascocarps had opened in the water test. In five days, when every second needle had mature ascocarps, the amount of ascospores in the air multiplied. Thereafter, spores were trapped in the air continuously, nearly every day to the end of the observation period (16 July). The maximum number of ascospores reached 40 000 per cubic metre of air.

Sporulation started 1—3 hours from the beginning of rain (Fig. 8). Even a short shower induced a sporulation of 4—8 hours; after a rain it normally ceased, although the relative humidity (RH) remained at 90—92 %. However, sporulation was measured in as low a RH as 79 %. During heavy sporulation the RH was at least 91 %. In early July ascospores discharged without

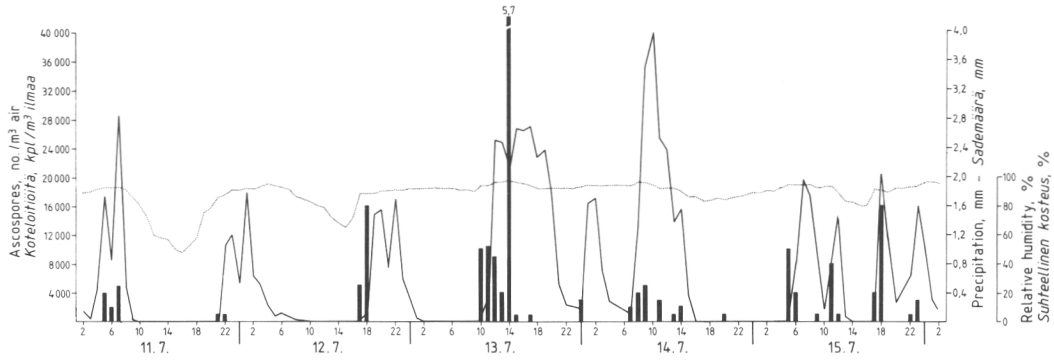

Fig. 8. Precipitation (columns), relative humidity (dotted line) and number of ascospores of *Lophodermella sulcigena* in the air (solid line) from 11 to 15 July 1984 at Rovaniemi.

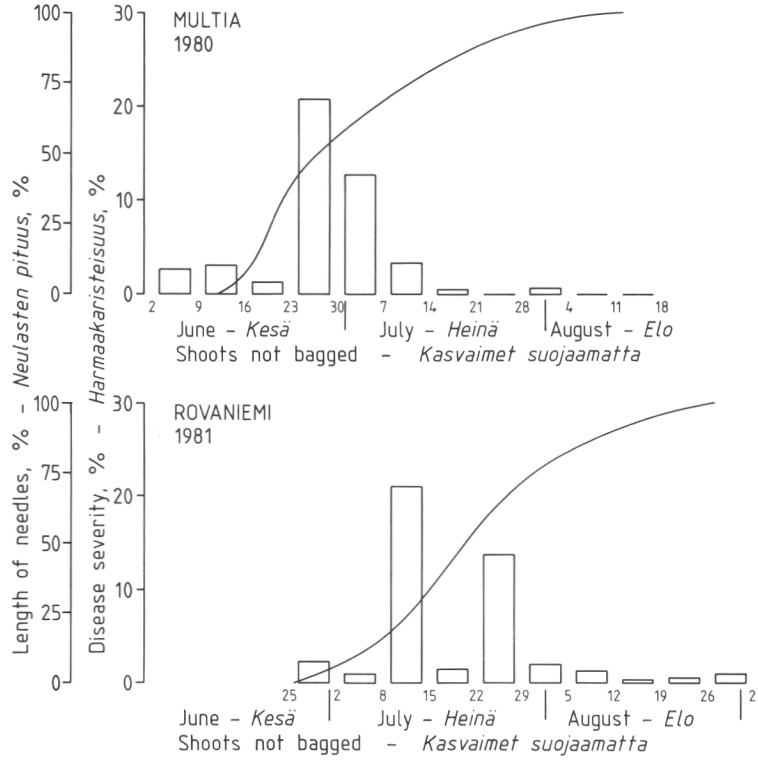

Fig. 9. Growth curve of needles and disease severity (DS) of shoots exposed in 7 days' periods to infections of *Lophodermella sulcigena* at Multia, central Finland in the summer of 1980, and at Rovaniemi, northern Finland in 1981 in bagging experiments. DS of shoots exposed all the time valued 95.3 % at Multia and 87.0 % at Rovaniemi. Shoots with no exposure had the DS of 2.4 % at Multia and 0.3 % at Rovaniemi.

rain during the night time. This was no longer observed in mid-July.

413. Infection time of shoots

Although ascocarps are mature and spores can be discharged and deposited on the needles, infection does not always take place, owing to unsuitable environmental factors. This was demonstrated by the different bagging experiments in which needles were infected in different ways.

The needles in the Kaksoiskivi plantation were infected in June 1979. In the neighbouring plantation (Halkivaha at Loppi) the infection of the year 1978 was regarded as having occurred mainly in the latter part of June and only to a minor extent in early July (Fig. 12, white columns). The infection of 1980 at Multia concentrated at the end of June and early July (Fig. 9). The early summer of 1980 was very warm.

Pines at Rovaniemi were infected mainly during the second and last week of July in the rather cold summer of 1981. Slight infection may have occurred as early as the end of June (Fig. 9). In the summer of 1984 needles of the stand were infected mainly from 19 to 26 June and from 3 to 10 July (Fig. 10). Times favourable for infection were concentrated in 1985 between 16 and 23 July, when needles had already grown to three quarters of their final length. Slight infection occurred towards the end of August (Fig. 11). The lowered growth rate at the end of the needle elongation may have contributed to the exceptionally low DS (11.7 %) in the summer of 1985.

With the exception of summer 1985 infections by *L. sulcigena* coincided with the first half of needle elongation (2—3 weeks from the beginning of it and from the maturation of the first ascocarps).

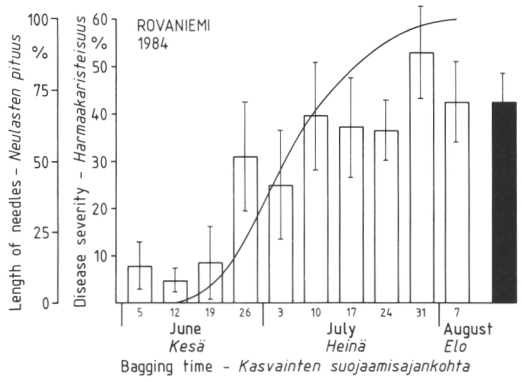

Fig. 10. Growth curve of needles and disease severity (DS) of shoots exposed to infections of *Lophodermella sulcigena* in the summer of 1984 at Rovaniemi. The date is the day when exposing to infection was hindered (= shoots were bagged) with a terylene bag. The DS of unbagged shoots was 42.5 % (black column).

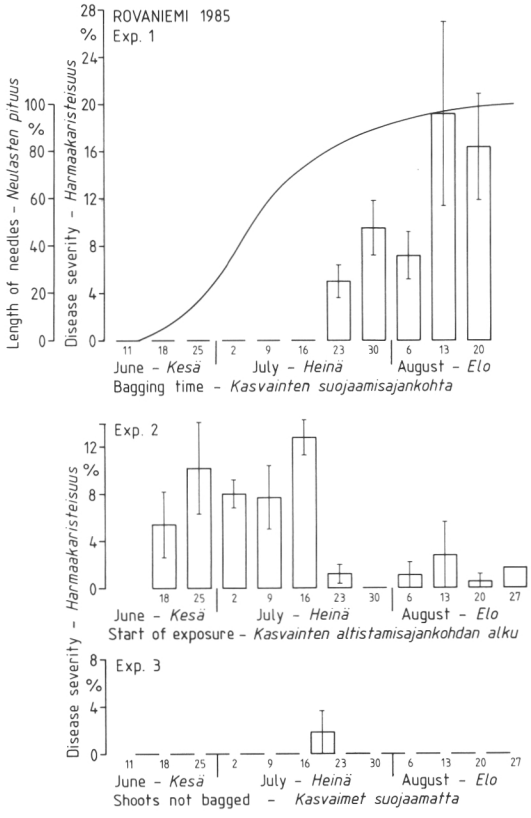

Fig. 11. Growth curve of needles and disease severity (DS) of shoots exposed to infections of *Lophodermella sulcigena* in the summer of 1985 at Rovaniemi. All three experiments have been carried out in the same trees. The date is the day when exposure to infections began (= bags were removed). In the Exp. 3 shoots were without a bag for a week. Shoots exposed all the time had a DS of 11.7 %.

414. *Disease symptoms*

At the time of the last infections, the initial disease symptoms appeared on other needles at Halkivaha in 1978, the exact date being 19 July. The site of infection on the needles turned greyish green, this colour lasting not more than a week. Soon, the base colour of the needles changed to brown or yellow. In most needles the first disease symptoms were seen in the last week of July or in the first week of August. About 90 % of the records came from this two-week period (Fig. 12, black columns). The appearance of symptoms on new needles was no longer found from 9 August onwards.

On some needles *L. sulcigena* caused only a spot of a few millimetres in the infection site. In most cases the initial colour change expanded to both the tip and base of the needle. This led to quick spread of necrosis (except in the green base of the needle) in about two months from the initial symptoms in mid-July to its cessation (Fig. 12, line B). The disappearance of green colour was fastest in the needles which showed the symptoms first, the maximum rate of spread being 12.2 mm a week. The needle shedding which started as early as September, is dealt with more extensively in Section 43.

Three damage types were described according to the symptoms caused by the primary pathogen and secondary colonisers together. The descriptions are based on visible symptoms only.

1) Spot type: The main symptom is the infection spot caused by *L. sulcigena*. It is a 1—3 mm wide yellow or brown band or, often, a round fleck. Many spots may be in a single needle. Spots remain unchanged until the needles shed normally at the age of 3—4 years. Needle tips stay healthy and green. Resin flow was seen sometimes on the infection spot. Neither ascocarps of *L. sulcigena* nor pycnidia of *H. acicola* develop in this type of needle. The spots seem to have no harmful effects on trees.

2) Slowly expanding damage: The tip of the needle turn reddish brown in late summer of the infection year. A clear boundary is seen between the necrotic tip and the green base. The pathogen does not kill the green base of the needle during the infection year, but it dies after the winter, turning light brown. Needles shed down in the summer following the infection. Ascocarps mature mostly in this type of needle.

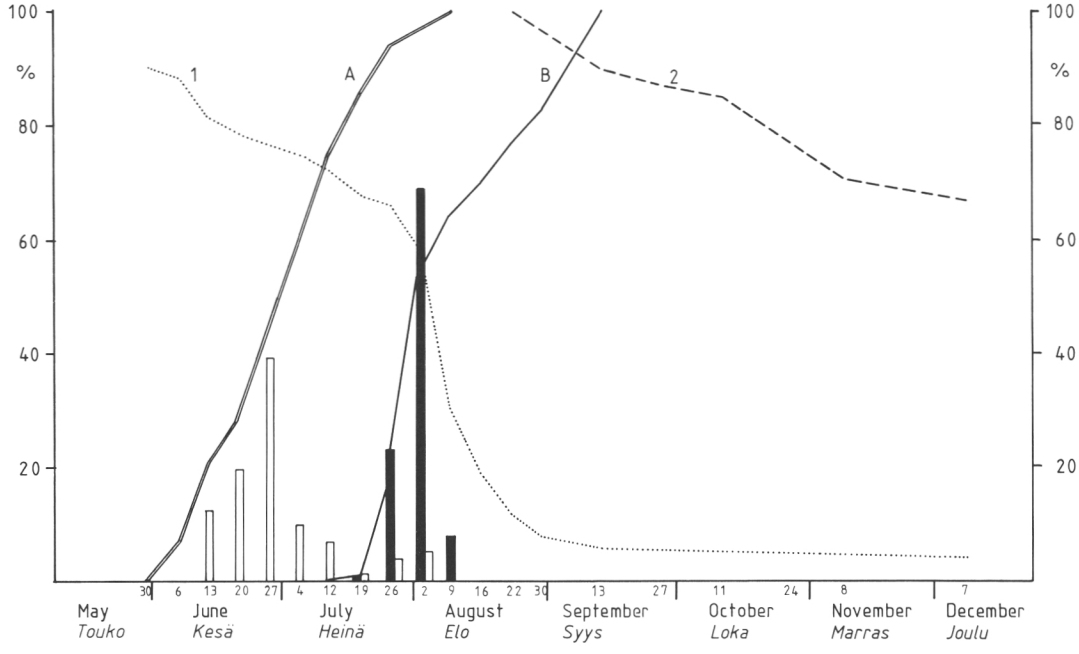

Fig. 12. Infections by *Lophodermella sulcigena* of 1978, the appearance and development of disease symptoms and growth curve of needles and needle shedding at Halkivaha, Loppi. Symbols: A = growth curve of current-year needles; B = proportion of necrotic area from the final length of necrotic needle part; white columns = percentage of infection at different times; black columns = percentage of appearance of first disease symptoms at different times; number of previous-year (1) and current-year (2) needles in shoots.

3) Rapidly expanding damage: The pathogen colonises the bare part of the needle very quickly, making it reddish brown as early as end of August. The base in the fascicular sheath may stay green if the short shoot remains in the long shoot over the winter. Still, these needles start to fall as early as September, at the age of three months. In some trees, infections cause a hypersensitivity reaction which can turn a needle light or yellowish brown in the autumn of the infection year. However, the needles would in any case shed down when touched the following June. These needles play a minor role as an inoculum source of *L. sulcigena.*

The symptoms in these main types were affected by other fungi, especially *H. acicola*, whose presence was most easily seen in type 3. The role of *H. acicola* as regards needle colour and destruction has been discussed elsewhere (Jalkanen 1985, Jalkanen and Laakso 1986).

42. Dynamics of the epidemic

The *Lophodermella* epidemic was detected in the stands of Halkivaha and Kaksoiskivi in the summer of 1977. Judging by symptoms and the shedding of older needles, the epidemic had started in 1976. It ceased in the summer of 1982 preceded by a low DD in the summer of 1981.

The DI varied from 74.5 to 82.7 % in the years 1977—1980 at Halkivaha and from 80.5 to 83.5 % in the years 1977—1978 at Kaksoiskivi. The DD varied from 16.0 % to 22.2 % at Halkivaha and from 20.9 % to 22.7 % at Kaksoiskivi. Despite the decrease in DI, the DD increased towards the year 1981, when both the DI and the DD dropped (Table 3). If one considers the years 1977—1978 the disease seems to have been more severe at Kaksoiskivi than at Halkivaha. However, the stand of Kaksoiskivi recovered in the same year as Halkivaha.

The number of trees in the disease classes varied yearly. The proportion of trees in DC 3 and 4 was at its lowest in the first and last years of the epidemic. At its highest, it increased to 21.6 % in the year 1980, whereas in the year 1977 it had been 6.5 % (Table 4). The proportion of these classes at Kaksoiskivi was 15.9 % in 1977 and 20.2 % in 1978. Judging by the number of trees in DC 3 and 4 the three middle years were the heaviest years of the epidemic.

At the beginning of the epidemic the disease was most severe in the thickets of the lowest part (127—130 m above sea level) of the Halkivaha stand, and the healthiest areas were situated in the uppermost part of the stand (133—136 m above sea level). The lowest part reached its highest DD in the second year of the epidemic and started to recover in the third year, towards the end of the epidemic (Table 5). At the same time

Table 3. Disease incidences (DI) and disease degrees (DD) at Halkivaha in the years 1977—1981. Trees were diseased if they belonged to one of the disease classes 2, 3 or 4.

	The year of infection				
	1977	1978	1979	1980	1981
DI, %	82.7	78.5	74.5	75.8	3.4
DD, %	16.0	19.3	21.2	22.2	0.5

Table 4. Number of trees in different disease classes (DC) at Halkivaha in the years 1976—1982.

The year of infection	Disease class				
	DC 1	DC 2	DC 3	DC 4	Total
	Trees, %				
1976	100.0	0.0	0.0	0.0	100.0
1977	17.3	77.2	4.0	2.5	100.0
1978	21.5	64.6	8.8	5.1	100.0
1979	25.5	54.8	12.2	7.4	100.0
1980	24.2	54.2	14.0	7.6	100.0
1981	96.6	3.4	0.0	0.0	100.0
1982	100.0	0.0	0.0	0.0	100.0

Table 5. The disease degrees (DD) and disease indices (DX) at different elevations at Halkivaha in 1977—1981.

Elevation, m a.s.l.	The year of infection									
	1977		1978		1979		1980		1981	
	DD	DX	DD	DX	DD	DX	DD	DX	DD	DX
127—130	19.7	123	25.0	130	22.6	107	20.8	94	0.35	64
130—133	15.4	96	20.0	104	23.6	111	25.2	114	0.69	125
133—136	12.0	75	12.4	64	17.0	80	20.1	91	0.60	109
Mean	16.0	100	19.3	100	21.2	100	22.2	100	0.55	100

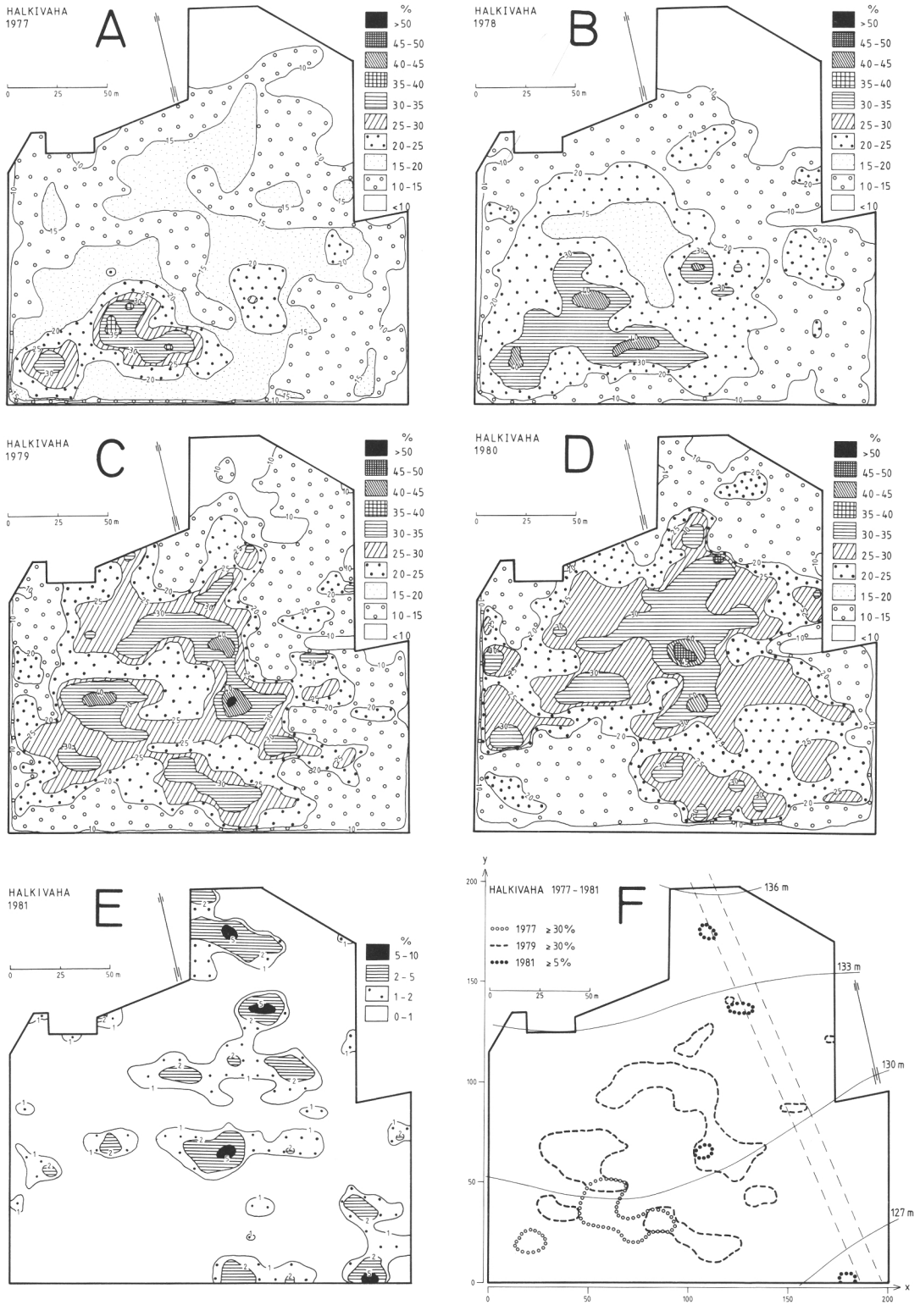

Fig. 13. The distribution of the *Lophodermella* needle cast at Halkivaha stand in 1977—1981 (A—E) and the occurrence of the worst disease centres in 1977, 1979, and 1981 with contour lines, the location of the country road built during the epidemic, and the coordinate axes for individualizing the trees (F).

the middle and uppermost parts of the stand became more diseased through the year 1981. Compared to the stand mean of the year 1977 the disease index (DX) was above 100 in the lowest part of the stand and below this in both of the other parts. In the last year (1981) of the epidemic the situation was exactly the reverse. The differences between disease indices were highly significant ($\chi^2 = 45.0$; d.f. = 8). The differences between the lower and upper parts of the stand were clearly explained by the number of trees in DC 4. While 73.4 % of the heavily infected trees in 1977 were situated in the lowest part of the stand, the amount in 1980 was only 26.7 %. Thus, the number of heavily infected pines increased in the upper parts during the epidemic, whereas the number of healthy trees decreased.

There were many disease centres changing place and extent in the stand. The DD of the heaviest disease centre was over 35 % in the year 1977 and over 40 % in 1978. This area differed distinctly from its surroundings, which in the uppermost parts and edge of the stand showed DDs of not more than 10 % (Fig. 13 A). In the second year (1978, Fig. 13 B) a new, small but heavy disease centre had appeared in the middle of the stand. The uppermost parts and edge remained the healthiest. In the third year (1979) the heavy disease centre, which had formed two years earlier, began to disappear, and many new disease centres appeared which were small in extent and of a lower DD. The heavy disease centre of the middle part had increased and its DD had risen to over 50 % (Fig. 13 C). The area with a DD of not more than 10 % had become smaller. In the fourth year (1980) the worst disease centres had moved further upwards in the stand, and one of them (maximum DD over 45 %) was already located in the uppermost part. The disease centre in the middle of the stand increased, and a new centre with a DD of over 20 % had appeared in the uppermost part (Fig. 13 D). The lowest part of the stand had recovered such that in the place of the heaviest disease centre of the first year of the epidemic the DD was only 10—20 %. In the fifth year (1981) the epidemic nearly ceased. The DDs of the worst disease centres varied between 5 and 10 %. In most parts of the stand the level was not more than 1 %. One of four centres with a DD of over 5 % was located in the uppermost part, which had been part of the healthiest area during the whole epidemic (Fig. 13 E).

Table 6. The disease degree at different elevations and on soils with the mean thickness of the peat layer at Kaksoiskivi in 1977 (according to the number of trees in various disease classes and in elevation/thickness types the χ^2-test value was 1126***; d.f. = 6) and in the year 1978.

The year of infection	Subarea 1	2	3	On the average
	Elevation, m a.s.l. 114—115	115—116	116—117.5	
	Mean thickness of the peat layer, m 0.30	0.55	0.85	
	Disease degree, %			
1977	27.3	21.6	13.0	20.9
1978	28.3	22.7	17.1	22.7

During its 5 years the *Lophodermella* epidemic broke out with heavy disease centres in the lowest part of the stand in 1977, continued with the heavy centres in their new locations in the middle and uppermost parts of the stand; at the same time, the trees of the oldest centres of the lowest part started to recover and the centres began to disappear (Fig. 13 F). The Halkivaha stand was healthy by the year 1982.

At Kaksoiskivi the differences in DDs between different elevations were statistically significant ($p < 0.001$), but as the differences in elevation between the lowest and the uppermost subarea was only 3 metres, the differences in DDs may be due to the combined effect of elevation and soil type (Table 6). The uppermost part was partly on mineral soil, whereas the lowest part of the stand had as much as one metre of peat in places. The disease centres had higher DDs at Kaksoiskivi (Fig. 14) than at Halkivaha.

Both at Halkivaha and at Kaksoiskivi, the DDs (33.5 and 32.7 %) of the trees (max. of 8 pines) around the heavily infected trees (DC 4) were higher to a statistically significant extent ($p < 0.001$) than the ones in the whole stand (16.0 and 20.9 %) in 1977. Heavily infected trees had other trees in DC 4 among their nearest trees more often than on average in the whole stand (Table 7).

Both at Halkivaha and at Kaksoiskivi, the *Lophodermella* needle cast was most severe in the pine thickets of the inner parts of the stand. In general, the edges of the stand were the healthiest parts. One exception to

this was that the southwestern edge of the Kaksoiskivi stand had, in fact, rather heavy disease centres. This was due to another plantation of the same size and age just beneath the Kaksoiskivi stand. The northeastern edge of the Kaksoiskivi stand was bounded by a 15-hectare cultivated field. The nearer the tree row was located to the field, the lower its DD (Fig. 15). The DD increased through rows 12—14, i.e. 24—28 metres from the field. The mean DD of the three subareas numbered 1—3 in Fig. 14 was 9.6 % in the 1st row from the field, and 26.3 % in the 14th row in 1977.

The trees below medium size were statistically significantly ($p < 0.05$) healthier than trees of medium size or above medium size. In 1977 the most heavily diseased pines at Halkivaha were the trees above medium size. In 1978—1980 the differences between these three groups became clearer and were highly significant ($p < 0.001$, Fig. 16). In 1977 the trees below medium size were 8 % healthier than the trees of medium size. In 1980 the difference was 47 %. The trees above medium size were 9 % and 36 % more diseased than the trees of medium size in these years, respectively. In the plantation of Kaksoiskivi nearly 90 % of the pines were classified as being of medium size, and no differences in DD between trees of various sizes were shown.

When the shoot contact of pines with their neighbouring trees was zero or 100 % at Halkivaha in 1977, the amount of *Lophodermella* needle cast was at its lowest.

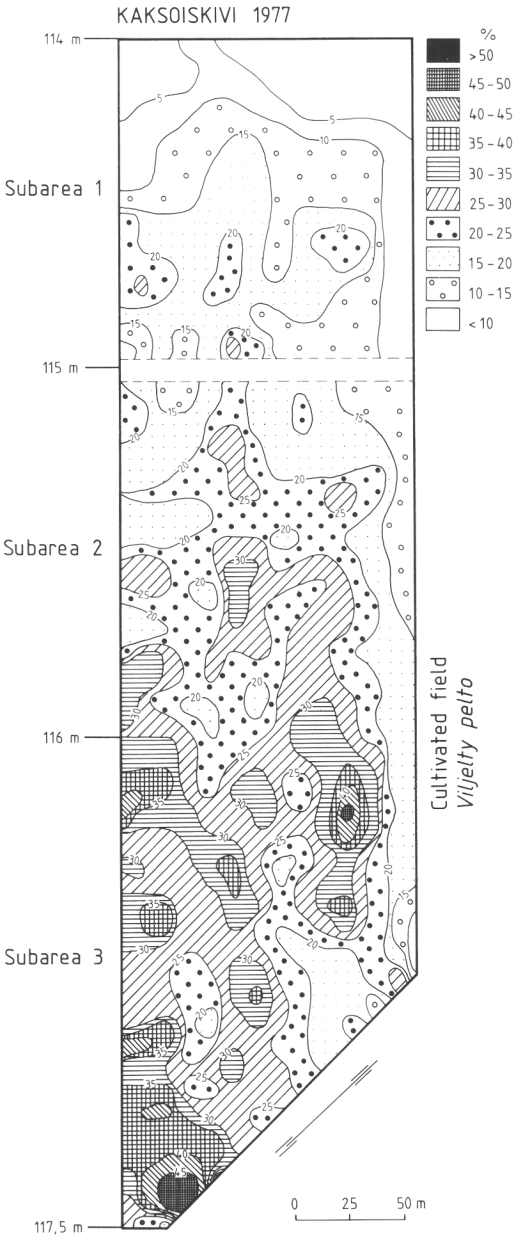

Fig. 14. The distribution of the *Lophodermella* needle cast at Kaksoiskivi stand in 1977. The partition of to subareas 1—3 and corresponding elevations (m a.s.l.) are shown in the southwestern edge of the stand, wherefrom another planted Scots pine stand of similar age and size continued.

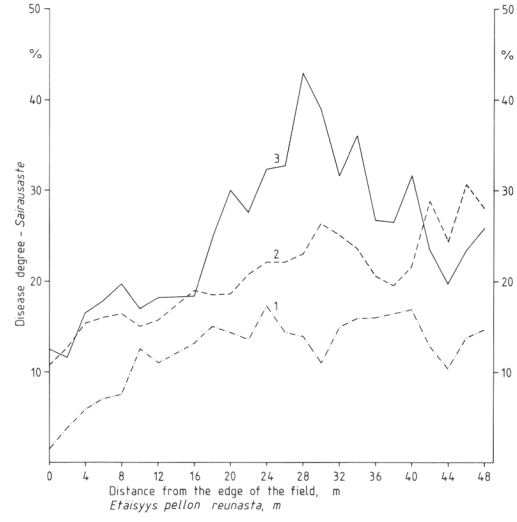

Fig. 15. The dependence of the amount of the *Lophodermella* needle cast on the distance of tree row from the field-side edge of the Kaksoiskivi stand in subareas 1—3 shown in the map of Fig. 14.

Table 7. The distribution of trees as to disease class in small squares around the heavily infected trees (A), and in the whole stand (B) of Halkivaha and Kaksoiskivi in 1977. The number of squares was the same as the number of trees in disease class 4.

Area	Disease class: DC 1 Trees no.	DC 1 %	DC 2 no.	DC 2 %	DC 3 no.	DC 3 %	DC 4 no.	DC 4 %	Total no. of trees
				Halkivaha ($\chi^2 = 464.4^{***}$)					
A	5	2	182	60	74	24	42	14	303
B	779	17	3487	77	180	4	69	2	4515
				Kaksoiskivi ($\chi^2 = 558.1^{***}$)					
A	101	4	1366	61	408	18	371	17	2246
B	1413	16	5877	68	902	10	478	6	8670

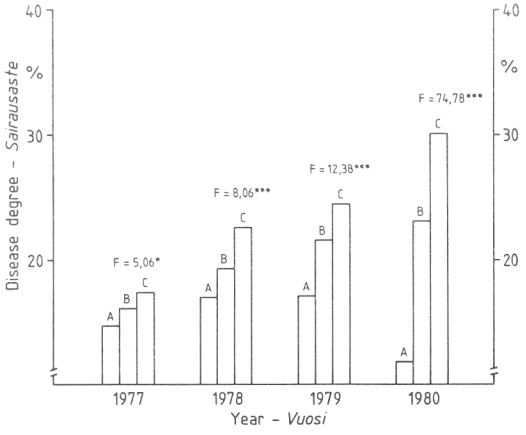

Fig. 16. *Lophodermella* needle cast degree in the trees of different size, the test values of one-way analysis of variance and their significance at Halkivaha in the years 1977—1980. Symbols: A = trees below, B = trees of, and C = trees above medium size compared to the nearest surrounding trees.

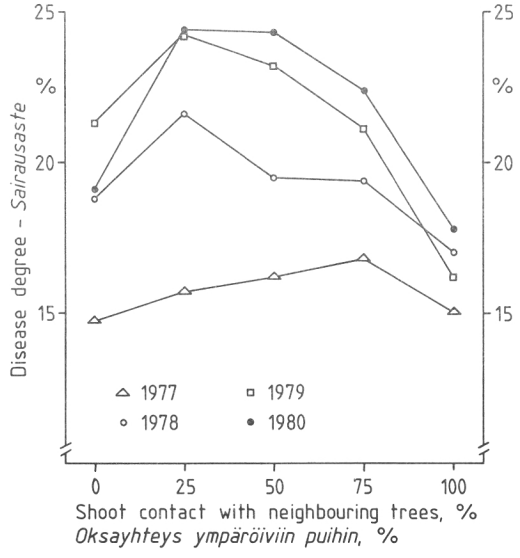

Fig. 17. *Lophodermella* needle cast degree in the trees with different shoot contacts with neighbouring trees at Halkivaha in 1977—1980.

The trees were more diseased when the amount of shoot contact was 25—75 % (Fig. 17). In the years 1978—80 the highest DD was reached, when shoot contact (classified before the growth of 1978) was 25 %. The results were similar at Kaksoiskivi.

Because the DD of the stand varied annually, the DC of a single tree necessarily varied as well. The highest stability of the same DC from 1977 to 1978 was in heavily infected trees (96.9 %). This means that nearly all pines of DC 4 in 1977 were similarly infected in 1978. Healthy (71.3 %) and slightly infected trees (77.0 %) had also high stability when compared to the low level of the trees in DC 3 (37.0 %). An explanation for the low level may be the relatively greater difficulty of placing a tree in DC 3 as compared to classes 2 or 4. The average stability in the same DC between two successive years was 70.7 %. The percentage of trees which were in the same DC in all four years 1977—1980, was 39.0 % in DCs 1 and 2, 29.0 % in DC 4, and only 2.0 % in DC 3. In the first year of the epidemic 741 pines stayed healthy. Fully half of them were healthy also in 1980, but nearly 2 % of them had become heavily infected. Correspondingly, none of the trees which were heavily infected in 1977 had recovered totally in 1980, although two-fifths were only slightly infected (Table 8).

Table 8. The distribution of the trees, which belonged to the disease classes 1—4 in the year 1977 as to disease class three year later (in 1980).

Disease class in 1977	Disease class in 1980 DC 1	DC 2	DC 3	DC 4	Trees together %	no.
	Trees in the class, %					
1	52.1	42.9	3.2	1.8	100.0	741
2	19.4	57.0	15.5	8.1	100.0	3237
3	2.4	54.6	26.7	16.4	100.0	165
4	0.0	40.6	29.7	29.7	100.0	64

43. Effect of the *Lophodermella* needle cast on elongation and shedding of needles

Current-year needles infected by *L. sulcigena* started to loosen from the shoots over a month from the initial disease symptoms and at the age of only three months (Fig. 12, line 2). The amount of current-year needles decreased evenly through the end of the infection year, when only two thirds (67.9 %) of the needles were in shoots. At the end of July of the year following infection the amount was 44.6 % and at the end of the same year (1979) 16.6 %, most of them diseased. One year earlier, the number of needles over one year old was only 4.3 %, of which the majority were green. This level was reached already at the end of the summer, which was preceded by a rapid decrease in late July—early August in the number of one year old needles infected by *L. sulcigena* (Fig. 12, line 1). This decrease coincided with the time of first visible disease symptoms of current-year needles. When 50 % of the infections of developing needles are judged to have occurred about three-quarters of the one year old needles (with mature ascocarps) were still in shoots.

Of the total needle litter within three years, 35 604 short shoots, 42.1 % consisted of current-year needles affected by *L. sulcigena*. The proportion of *Lophodermella* needles varied annually between 22.1—51.2 %. This variation was similar in both plantations (Table 9). Shed needles which were not affected by *L. sulcigena* were 3—5 year old. Their shedding was considered as the normal fall of oldest senescent needles. The number of shed *Lophodermella* needles was at its highest in 1978 and at its lowest in 1980. The main reason for the decrease may be that the litter funnels were situated in the same places all the time.

During the three years (1978—1980) the needle shedding was concentrated clearly in the summer with two peaks, the former in June and the latter at the end of August. The shedding of the *Lophodermella* and oldest needles coincided in June, but the late summer shedding peak of *Lophodermella* needles was in early August, whereas that of the oldest needles was at the end of August—early September. Variation between years was slight in this respect (Fig. 18). The first current-year needles, infected in June—July, began to shed already at the end of August. Only a minute number of needles fell in winter time. The results for Halkivaha fit in well with those for Kaksoiskivi.

The highest amount of fall of *Lophodermella* needles was 1400 short shoots per square metre in a week. The corresponding amount for normal shedding was 2000 short shoots/m^2/week. The maximum amount of all fallen needles reached the weekly level of 3200 short shoots/m^2 at the end of August 1978 at Halkivaha.

There were 7658 diseased short shoots in the litter funnels in 1978—1979. Of these, 64.7 % had infections in both needles. This double infection evidently hastened premature shedding, for at the beginning of

Table 9. Total needle litter fall at Halkivaha and at Kaksoiskivi in the years 1978—1980.

Cause of shedding	1978		1979		1980		1978—1980	
	Short shoots							
	no.	%	no.	%	no.	%	no.	%
	Halkivaha							
L. sulcigena	3692	49.6	4265	42.8	1760	31.8	9717	42.3
Other	3752	50.4	5697	57.2	3768	68.2	13217	57.7
	Kaksoiskivi							
L. sulcigena	2425	51.2	2231	43.6	625	22.2	5281	41.7
Other	2308	48.8	2887	56.4	2194	77.8	7389	58.3

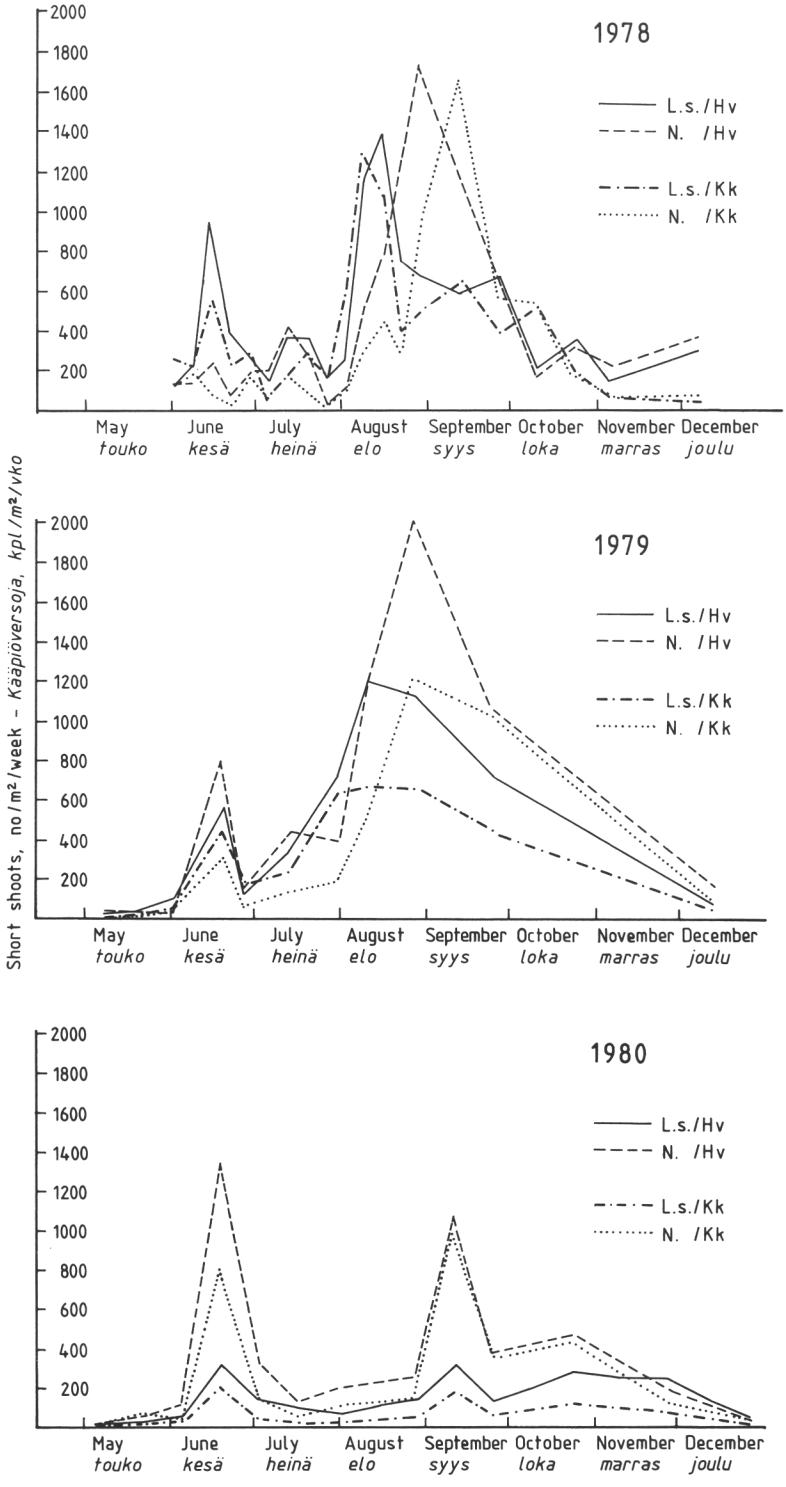

Fig. 18. The distribution of needle fall at Halkivaha (Hv) and at Kaksoiskivi (Kk) in 1977—1980. Explanations: L.s. = short shoots fallen due to the *Lophodermella* needle cast; N. = normal shedding of senescent short shoots.

shedding the proportion of double-infected short shoots was higher than later on.

H. acicola was present on the surfaces of fallen current-year and needles over one year old during the inspection period. However conidia were more common in needles which fell in late summer and autumn than in early and midsummer.

There were no statistically significant differences in needle lengths between differently infected short shoot types at Juotas, where the mean length of fully grown needles was 25.5 mm. The shortest were the short shoots with two diseased needles, only 24.6 mm. In one tree only was the difference in length between needles of its healthy short shoots statistically significant ($p < 0.05$).

44. Effect of the *Lophodermella* needle cast on tree growth

441. Height increment and mean height

When comparing the growths of trees of various DCs in the following, the growth level, if not mentioned separately, always indicates the relation of the growth of trees in the DC to the growth of healthy trees. In Fig. 19 the growth level of the healthy trees is 100 %.

Before the *Lophodermella* epidemic the trees in DC 4 grew in height about 10 % more than the trees in DC 1. However, the difference was not statistically significant. In the first severe year of the epidemic (1978) height increment was also similar in all DCs (Fig. 19 A). Later, the disease had no effect on the height growth of the slightly infected trees. The growth of moderately infected trees started to become retarded in 1980. Their height increment was at the lowest in 1981 (93.2 % of the level of the healthy trees). The difference was not significant.

Only the height growth of the trees in DC 4 was retarded significantly as compared to DC 1. In 1981, when the height growth level in DC 4 was 80.2 %, differences in height increment between DCs were highly significant ($F = 17.9$; d.f. = 3/206), and also significant ($p < 0.05$) between the height increments of the heavily and the slightly or the heavily and the moderately infected trees. The heavily infected trees began to recover quickly, reaching a growth level of 91.4 % in 1982, which was still statistically significantly ($p < 0.01$) less than in DC 1.

As one stipulation was that the sample trees be medium-sized in spring 1978, the mean heights of the trees in various DCs did not differ before, during or after the epidemic significantly. However, the height in DC 4, especially, was visibly greater than that in DC 1 even during the first years of the epidemic (Fig. 20 A). The mean height of the trees in DC 4 started to drop slightly in 1979 and then more in 1980—1982, which resulted in a mean height equal to that of the healthy trees in 1982. The mean height in DC 3 also dropped to the same level; their mean height started to decrease a year later (1980) than that in DC 4.

442. Radial increment and mean diameter

The trees in DCs 2—4 had a greater radial increment than did the trees in DC 1 for nearly every year before the epidemic (Fig. 19 B). The radial increment of the trees in DC 4 differed from the growth of the trees in DC 1 at the significance level of $p < 0.01$ in 1973 and 1976 and $p < 0.001$ in 1975. As a result of the epidemic the radial increment of the trees in DC 2 indicated a small but statistically insignificant reduction in the years 1980—1982.

The radial increment of the trees in DC 3 dropped to the growth level of DC 1 in 1979, the third year of the epidemic. The growth (77.4 %) in 1981 did not differ from the growth of the healthy trees, but one year earlier the trees in DC 3 had thicker increments than the trees in DC 4 ($p < 0.05$). The trees in DC 3 began to recover fairly quickly, reaching a growth of 90.9 % in 1982.

The first signs of retardation in the radial increment of the trees in DC 4 were observed in the second year of the epidemic (1978). A year later the radial increment dropped below 100 %. The lowest level (56.8 %) was reached in 1980, and it was nearly as low (60.9 %) in 1981. A quick recovery began in 1982, when the radial increment was already 87.3 % (Fig. 19 B). Differences between radial increments of various DCs in 1980 were highly significant

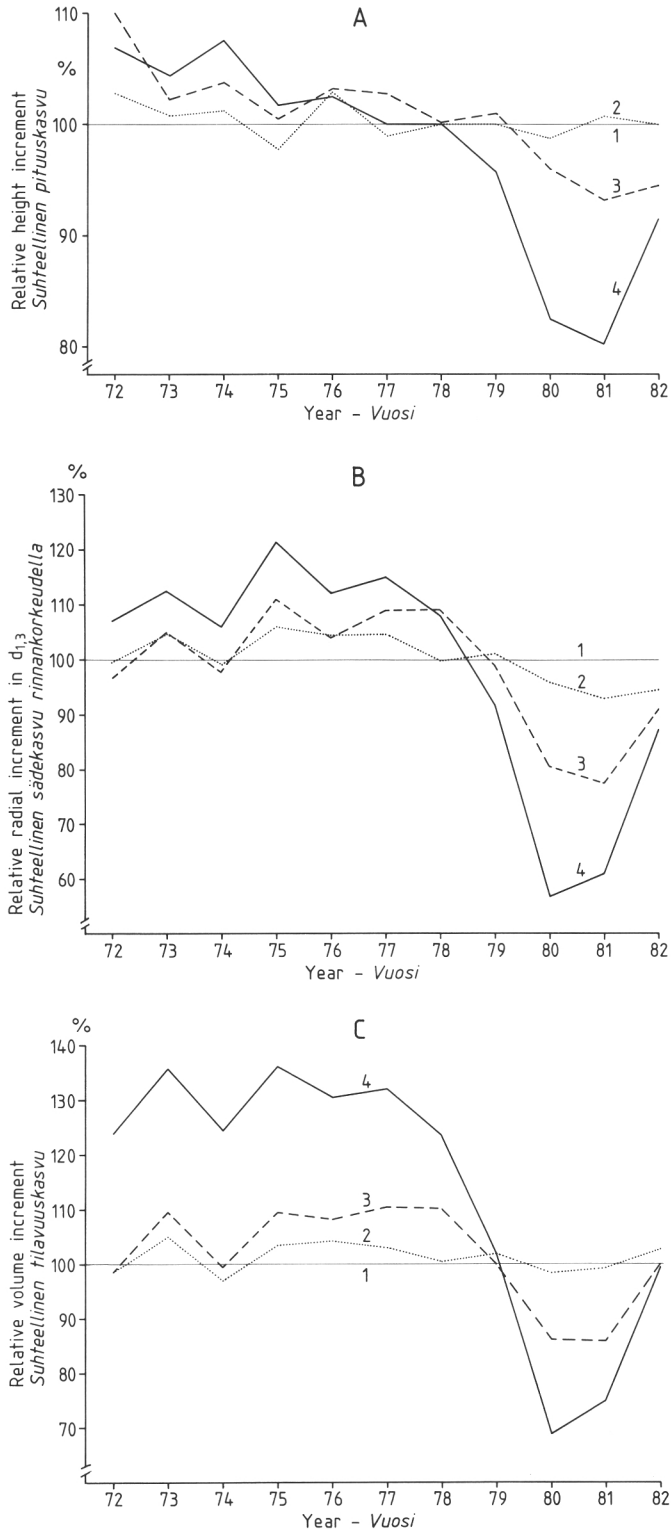

Fig. 19. The effect of *Lophodermella* needle cast on the relative height (A), radial (B) and volume (C) increments of Scots pine in disease classes 1 (healthy trees), 2 (slightly), 3 (moderately) and 4 (heavily infected trees) at Halkivaha. The epidemic began in 1977 and ceased in 1982 after a very low disease incidence in 1981. The reference value of the healthy trees is 100 %.

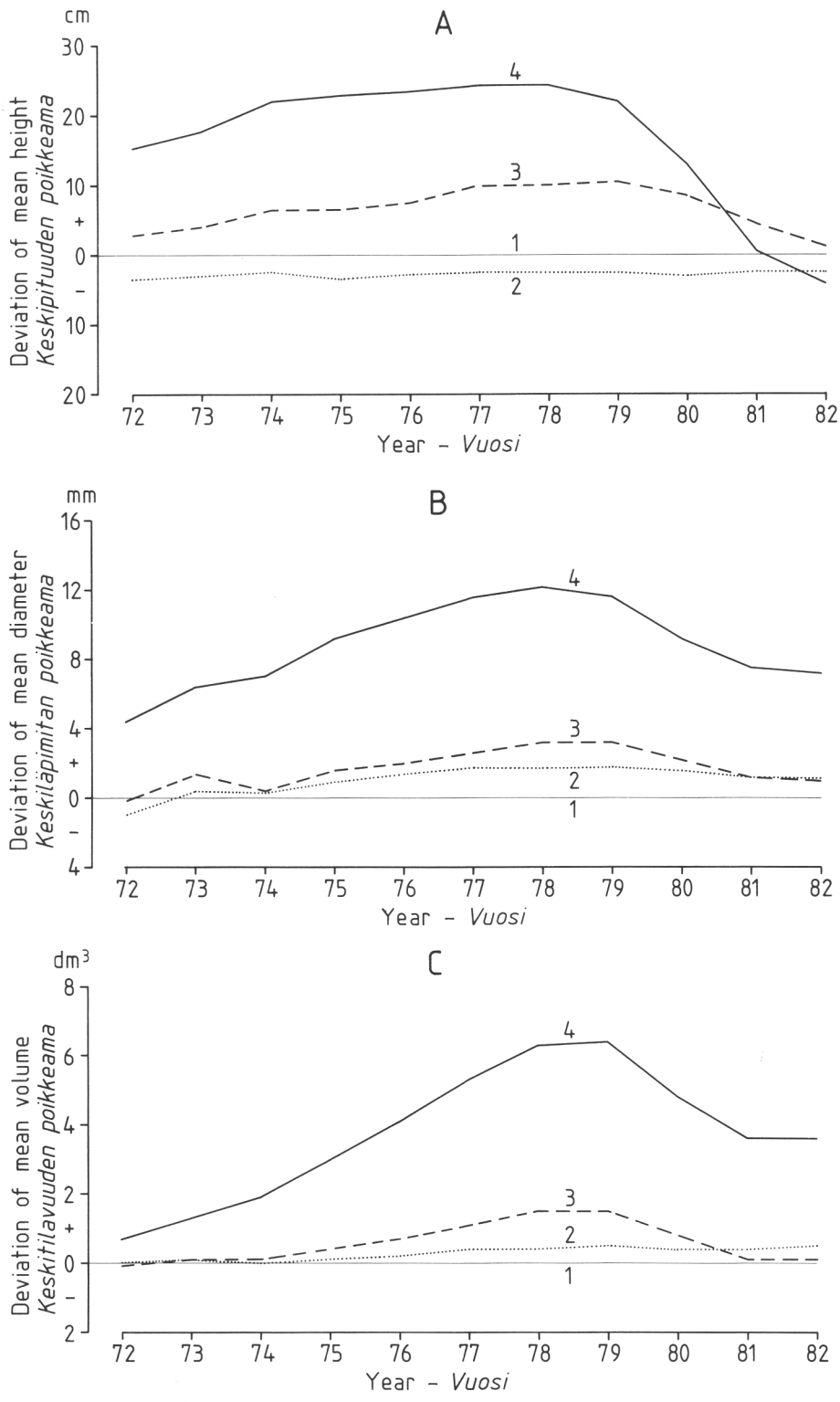

Fig. 20. The effect of *Lophodermella* needle cast on mean height (A), diameter (B) and volume (C) of Scots pine in disease classes 2—4. Reference value is 100 % in disease class 1. Other explanations: see Fig. 19.

($F = 11.5$; d.f. = 3/206). Comparisons of means with Tukey's t-test showed that the radial increment in DC 4 was significantly less than the increment in any other DC at least at $p < 0.05$. The radial increment differences were still highly significant in 1981, but ceased to be so in 1982 due to the rapid recovery.

Even before the epidemic the trees in DCs 2—4 were thicker in breast height than the ones in DC 1. The trees in DC 4 differed statistically significantly from DC 1 in 1973—1976, from DC 2 in 1972 and 1975—1976, and from DC 3 in 1975—1976 (Fig. 20 B). There was no difference between DCs 1 and 2. In the first years of the epidemic the heavily infected trees stayed significantly thicker than the healthy trees in 1977—1978 ($p < 0.001$), remaining so in 1979 ($p < 0.01$), and still in 1980 ($p < 0.1$). No differences were observed in 1981—1982.

Regardless of the disease class, the greatest radial increments before the epidemic in relation to the growth of the trees staying healthy were measured at heights of 0.5—2.0 metres, and the lowest radial increment at a height of 0.1 m. The height of 0.5 m of the trees in DC 2 did not fit in with this. Especially in DCs 3 and 4 the trees had strengthened their stem butts. For instance, the thickness of trees at the height of 0.5 m in DC 4 had increased annually 10—22 % as compared to the trees in DC 1 in 1973—1977 (Fig. 21). The radial increment at the height of 4.0 metres seemed to be nearest to the growth in DC 1. Comparisons at the uppermost heights are not reliable due to the small number of trees which were high enough before the epidemic. The mean height of 6 metres of the sample trees was not reached until the summer of 1979. The mean height of over four metres was measured first in 1976.

After the outbreak of the epidemic the greatest reduction in radial increment of trees in DCs 2—4 in relation to DC 1 took place in the stem butts. As late as 1981, increments of the butts in DC 3 continued to decline. In general, the uppermost heights (4—6 m) did not show retardation as severe as that at the lower heights. The effects of height were most clearly visible in DC 4, where the differences were statistically significant in 1980. The recovery after the epidemic was quickest at the uppermost heights, from 4 metres upwards and slowest at stump height. At the heights of 4 and 6 metres the trees in DC 3, and, at 4 metres, in DC 4 already grew more than the trees in DC 1 in 1982, although the growth level at breast height in DCs 3—4 was only 87.3—90.9 % of the level of DC 1. An exception among the various heights of the trees in DC 2 was the stump height, the radial increment of which in 1982 dropped drastically and was 16 % lower than the radial increment of the healthy trees at stump height.

According to the taper curves the *Lophodermella* epidemic had reduced significant differences of diameters at all heights between the trees in DC 1 and DC 4 (Fig. 22). While the diameters in DC 4 in 1975 were, regardless of the height of measurement, 3.1—4.6 mm greater than those of healthy trees, the differences in diameters had changed greatly in 1982 (−0.3—+3.6 mm) and become insignificant.

443. Volume increment and mean volume

As early as the 1960's, ten years before the epidemic, the trees in DC 4 showed a better volume increment than did the trees in other disease classes. In 1970 the volume increment in DC 4 was 19.0 % greater than the growth of the trees in DC 1 ($p < 0.05$). This pre-epidemical difference varied from 19.0 to 36.2 % in the 70's being significant every year at least at $p < 0.05$ but even at $p < 0.001$ in 1975 and also in 1977, when the epidemic had already begun (Fig. 19 C). The volume increment in DC 3 in 1975—1978 was approximately 10 % above the growth in DC 1, which, however, is not statistically significant. The trees in DC 2 had grown to the same extent or only some percent more than the trees in DC 1.

The *Lophodermella* epidemic did not affect the volume increment in DC 2 significantly. A small retardation and recovery thereafter possibly occurred in 1980—1982. Instead, the volume increment in DC 3 dropped sharply in 1979 and that in DC 4 in 1978 (Fig. 19 C). Due to the growth retardation the volume increment was the same in all DCs in 1979. The lowest volume increment level in DC 4 was measured in 1980 (69.0 %) and in DC 3 one

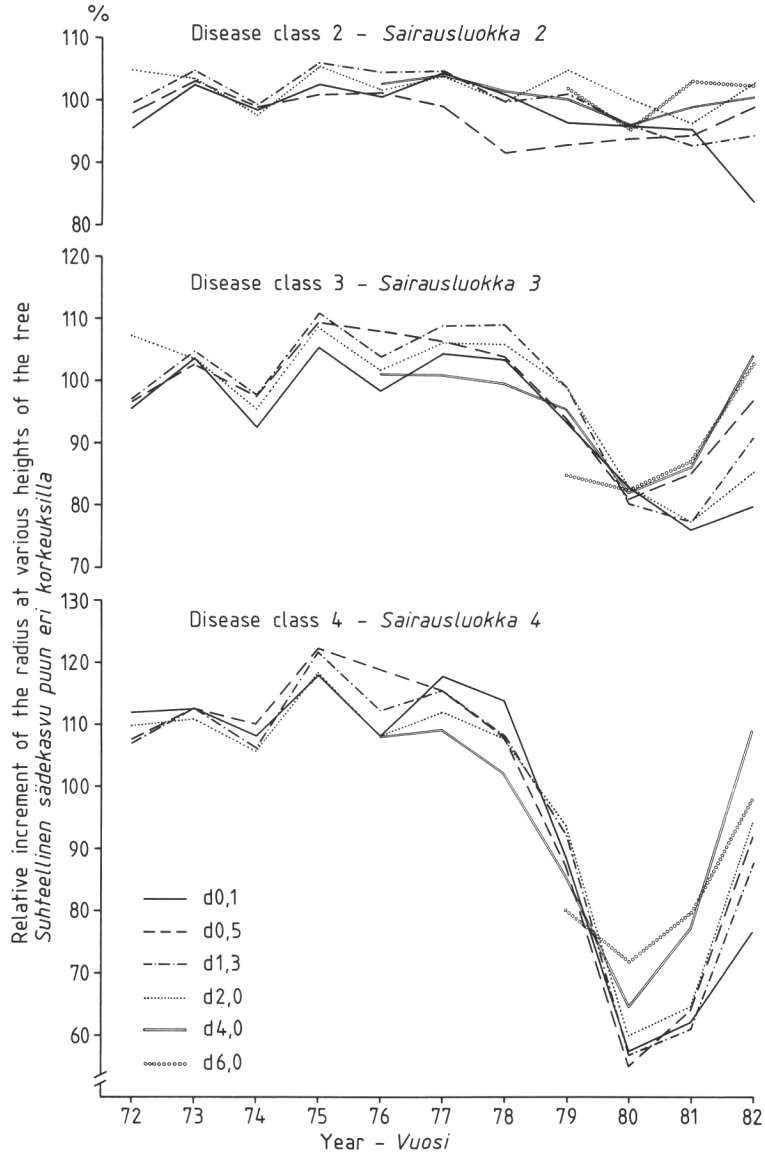

Fig. 21. The effect of the *Lophodermella* needle cast on the relative radial increments of Scots pine at various heights in disease classes 2—4 compared to the growth level of 100 % of the healthy trees. Other explanations: see Fig. 19.

year later. Volume increments between DCs differed statistically significantly only in 1980 ($p < 0.01$). The volume increment in DC 3 did not drop to a level significantly lower than in DC 1. In 1982 the volume increments of all DCs were the same.

The annual volume increment was, at its greatest, about 5.5 dm^3 per tree, which was reached in all DCs in the same year, 1979 (Fig. 23). With the density of 1500 pines per hectare the average annual volume increment was estimated at about 8 m^3/ha. Because of the overdensity the growth in DC 1 also began to decline in 1979.

The greater the mean volumes of the trees in the pre-epidemical period were, the more severely the trees became infected during the epidemic (Fig. 20 C). The first statistically significant differences ($p < 0.05$) in mean volumes between DCs were

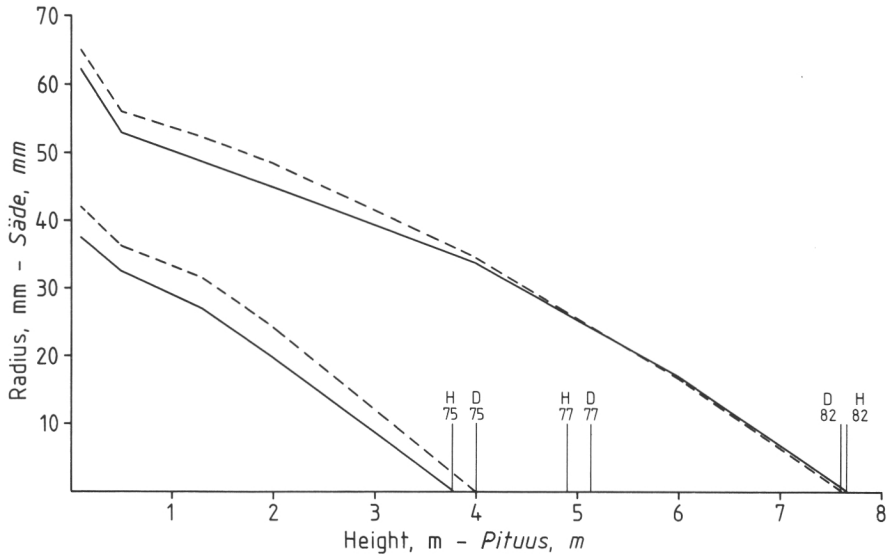

Fig. 22. Taper curves of the healthy (solid line) and the heavily infected (broken line) Scots pines before the *Lophodermella* needle cast epidemic in 1975 and after the cessation of the epidemic in 1982. The epidemic began in 1977. Mean heights of the healthy (H) and the heavily diseased (D) trees have been marked for the years 1975, 1977 and 1982.

measured as early as 1972. In 1978, two years after the beginning of the epidemic, the mean volume in DC 4 was 6.3 dm^3 greater than in DC 1 at $p < 0.01$. Due to the disease the differences in mean volumes of various disease classes were no longer statistically significant in 1980—1982.

444. Growth losses

When a tree was infected by *L. sulcigena*, it had some loss in height, radial and volume growth. The greatest losses occurred in 1980—1981, i.e. clearly towards the last years of the epidemic. The losses of these two years accounted for 43—64 % of all losses in height growth, 59—94 % in radial growth and 67—90 % in volume growth, depending on the DC (Table 10).

The trees in DC 4 lost 42.2 cm (14.5 %) during 5 years in their height growth, 7.4 mm (26.0 %) in their radial growth and 8.8 dm^3 (28.8 %) in their volume growth. The nearly 9 dm^3 volume growth loss was about 1.5-fold compared to the highest annual volume increment. The trees in DC 3 or DC 2 lost significantly less (Table 10). The height increments of 0.50—0.55 m in each

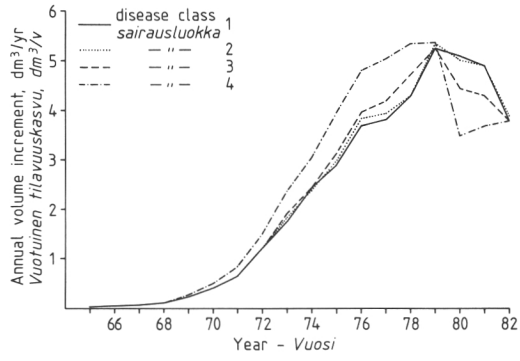

Fig. 23. Annual volume increment curves of Scots pine in disease classes 1—4. Other explanations: see Fig. 19.

class during the epidemic indicated a very fertile forest site. During the lifetime of the Halkivaha stand (20 yrs) the trees in various DCs lost 0.4—5.2 % in height, 1.4—6.6 % in diameter and 0.9—16.4 % in volume (Table 11).

Using the relative height, radial and volume growth loss values presented in Table 10 relative growth losses of the stand caused by *Lophodermella* needle cast were

Table 10. Annual height, radial and volume increment losses of Scots pine in the disease classes 2—4 during the epidemical period at Halkivaha. It was presumed that the *Lophodermella* needle cast did not affect directly the growth of the healthy trees (DC 1).

Disease class	Year 1978	1979	1980	1981	1982	Growth loss absolute	relative
			Height increment loss, cm				
DC 2	0.5	0.5	1.2	0.1	0.6	3.0	1.1
DC 3	2.0	1.7	4.1	6.6	5.3	19.7	6.8
DC 4	2.6	5.1	11.6	15.3	7.6	42.2	14.5
			Radial increment loss, mm				
DC 2	0.11	0.05	0.19	0.22	0.11	0.69	5.2
DC 3	−0.21	0.13	0.63	0.55	0.16	1.25	9.5
DC 4	0.13	0.65	1.53	1.10	0.32	3.73	26.0
			Volume increment loss, dm^3				
DC 2	0.068	0.007	0.177	0.131	−0.026	0.358	1.5
DC 3	−0.231	0.263	0.954	0.936	0.189	2.110	8.6
DC 4	0.269	1.498	3.117	2.718	1.155	8.757	28.8

estimated with equations (5), (6) and (7), for height growth

$$(5) \qquad \text{Loss h} = \frac{1}{100}(1.1n_2 + 6.8n_3 + 14.5n_4),$$

for radial growth

$$(6) \qquad \text{Loss r} = \frac{1}{100}(5.2n_2 + 9.5n_3 + 26.0n_4),$$

and for volume growth

$$(7) \qquad \text{Loss v} = \frac{1}{100}(1.5n_2 + 8.6n_3 + 28.8n_4),$$

where n_2, n_3 and n_4 are the relative amounts of trees in DCs 2, 3 and 4 in the stand.

The equations produced fairly similar results for the Halkivaha stand when using any of the four years 1977—1980. In the following, however, the numbers are based on the mean values of the three intermediate years 1978—1980 of the epidemic (from Table 4). The relative growth losses were also calculated for the 20-year lifetime of the stand by altering the coefficients of equations (5), (6) and (7) according to Table 11. By calculating in this way the Halkivaha plantation had lost, depending on growth type, 2.4—5.9 % of its growth during the epidemic and 0.9—2.2 % of its growth in 20 years (Table 12). Likewise, the height, radial and volume growth loss estimates for the Kaksoiskivi plantation varied between 2.7—6.4 % in the epidemical period and 1.0—2.5 % in 20 years.

Table 11. The heights, diameters and volumes and their losses for 20 years (the age of the Halkivaha stand) of the trees of various disease classes caused by the 5-year-long *Lophodermella* epidemic.

Disease class	Mean of the variable measured in 1982	estimated in 1982	Relative loss %
	Height, m		
DC 1	7.67	7.67	0.0
DC 2	7.64	7.67	0.4
DC 3	7.68	7.88	2.5
DC 4	7.63	8.04	5.2
	Diameter, mm		
DC 1	97.2	97.2	0.0
DC 2	98.4	99.8	1.4
DC 3	98.2	100.6	2.5
DC 4	104.4	111.8	6.6
	Volume, dm^3		
DC 1	41.0	41.0	0.0
DC 2	41.5	41.8	0.9
DC 3	41.1	43.2	4.9
DC 4	44.6	53.3	16.4

Table 12. The estimates for the relative growth losses caused by the *Lophodermella* needle cast in the whole stand of Halkivaha during the epidemical period (1978—1982) and in 20 years.

Growth type	Growth loss, % in 1978—1982 $\bar{x}$	S.E.	in 20 years $\bar{x}$	S.E.
Height growth	2.40	0.10	0.87	0.04
Radial growth	5.86	0.10	1.54	0.04
Volume growth	3.80	0.18	2.19	0.10

5. DISCUSSION

51. Methodological aspects

Although the majority of the experiments were carried out in northern Finland, the assumption that the maturation of ascocarps coincides with the beginning of needle elongation, can be considered applicable in the whole of Finland. The maturation and emptying of ascocarps show that the main sporulation period is restricted to the needle elongation period. Therefore the results of the bagging experiments are slightly confusing in that necrotic needles also appeared in shoots which were predisposed to infection outside of the elongation period and even in periods when ascocarps were immature. Because *L. sulcigena* (Watson and Millar 1971) and generally *Lophodermella* species (Millar 1984) infect growing, semimature needles only, the cause of the necrotism lies in the bagging system. In assessing the disease severity for shoots, determinations were based on macrosymptoms; no isolations were carried out. Thus the primary cause of necrotic needles could have been something other than *L. sulcigena.* According to Wood (1967) a needle infected by a pathogen (*L. sulcigena* in this case) increases its respiration. In the hot and dry air in the bag, the needle can wither (Watson 1971). Signs of exceptional conditions in the bags were indicated by the abnormal shape and form of the shoots and needles, which were bagged for a long time. It is possible that some infections have taken place through the opening of the bag during the elongation period. The bags used in experiments were pollination bags, so it is not possible that ascospores penetrated the bag. The minute number of necrotic needles in shoots free before the elongation period, however, does not vitiate the fact that *L. sulcigena* infects mainly during the first half of the needle elongation as is the case in Scotland, too (Watson 1971). Still, there seems to be a need to develop a safer bagging system.

Keeping the needles in water easily shows whether the ascocarps are mature or not. However, the test does not show whether the asci and ascospores are mature. In any case, the period between the maturation of ascocarps and ascospores is no longer than some days, judging by the beginning of sporulation (see also Campbell 1973, Campbell and Syrop 1975, Minter and Cannon 1984).

The thesis shows for the first time the course of the *Lophodermella* epidemic from beginning to end. The data collection for this, annual disease surveys, is fairly easy and quick. In stand level studies, the whole stand survey gives more than a survey based on sampling. However, when the trees are classified into disease classes, there may be some error between surveys of successive years. Nevertheless, the classification of diseased trees into three classes seems to be suitable, although the smallest correlation between disease classes of successive infections in moderately infected trees revealed a slight tendency to consider a tree either slightly or heavily infected. If more disease classes had been used, it would have made the sampling of growth loss trees more difficult and would have hampered the interpretation of the results of growth measurements and calculations. As there were no differences between healthy and slightly infected trees in growth, however, it would have been sensible to place those trees which had up to 10 percent of current-year needles infected by *L. sulcigena* into disease class 1 (healthy trees). Thus, it obviously might have been possible to distinguish the growth of slightly infected and healthy trees.

It was not possible to show any significant effects of *L. sulcigena* on needle elongation, although the short shoots with two diseased needles were the shortest. The material can be considered large enough. Instead, the material where the infections of the needles have occurred in the beginning of the needle

Table 13. Growth reductions in height (h), diameter (d) and volume (v) caused by *Lophodermella sulcigena* during the epidemical period (1978—1982) in various disease classes by comparing the heights, diameters, and volumes of the years 1977 and 1982; their ratio (R) was calculated with the equation 100 (variable 1982 — variable 1977)/variable 1977.

Disease class	R, %			Difference to DC 1, %-units		
	h	d	v	h	d	v
DC 1	56.5	35.8	133.0	0.0	0.0	0.0
DC 2	56.9	34.1	131.0	+0.3	−1.7	−2.0
DC 3	53.6	32.0	121.0	−2.9	−3.8	−12.0
DC 4	48.2	25.5	94.8	−8.3	−10.3	−38.2

elongation period — as was demonstrated to occur normally — should be used. Then further research with early infections might show the effects.

Due to the differences between growths of trees in various disease classes the method whereby the trees were cut into sections for using the equation of a cone of frustrum (Snellman 1984) can be considered a more reliable though more laborious system when compared to the use of a growth equation based on the height and the radius in breast height of the whole tree only. Differences in reactions between trees at various heights were taken well into account when measuring the trees at many heights. The use of spline functions would have been unreliable in early stages of the stand development, when not enough measuring heights exist (Snellman 1984).

The growth loss calculations were based on the growth ratio, which tells how much the slightly, moderately and heavily infected trees grew as groups in relation to the growth of the healthy trees before the epidemic. The growth of diseased trees which would have occurred had there been no epidemic, was predicted with the help of growth ratios and the growths of healthy trees during the epidemic. Another method could have been the prediction of the growths for each trees based on the development of single trees before the epidemic. On the other hand, testing the differences in volumes of 1970 and 1982 between disease classes (see Harvey 1976, Mitchell et al. 1976a) would not have shown the accurate growth losses in my case, because, as was shown, the more the trees grew before the epidemic, the more their growth was retarded during it, and because the volumes of different disease classes within 1970 and within 1982 were the same. However, if the heights, diameters and volumes of the years 1977 and 1982 are compared, the reductions in height, diameter and volume growth of the heavily infected trees are 8.3, 10.3 and 38.2 percentage units (Table 13). The numbers show a distinct loss especially in volume of heavily infected trees, and resemble those of Mitchell et al. (1976a).

52. Infection period of *L. sulcigena*

L. sulcigena seems to have adapted well to the growth of the needles of its host (*P. sylvestris*) (Fig. 24). *L. sulcigena* is known to infect only current-year needles (e.g. Liro 1924); the infections cannot occur on mature needles (Watson and Millar 1971). With the aid of infection spots and the growth curve of needles, I could confirm the statement of Watson (1971) that infections occur only on a narrow zone just distal to the fascicular sheath on the emerging needle. This seems to be true in all pines parasitized by different *Lophodermella* species (Millar 1984). Further from the base all ascospores remain ungerminated, the reason for which may be changes in the epidermis, cuticle structure and microflora as the needle ages (Campbell 1972).

The sporulation period was 6—8 weeks, which corresponds remarkably to the needle elongation period. This has been reported by Watson (1971) in Scotland, too. Although ascocarps opened in one and half months, the maturation of the first hysterothecia coincided from year to year nearly to the day with the beginning of needle growth. This kind of relationship has been found on *Lophodermella morbida* Staley & Bynum, whose ascocarps mature during the flowering of *Rubus parviflorus* Nutt. (Harvey 1976). These features are, in general, connected with the phenological development, which, in turn, follows the development of the temperature sum (Sarvas 1972). Due to different growing periods and geographical locations, the first ascocarps can mature earliest at the end of May in southern Finland and latest in the first half of July in northern Finland. Maturation,

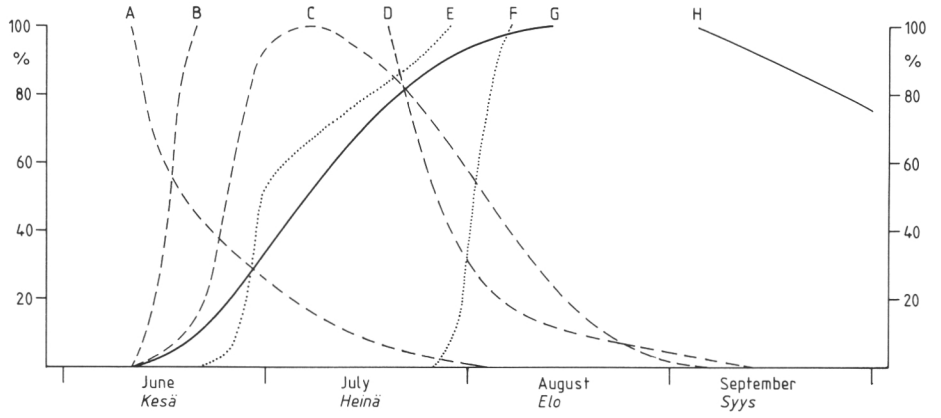

Fig. 24. A schematic presentation of different stages of the *Lophodermella* needle cast during the growth period with average temperature sum in northern Finland. A = amount of immature ascocarps; B = amount of needles with mature ascocarps; C = amount of ascospores in the air (maximum amount = 100 %); D = amount of ascocarps with ascospores; E = the time of infection; F = the time of the appearance of first disease symptoms in needles; G = needle elongation curve, and H = amount of infected needles in shoots.

though equally long, occurs a little earlier in Finland than in Scotland (Watson 1971). This may be due entirely to differences in growth rhythms between pine species. In Italy ascocarps become mature from March to June (Moriondo 1963), and the growth of needles also commences earlier than in Scandinavia, for example.

Mature ascocarps open with a longitudinal slit as soon as they have enough humidity. As generally is known among needle diseases (e.g. *Lophodermella conjuncta* Darker, Kurkela 1978), ascospores of *L. sulcigena* also discharge only under sufficient relative humidity. My results confirm Millar's (1970) observation that only a few first hours of rain suffices for the start of sporulation. Also, the related fungus, *L. morbida*, is quick in sporulation (Harvey 1976). Exceptional as regards earlier works was the lowest RH (79 %) in which ascospores were still trapped. An explanation for this lies possibly in the distance between the meteorological observation box (at the height of two metres) and the spore trap (just above the soil surface).

Sporulation and inoculum potential are maximal soon after the first hysterothecia are mature (Millar 1970). That is why most infections coincide with the first half of needle elongation, which in most cases is the period of the fastest growth of needles.

Elongation periods unfavourable for infections are very rare in Finland. When there is one — such as the summer of 1985 — the disease severity remains low. On the other hand, Jalkanen and Laakso (1986) have shown *Hendersonia acicola* to have an important role in the cessation of the epidemic of *Lophodermella* needle cast. They have stated that climatic factors have a more important role for the beginning than for the cessation of an epidemic lasting many years. It is noteworthy that when a needle's growth slows during the latter half of its elongation, it becomes more resistant. Abundant infection sources in particular have less success among them than among the actively growing needles.

The period between infection and initial disease symptoms seems to be as long (a month) in Finland as it is in Scotland (Watson 1971). However, development from partially to completely necrotic takes a shorter time in Finland than in Scotland, where the colour changes of infected needles did not cease until October. The main colour of autumnal needles infected by *L. sulcigena* seems to be the same in both countries: reddish brown (see Jalkanen 1981, colour plate) or light ochraceous salmon (as described by Watson 1971). There are no differences in needle shedding models between the two countries. Also *L. morbida* causes a high late summer peak in the shedding of one year old needles (Harvey 1976).

When comparing the Scottish results (e.g.

Watson 1971) to the Finnish ones, it is easy to see similarities, for instance, in the timing of appearance of symptoms and the life cycle in general. This may seem confusing because northern Finland represents a slightly continental climate, whereas Scotland belongs to an oceanic zonal section. However, the most heavily infested areas in E and NE Scotland have nearly the same rainfall as Lapland has (500—600 mm/year).

Watson (1971) used fungicides in her infection time studies. Her findings showed that *L. sulcigena* can be controlled with Dithane. According to my results, however, the infection period is rather too long for the economical use of Dithane with good control effects, especially in rainy seasons when newly emerged needle bases always exist.

53. The epidemiology of *L. sulcigena*

Both the Halkivaha and Kaksoiskivi plantations were representative for the *Lophodermella* needle cast epidemic in the years 1976—1984. They were established on fertile soil, which has been demonstrated as increasing the susceptibility of pines to *L. sulcigena* (Jalkanen 1985).

The epidemic doubled the normal amount of needle fall from the heavily infected trees. However, the effect of excess shedding on the growth of trees must have been manifold, as the most effective needles for assimilation were lost. As the disease occurred heavily for 3—4 years, the epidemic left many trees with only a small amount of needles in whole trees (Fig. 25). However, this loss of needles did not lead to the death of pines either primarily or secondarily, contrary to what had been suspected (e.g. Jørstad 1925). The measurements confirmed the results of Mitchell et al. (1976b) that the disease reduces the age of needles by 2—4 years. In northern Finland the needles may fall as many as 6—7 years earlier than normal, which, however, might indicate that the disease has a lesser effect on growth of Scots pine there than in southern Finland.

The needles infected by *L. sulcigena* had two peaks in needle shedding, one in June and the other in August. This fits in well with the results of Watson (1971) in Scotland. Especially at the beginning of the epidemic, the majority of the current-year needles stay in the shoots for over a year so that the pathogen can complete its life cycle and the ascospores can disperse onto new flushing needles. No evidence shows *L. sulcigena* to be able to sporulate and cause infection on the soil surface (see Jalkanen and Laakso 1986). The fall of needles before the maturing of ascospores thus affects the control of the *Lophodermella* needle cast. *H. acicola* has been shown to contribute to the very early shedding of needles (Jalkanen and Laakso 1986). The autumnal shedding peak caused by *L. sulcigena* happened a month earlier than the normal drop of senescent needles. The start of the normal needle fall was in agreement with the results of Mälkönen (1975) in stands 2—3 times older, where, however, the heavy fall of needles continued until as late as October.

The *Lophodermella* epidemic broke out in the thickets of the lowest parts of the stand, where the optimum conditions for needle pathogens most likely exist. The open edges of the stand were less diseased areas, which conflicts with the opinions of many authors (Lagerberg 1910, Terrier 1944, Robak 1963) that the most susceptible trees are located on the egde of the stands or even in clearings. Both on the edge and in the inner parts of the stand cases mentioned by Roll-Hansen (1967), in which there can be a healthy tree beneath a heavily infected tree, were found in abundance. If these two pines grow side by side on the edge of a stand, there may be no differences between the two trees at least in spore deposition or in edaphic or climatic factors. Thus the difference in susceptibility points strongly to genetic characteristics.

It was surprising that the trees below medium size were healthier than the trees of medium size, and, on the other hand, that the disease degree of the trees above medium size was higher. This seems to conflict with the low disease degree of edge trees. However, it is concluded that the relative humidity which favours the pathogen is reached at the stand level only in dense parts, i.e. in general the inner parts of the stand. On the tree level, spores can be deposited more easily on pines above medium size than on trees below medium size; both have a humidity high enough for spore germination and fungus penetration.

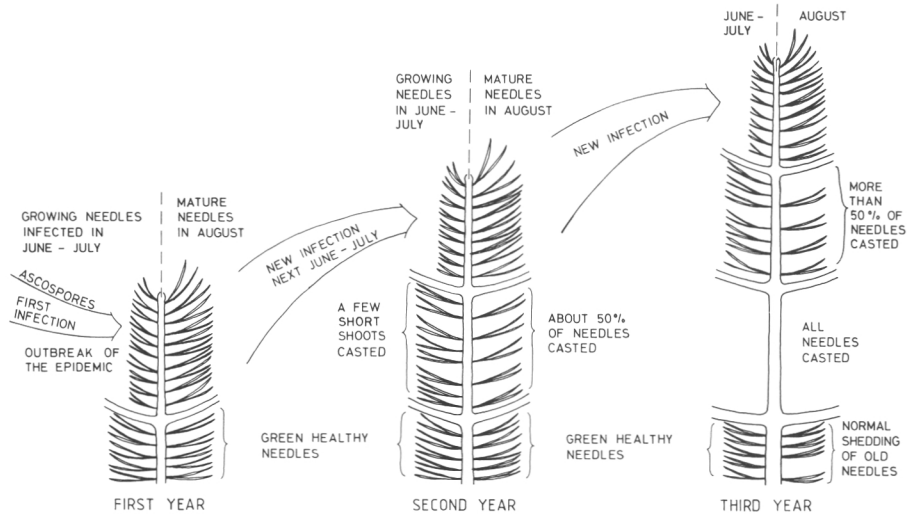

Fig. 25. A schematic presentation of the effect of repeated heavy infection by *Lophodermella sulcigena* on the shedding of short shoots and habitus of the shoots.

The surrounding big trees possibly can very effectively prevent the pathogen from depositing on the needles of small pines. A question arises as to whether differences in the anatomical or biochemical structure of the needles in trees below medium size exist and whether these factors are connected with growth rate.

In normal thinning, the trees below medium size are removed. If this were done in a stand like Halkivaha, it would lead to a higher mean susceptibility of trees to *L. sulcigena*. The goal of the thinning operation ought to be the opposite. Therefore only experimental thinnings can elucidate the effect on the amount of *Lophodermella* needle cast. Another question is that of the best spacing as regards the susceptibility of pines to *L. sulcigena*, or of whether the genetic background of pines has a more important role than the stock density. The evidence is in favor genetic background, because even with a spacing of 5—7 metres some clones are very susceptible (Jalkanen 1982).

It seems that *L. sulcigena* can produce differentially virulent races during an epidemic. This was indicated by the occurrence and spreading of the disease in the stand. The trees around a heavily infected tree were more diseased than the trees of the stand on the average. By the second half of the epidemic the areas of heaviest infections had moved about 50 m up the slope of the stand. *L. sulcigena* proved to be slower in spreading than *Lophodermella arcuata* (Darker) Darker, which has spread about 50 metres per year from its outbreak at the edge of a plantation (Burleigh et al. 1982). When the disease spread upwards at first to less diseased trees and areas, the trees and areas most heavily infected at first started to recover. The reason for this was not examined. Climatic factors can not explain why some trees of the same stand recover and others become more diseased. One considerable factor in the recovery may have been *H. acicola*, which was present on needle litter especially in the autumn. The autumnal occurrence of *H. acicola* has been proven to affect harmfully the fruiting of the pathogen (Jalkanen and Laakso 1986). *H. acicola* appears 2—3 years after the outbreak of the *Lophodermella* epidemic in the stand (Jalkanen 1985).

54. The effects of the epidemic

The *Lophodermella* needle cast had a more distinct effect on radial than height growth. The effect was most evident in leader shoots, which became thinner in the first years of the epidemic. However, especially at the beginning, the leader shoots

had the same length as on healthy trees. However, when considering the effects of the whole epidemic the height growth of heavily infected trees was reduced strongly. This has not been demonstrated for *L. sulcigena* in earlier papers, perhaps due to insufficient material. For instance, Mitchell et al. (1976a) measured differences only in radial growth of Corsican pine. Perhaps there is variation between pine species in response to *L. sulcigena.*

My results are supported by the finding of Burleigh et al. (1982) that both the height and radial growth have been affected by *L. arcuata.*

The radial growth of diseased trees was lowered for years. This fits in well with most reports on both *L. sulcigena* (Mitchell et al. 1976a) and other diseases (Wagener 1959, Christensen and Gibson 1964, Schütt 1964, Kurkela 1981, van der Pas et al. 1984). The high reduction in volume growth of the heavily infected trees even on the annual level compared to that of the healthy trees conflicts with Whyte's (1968) opinion that needle casts do not reduce the annual volume increment.

The growth of the slightly infected trees was the same as that of the healthy trees. This confirms the opinion that there are more new needles in the healthy shoots than a tree needs. According to Oker-Blom and Kellomäki (1983), shading is often greater within trees than between trees. Thus, it would be quite harmless or even a positive thing if 10—20 % of current-year needles were lost through needle cast. The same kind of effect is produced when all current-year needles are lost in only one year (Mitchell et al. 1976a).

The growth potential of the heavily infected trees must have been high. This was supported by their superiority in growth before the epidemic and also in the recovery after the epidemic. Regrettably it was not possible to fell the sample trees some years later to see to what level and how fast the growth of the moderately or the heavily infected trees would have risen after the epidemic. One factor diminishing the growth recovery would have been the overdensity of the stand, the stocking being about 1500 stems/ha at a mean height of 7 metres. It has been estimated that recovery to the pre-epidemical level would take three years (Mitchell et al. 1976a). The present findings lend credence to this supposition.

Although the sample trees for growth calculation were restricted to trees of medium size according to the tree classification made in the beginning of the epidemic, differences in height, radial and volume increments between trees of various disease classes were found during many years before the outbreak of the *Lophodermella* epidemic. This means that the more the trees grow before the epidemic, the heavier they will be diseased at its outbreak. This hypothesis is supported slightly by the results of van der Pas et al. (1984) for *Cyclaneusma minus* (Butin) DiCosmo et al. and more strongly by the measurements of Jalkanen and Kurkela (1984) for *Melampsora pinitorqua* (Braun) Rostr. and Kurkela et al. (1978) for *Heterobasidion annosum* (Fr.) Bref. This was visible in Mitchell's et al. (1976a) height growth data redrawn by Jalkanen (1981). On the other hand, as trees on fertile soils are the most susceptible to *L. sulcigena* (Jalkanen 1985) and as the growth of these trees is rapid, the question arises as to whether any artificial increase in growth makes trees more susceptible. Therefore, great caution should be exercised when introducing new ways to increase the growth of Scots pine. Especially in the case of forest tree breeding the question of resistance should be carefully taken into account. If the bred increase in growth makes pines at the same time more susceptible to pathogens, the wider use of the bred seed might lead to a increase in the amount of disease. It is believed that resistance to *L. sulcigena* can be bred (Terrier 1944, Roll-Hansen 1969); at least the great differences within and between progenies and clones (Jalkanen 1982) indicate this.

Although growth rate affects susceptibility, the basic reason why a healthy tree can exist beneath a heavily diseased tree (Roll-Hansen 1967) is not known. Kurkela and Jalkanen (1981) have demonstrated the differences in susceptibility as due to imbalanced nutrient status. However, more should be understood about the biochemistry of the needle functions and structures, especially in the needle surfaces, because penetration by *L. sulcigena* is suspected as mainly occurring directly through the cuticle (Millar 1981) and partly through stomata (Jalkanen et al. 1981). Some preliminary results on the dependence of disease

severity on buffering capacity, pH and total phenol content of needles have been published (Jalkanen 1983).

55. *L. sulcigena* in forestry

Is there any reason to say that *L. sulcigena* is, at present, commoner than ever in Finland (Jalkanen 1985), and if so, that the disease has become more general in Finnish forests only? Indeed, when checking the neighbouring countries, there are numerous notes and reports on the abundant occurrence of *L. sulcigena* in the late 1970's and early 1980's in Sweden (Karlman 1980, 1984, Martinsson 1982). By contrast, the disease has been of only minor importance in Norway (Roll-Hansen, pers. comm.) and the Soviet Union (Krutov 1979, Krutov, pers. comm.) at the same time. In comparing forestry in these four countries, Finland and Sweden resemble each other most; their common feature has been plantation forestry, which favours Scots pine most of all. The main planted tree species in Norway has been Norway spruce, and the extensive forestry in the Soviet Union has evidently not produced an unusual large number of Scots pine plantations. It has been written that trees on the best forest sites are the most susceptible to *L. sulcigena* (Krutov 1979). Indeed, a remarkable increase in the amount of the disease has been noticed on fresh forest sites (Jalkanen 1985). Such sites are normally planted with pine, and, when better sites and even fields have been cultivated for Scots pine (Selby 1975, 1980), there exist tens of thousands of hectares of Scots pine plantations susceptible to infections by *L. sulcigena* in Finland. That is why only after the introduction of plantation forestry it has been possible to have so many susceptible 10—20-year-old closing plantations on forest sites which naturally are for Norway spruce. This situation may be a significant reason for the generality of the *Lophodermella* needle cast in Sweden, too. According to the known history of the *Lophodermella* needle cast in Finland (Jalkanen 1985), a new epidemic is expected within 10—15 years.

If the *Lophodermella* needle cast is to be avoided or diminished, it will be best to avoid the planting of Scots pine on fertile soils, i.e. on so-called spruce habitats (Jalkanen 1985). It may be possible to develop biological means for controlling an epidemic once it has broken out (Jalkanen and Laakso 1986).

At present, there has been controversy in Finland, as to whether the so-called acid rain has increased forest diseases or not. However, no results indicate that the acid rain has increased the *Lophodermella* needle cast in Finland (cf. Huttunen 1984), but more research is needed in this respect.

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SELOSTE

Männynharmaakariste Suomessa

Harmaakariste on ollut yleinen tauti männyllä Suomessa 1970- ja 1980-luvuilla. Altteimpia taudinaiheuttajalle (*Lophodermella sulcigena* (Rostrup) Höhnel) ovat olleet tasaikäiset, 10—20-vuotiaat viljavalle maalle perustetut mäntytaimikot, jotka ovat vasta sulkeutuneet tai vasta sulkeutumassa (Jalkanen 1985). Harmaakaristeen tiedetään esiintyneen Suomessa noin 10—15 vuoden välein ainakin 1890-luvulta lähtien. Harmaakariste-epidemia on yleensä kestänyt 3—5 vuotta, mutta viimeisin niistä alkoi vuonna 1976 eteläisessä Suomessa ja näytti päättyneen kesällä 1985 Pohjois-Suomessa. Vaikka puut eivät kuole harmaakaristeeseen, pitkäaikainen epidemia heikentää männyn kasvua merkittävästi (Mitchell ym. 1976a). Taudin torjumiseksi pitäisi välttää männyn viljelyä viljaville maille (tuoreille tai sitä paremmille kasvupaikoille), missä männyn luontainen harmaakaristeenkestävyys heikkenee tai jopa katoaa kokonaan (Kurkela ja Jalkanen 1981, Jalkanen 1985). Harmaakaristeen yhteydessä esiintyy luontaisesti sekundaarinen *Hendersonia acicola* Münch & Tub. -sieni, jonka on epäilty vaikeuttavan taudinaiheuttajan lisääntymistä (Mitchell ym. 1976b). *H. acicola* -sienen on sittemmin osoitettu estävän taudinaiheuttajan lisääntymisen, ja harmaakaristeen torjuntakokeet ovat indikoineet mahdollisuutta torjua tautia biologisesti (Jalkanen ja Laakso 1986).

Tämän työn tarkoituksena oli tutkia *L. sulcigena* -sienen elämänkiertoa, männyn saastumista, taudinoireita, harmaakaristeen vaikutuksia männyn neulastoon ja puuston kasvuun sekä taudin epideemistä esiintymistä metsikössä.

Aineisto ja menetelmät

Tutkimusaineisto kerättiin vuosina 1978—1985 Lopen Halkivahasta ja Kaksoiskivestä, Multian Kekälisestä ja Rovaniemen maalaiskunnan Sieväkarista, Kaihuavaaralta ja Juotaksesta (kuva 1). Männyn kasvaimia pussitettiin kasvukauden aikana niin, että uusien, taudille alttiiden neulasten harmaakaristeisuuden perusteella voitiin päätellä pääsaastumisajankohdat. Sama tehtiin myös epäsuorasti käyttämällä hyväksi taudinoireiden sijaintia neulasissa ja neulasten pituuskasvukäyrää (kuva 2). Itiöemien kypsyneisyyttä neulasissa seurattiin vesitestein ja itiöiden vapautumista ilmaan erilaisissa kosteus- ja sadeoloissa itiöpyydyksin. Itiöemien tyhjentyminen koteloitiöistä varmistettiin valomikroskooppisesti. Neulasten karisemista seurattiin joko suoraan oksista laskemalla irronneet kääpiöversot tai karikkeesta sijoittamalla harmaakaristeisten puiden alle yksitoista karikesuppiloa (kuva 3), jolloin voitiin tutkia myös neulasten luontaista karisemista. Se, miten harmaakariste oli vaikuttanut neulasten pituuksiin, selvitettiin kesällä 1985 Juotaksesta kerätystä 1224 kääpiöverson näytteestä.

Harmaakariste-epidemian puhkeamista, kestoa, leviämistä ja pysähtymistä sekä puun kokoon ja sijaintiin liittyviä harmaakaristealttiuteen vaikuttavia tekijöitä tutkittiin Lopella Halkivahan ja Kaksoiskiven metsiköissä. Molemmat pellolle v. 1963 viljellyt reheväkasvuiset metsiköt olivat tyypillisiä vuosina 1976—1984 vallinneen harmaakariste-epidemian aikana sairastuneita taimikoita (Jalkanen 1985). Molempien metsiköiden kaikkien mäntyjen (yhteensä 13 185) harmaakaristeisuus määritettiin silmävaraisesti johonkin neljästä karisteisuusluokasta, jotka olivat: 1) = terveet puut (uusimasta neulaskerrasta saastunut alle yksi prosentti), 2) = lievästi (saastunta 1—33 %), 3) = keskinkertaisesti (34—66 %) ja 4) = ankarasti sairaat puut (taul. 1). Puiden harmaakaristeisuus tarkastettiin Halkivahassa vuosien 1977—1981 ja Kaksoiskivessä vuosien 1977—1978 saastunnan perusteella.

Harmaakaristeen kasvuvaikutusten selvittämiseksi vuosien 1978—1980 tautisuuden perusteella Halkivahan metsiköstä valittiin 210 kasvukoepuuta (5 % metsikön sen hetkisestä runkoluvusta), jotka jakaantuivat suunnilleen tasan edellä mainittuihin harmaakaristeisuusluokkiin (taul. 2). Puut hakattiin syyskesällä 1982, jolloin puiden rinnankorkeusläpimitta ja pituuskasvut vuosilta 1970—82 mitattiin. Paksuuskasvujen selvittämiseksi 0.02 m:n paksuiset näytekiekot sahattiin 0.1, 0.5, 1.3 ja 2.0 metrin sekä sen jälkeen joka parillisen metrin korkeudelta aina latvaan saakka. Lustojen mittauksen jälkeen puut kuutioitiin katkaistuina kartioina (Snellman 1984). Kasvutappiolaskelmat perustuivat ns. kasvusuhdelukuun, joka osoittaa lievästi, keskinkertaisesti ja ankarasti sairastaneiden puiden pituus-, säde- ja tilavuuskasvun suhteen terveiden puiden vastaaviin arvoihin ennen harmaakariste-epidemian alkamista. Kasvusuhdeluvun ja terveiden puiden epidemian aikaisten kasvujen avulla ennustettiin, miten sairaat puut olisivat kasvaneet ilman tautia. Kasvutappiot määritettiin ennustettujen ja todellisten kasvujen perusteella kullekin sairausluokalle erikseen.

Harmaakaristesaastunta

Ensimmäiset taudinaiheuttajan itiöemät kypsyivät lähes päivälleen neulasten pituuskasvun alkaessa. Muutamassa viikossa kaikissa neulasissa oli kypsiä itiöemiä (kuva 5). Itiöitä alkoi vapautua heti itiöemien avauduttua; suurimmat itiötiheydet ilmassa mitattiin lähes kuukauden kuluttua ensimmäisten itiöemien avautumisesta. Itiöinti alkoi 1—3 tunnin kuluttua sateen alkamisesta ja loppui 4—8 tunnin ajan sateesta ja suhteellisen kosteuden laskettua alle 90 %:n. Itiöitä oli ilmassa myös sateettomina aamuyön tunteina (kuva 8). Itiöemät alkoivat olla tyhjentyneitä neulasten pituuskasvun päättymisen aikoihin, kuitenkin viimeistään syyskuussa (kuva 7).

Neulaset saastuivat muutaman viikon kuluttua pituuskasvun alkamisesta ja aina sen ensipuoliskolla (kuvat 9 ja 10) lukuunottamatta kesää 1985. Tuolloin pääasiallinen saastunta Juotaksessa Rovaniemellä ajoittui jaksolle 16.—23. heinäkuuta, jolloin neulaset olivat saavuttaneet jo yli kaksi kolmasosaa lopullisesta pituudestaan (kuvaa 11). Myöhäisen saastumisajankohdan suhteessa neulasten pituuskasvuun arvellaan olevan yksi syy harmaakariste-epidemian laantumiseen kesällä 1985. Saastunta ajoittui pääosin kesäkuuhun Etelä-Suomessa ja heinäkuuhun Pohjois-Suomessa.

Taudin vaikutukset neulastoon

Ensimmäiset tautisymptomit havaittiin noin kuukauden kuluttua neulasten saastumisesta, aikaisimmillaan heinäkuun puolivälissä Etelä-Suomessa ja myöhäisimmillään elokuun puolivälissä Pohjois-Suomessa. Harmaakaristeisen neulaston syysväri oli punaruskea; harmaantumisen on osoitettu (Jalkanen 1985) johtuvan *H. acicola* -sienen läsnäolosta. Neulasten tuhoutumisasteen ja -nopeuden perusteella harmaakaristeiset neulaset jaettiin kolmeen päätyyppiin: 1) täplätyyppi, 2) hitaan tuhoutumisen tyyppi ja 3) nopean tuhoutumisen tyyppi.

Ensimmäiset harmaakaristeiset neulaset irtosivat syyskuussa, vajaan kolmen kuukauden kuluttua saastumisestaan, herkimmin karisivat kääpiöversot, joissa molemmat neulaset olivat sairaita. Pääosin harmaakaristeiset neulaset karisivat kuitenkin kesä- tai elokuussa vuoden kuluttua infektiosta.

Elokuun karisemishuippu ajoittui kuukautta aikaisemmaksi kuin normaali, vanhimpien neulasten putoaminen (kuva 18). Tauti kaksinkertaisti ankarasti sairaiden puiden neulasmenetykset (taul. 9). Vaikka epidemia vaivasi samoja puita useiden vuosien ajan kaljuunnuttaen ne, yhdenkään puun ei todettu kuolleen harmaakaristeen takia.

Harmaakaristeen ei todettu vaikuttaneen neulasten pituuteen. Sen sijaan yhdessä puussa kokonaan terveiden kääpiöversojen pidempi ja lyhyempi neulanen poikkesivat toisistaan. Tulosten perusteella arvellaan kuitenkin, että harmaakaristeen vaikutukset neulasen pituuteen on mahdollista todeta silloin, kun neulaset infektoituvat normaaliin ajankohtaan eli muutaman viikon kuluttua neulasten pituuskasvun alkamisesta.

Harmaakariste-epidemia metsikössä

Harmaakariste-epidemia puhkesi Halkivahassa ja Kaksoiskivessä muutamissa puissa vuonna 1976, jolloin viimeisimmän epidemian on osoitettu alkaneen (Jalkanen 1985). Tauti laajeni lähes kaikkiin puihin molemmissa metsiköissä kesällä 1977 jatkuen vielä ankarampana aina kesään 1981 asti, jolloin metsiköt lähes tervehtyivät (taul. 3 ja 4). Metsiköt olivat terveitä kesällä 1982, mutta uusi lievä epidemia puhkesi kesällä 1983 (Jalkanen 1986).

Epidemia puhkesi taimikon alimpien osien tiheiköissä (kuva 13 A ja 14). Vuosien kuluessa tautipesäkkeet laajenivat, ja uusia pesäkkeitä syntyi metsikön keski- ja ylempiin osiin. Samaan aikaan epidemia alkoi kuitenkin hellittää siellä, missä pahimmat tautipesäkkeet olivat olleet ensimmäisenä vuonna (kuva 13 B—E). Viiden vuoden aikana taudin suhteellinen määrä kasvoi metsikön yläosissa ja väheni sen alaosissa (taul. 5). Sekä Halkivahassa että erityisesti Kaksoiskivessä metsikön reunaalueet olivat keskimääräistä terveempiä; harmaakaristeisuus lisääntyi aina jopa 30 metriin asti pellon reunasta sisälle metsikköön (kuva 15). Metsikön sisällä altteimpia taudille olivat keskimääräistä kookkaammat puut ja kestävimpiä alistetut puut, etenkin jos niillä ei ollut yhtään vapaata oksistoa (kuvat 16 ja 17). Yleisesti voitiin todeta, että ankarasti sairaan puun lähiympäristön puut olivat keskimääräistä sairaampia (taul. 7), vaikka jo pitkään on ollut tunnettua (esim. Roll-Hansen 1967) ja tässäkin työssä havaittiin, että kahdesta vierekkäisestä puusta toinen on täysin terve ja toinen täysin sairas. Syytä tähän ei voitu osoittaa, yleisesti kylläkin todettiin, että peräkkäisten vuosien harmaakaristeisuudet riippuvat selvästi toisistaan.

Harmaakariste ja puuston kasvu

Puut sairastuivat harmaakaristeeseen sitä ankarammin, mitä enemmän ne olivat kasvaneet ennen harmaakariste-epidemian puhkeamista. Toisaalta kasvu taantui sitä merkittävämmin, mitä ankarammin puut olivat sairastuneet. Lievästi sairaiden puiden kasvu ei kuitenkaan poikennut terveiden puiden kasvusta. Ankarasti sairailla puilla vuotuiset pituus-, säde- ja tilavuuskasvut olivat taudin takia pienempiä kuin terveillä puilla huolimatta siitä, että ne olivat ennen epidemiaa kasvaneet tilastollisesti enemmän kuin terveinä säilyvät puut (kuva 19). Ankarasti sairaiden puiden kasvu alkoi taantua epidemian toisena vuonna, keskinkertaisesti sairaiden puiden vuoden kuluttua siitä ja lievästi sairailla todettu taantuma vasta neljäntenä vuonna. Tulosten perusteella on pääteltävissä, että mänty voi vuosittain menettää 10—20 % uusimmista neulasistaan ilman kasvutappioita. Sairaat puut alkoivat toipua epidemian jälkeen sitä nopeammin, mitä harmaakaristeisempia ne olivat olleet epidemian aikana. Kahden vuoden kuluttua ankaran epidemian loppumisesta kaikkien sairausluokkien keskimääräinen kasvu oli sama. Nopeimmin puiden sädekasvu elpyi rungon yläosissa (kuva 21).

Ennen epidemiaa ja sen alkuvuosina todetut eri sairausluokkien väliset tilastolliset erot puiden keskitunnuksissa katosivat epidemian takia (kuva 20). Ankarasti sairaiden puiden runkomuoto kehittyi olennaisesti toisenlaiseksi kuin terveiden puiden (kuva 22).

Suurin osa harmaakaristeen aiheuttamista kasvutappioista muodostui vasta epidemian kahden viimeisen vuoden aikana. Kasvutappiot olivat luonnollisesti sitä suurempia, mitä ankarammin puut olivat sairastuneet. Epidemian takia lievästi harmaakaristeiset puut menettivät pituuskasvustaan 1.1 %, sädekasvustaan 5.2 % ja tilavuuskasvustaan 1.5 %. Vastaavat menetykset olivat 6.8, 9.5 ja 8.6 % keskinkertaisesti sairailla puilla ja 14.5, 26.0 ja 28.8 % ankarasti sairailla puilla (taul. 10). Koko 20-vuotisen elinaikansa pituus-, säde- ja tilavuuskasvusta lievästi sairaat puut menettivät 0.4, 1.4 ja 0.9 %, keskinkertaisesti sairaat puut 2.5, 2.5 ja 4.9 % sekä ankarasti sairaat puut 5.2, 6.6 ja 16.4 % (taul. 11). Edellä esitettyjen kasvutappiolukujen ja metsikön puiden tautiluokkajakauman perusteella muodostetun yhtälön avulla koko Halkivahan metsikön kasvutappioiksi arvioitiin kasvulajista riippuen 2.4—5.9 % epidemian aikana ja 0.9—2.2 prosenttia 20 vuodessa (taul. 12). Kaksoiskiven metsikön kasvutappiot olivat hieman suurempia.

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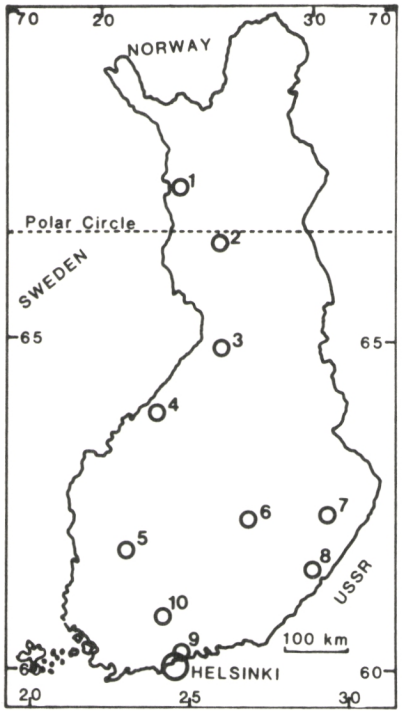

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FACTS ABOUT FINLAND

Total land area: 304 642 km^2 of which 60—70 per cent is forest land.

Mean temperature, °C:	Helsinki	Joensuu	Rovaniemi
January	-6,8	-10,2	-11,0
July	17,1	17,1	15,3
annual	4,4	2,9	0,8
Thermal winter (mean temp. < 0°C):	20.11.—4.4.	5.11.—10.4.	18.10.—21.4.

Most common tree species: Pinus sylvestris, Picea abies, Betula pendula, Betula pubescens

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