BLIGHT OF LUPINUS ARBOREUS IN NEW ZEALAND

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ABSTRACT

Decline of Lupinus arboreus Sims in the Pinus radiata D. Don forests established on New Zealand’s west coast sand dunes was first recognised in late 1988. Lupinus arboreus had played an important dual role in the sand dune forests, providing the shelter necessary for establishment of the young pine trees on the windswept sites and also supplying nitrogen to the ecosystem, thus decreasing the need for applications of artificial nitrogenous fertilisers. Colletotrichum gloeosporioides (Penzig) Penzig & Saccardo, previously unrecorded on L. aboreus in New Zealand, was found to cause rapid mortality of seedlings, and dieback and stem cankering of older plants. A 4-year programme monitoring the effect of the disease on lupin populations has demonstrated that both longevity of plants and production of seed have been reduced since the blight was first recognised. Lupin seed was shown to be a source of infection.

Keywords: Lupinus arboreus; lupin blight; Colletotrichum gloeosporioides.

INTRODUCTION

Stabilisation and afforestation of the shifting sand dunes on the west coast of the North Island was begun early this century with the objective of halting the sand drift and reclaiming the land for pastoral and agricultural purposes. The introduced marram grass (Ammophila arenaria L.) proved to be an effective and quick-growing initial cover on the bare sand. Once sand movement had been curtailed the yellow tree lupin, Lupinus arboreus Sims, was established to aid in the consolidation of the sand. The lupin was highly successful in this role because of its ability to thrive in the difficult environment of a windswept west coast sand dune and its non-palatability to exotic browsing animals. The exotic pine plantations which were established on the dunes initially as part of the stabilisation process proved to be an economically attractive alternative to pastoral farming (Wendelken 1974) and now cover approximately 50 000 ha. The tree lupin played a dual role in the development of these forests—in newly planted areas it was impossible to establish young trees without the shelter provided by the lupin plants, and in its presence nitrogen availability in the soil was increased, thus markedly reducing the requirement for applications of artificial fertiliser to the nitrogen-poor soils. Gadgil (1971c) estimated a nitrogen fixation rate of 160 kg/ha per
year in lupin-marram stands at Woodhill Forest. Exclusion of lupin plants from this ecosystem resulted in lowered productivity of marram grass and reduced growth rates, associated with symptoms of nitrogen deficiency, in *Pinus radiata* (Gadgil 1979).

The lupin population present at establishment of the *P. radiata* stands became suppressed and died out as the tree canopy closed. However, subsequent stand thinning operations stimulated germination of seed produced earlier in the vegetation sequence and a dense understorey of lupin developed, providing a further input of nitrogen to the site. Shade-induced decline of the lupin population set in between the third and fourth year after thinning (Gadgil 1971a,b).

The yellow tree lupin was the ideal plant: it played an important role in the initial control of the shifting sands, its presence reduced the need for applications of artificial fertiliser to the nitrogen-poor soils of the sand dune forests, it was self sustaining—a prolific seed producer and seed could survive in the soil for many years until soil disturbance and appropriate conditions stimulated germination. Because the populations naturally declined and died out under low light conditions (canopy closure) the lupin did not act as a serious competitor to the crop trees. Some releasing was required at pine seedling establishment only. A sudden reduction in the size and vigour of lupin populations in these forests was therefore of considerable concern, both because of the potential for increased wind erosion of the dunes after the decline in vigour of the marram and because of the loss of nitrogen input to the pine plantations.

**DECLINE OF THE LUPIN**

Recognition that there was a serious decline of *L. arboreus* did not occur until late 1988, when staff at Woodhill Forest (on the west coast north of Auckland) became alarmed at the lack of lupin on sites where prolific growth was expected. A survey indicated that lupin populations throughout the forest were in a state of decline and that on the coastal dunes little live lupin could be found. Decaying skeletons of previously large and vigorous plants were all that remained. Forest staff claimed that 12 months previously lupin had been so vigorous on similar nearby sites recently planted with *P. radiata*, that releasing had been necessary.

A nationwide survey undertaken by Forest Health Officers during January–April of 1989 revealed that a similar decline had occurred throughout the country. All sand dune forests were affected, as were coastal areas where lupins had been common. Reports came from Spirits Bay in the far north of the North Island and from Masons Bay on the west coast of Stewart Island. Forest Health Officers estimated that 90–95% of the known lupin in the North Island and 60–65% in the South Island was dead. All surviving plants were reported to show at least some symptoms of disease. The extent of the problem and the degree of breakdown and decay of some of the large woody plants suggested a period of lupin degradation of at least 2–3 years. Another indicator of a period of decline was the marked yellowing of some areas of *P. radiata* in locations which although marginal for soil nitrogen were usually adequately supplied by the lupin understorey (C. Scott, D. Bartram pers. comm.)

P. A. Williams, a correspondent to the June 1988 Newsletter of the New Zealand Botanical Society, reported that lupin had been dying in Golden Bay and Tasman Bay since March of that year and that there was widespread death of lupin from Karamea to Westport and up to
5 miles inland. This was followed by a further comment in the March 1989 issue of the Newsletter that during a January (1988) field trip “dead tree lupins were seen along the coast, both on the coastal sands and further inland amongst rank pasture and marram, from Kawerau to south of the Waipoua River mouth (an 8 km stretch). Five years ago the trip across the half km of semi-stabilised dunes to the two dune impounded lakes on this piece of coast was daunting on account of the 2 m high tree lupin covering the low native scrub. Now there are scattered broken down skeletons of tree lupin, which have the appearance of having been dead for some time (since last summer?). The only living lupins were a few recent seedlings”.

Molloy et al. (1991), who made a study of a stand of tree lupin on the Kaitorete Spit Scientific Reserve (a 27-km-long sand and gravel bar separating Lake Ellesmere from the Pacific Ocean), began their monitoring programme in December 1984. Periodic depredations of the lupin by larvae of the kowhai moth (Uresiphta polygonalis maorialis Felder) were recorded. In May 1986 the population was healthy. At the same time the following year widespread mortality and dieback were noted although the kowhai moth was not present.

Cause of the Lupin Decline

Examination of and isolations from surviving plants from Woodhill and Pouto Forests in late 1988 indicated that the disease was caused by the fungus *Colletotrichum gloeosporioides* (Penzig) Penzig and Saccardo, a species complex containing many morphologically related forms which infect an extremely diverse range of host plants including legumes (Sutton 1980). Considerable variation in conidial morphology and size and in colony characteristics has been recorded for the forms of *C. gloeosporioides*, to the extent that there has been confusion with other species of *Colletotrichum* (particularly *C. acutatum* Simmonds ex Simmonds) depending on the criteria that are accorded most significance by the individual identifier (Sutton 1992). Currently the potential for molecular techniques to aid in species differentiation in the genus *Colletotrichum* is being explored by a number of workers in the field. Sreenivasaprasad et al. (1994) found in one study of 12 isolates of *Colletotrichum* that the nucleotide sequence of the rDNA internally transcribed spacer (ITS) 1 region (180–181 bases) of *C. gloeosporioides* from *Lupinus* sp. was more homologous to that of previously characterised *C. acutatum* than to *C. gloeosporioides*. Although 55 plant species were recorded as hosts of strains of *C. gloeosporioides* in New Zealand (Pennycook 1989) *L. arboreus* was not included. The only records of a species of *Colletotrichum* on *Lupinus* spp. in New Zealand are those of *C. acutatum* and *C. acutatum* Simmonds f.sp. *pinea* Dingley and Gilmour on *L. aboreus*, and *C. acutatum* f.sp. *pinea* on *L. angustifolius* L. (Dingley & Gilmour 1972). These fungi are morphologically quite distinct from the species currently under investigation. The sudden appearance of the disease suggests that the strain of the fungus affecting tree lupin is probably introduced.

A series of pathogenicity tests was carried out after the initial diagnosis. Inoculations of twenty 1-month-old seedlings with a spore suspension of an isolate of the fungus resulted in death of 85% of plants within 21 days and dieback of the remainder, whereas 20 uninoculated plants remained healthy. Further inoculations of 4-month-old plants maintained in growth cabinets under different temperature regimes resulted in no mortality after 2 months. Stem lesions and dieback developed on all inoculated plants which had been artificially wounded (by lightly pricking the stem with a pin) (Table 1).
TABLE 1—Percentage of *Lupinus arboreus* plants developing dieback and stem lesions 2 months after inoculation with *C. gloeosporioides*, or untreated. Five plants per treatment.

<table>
<thead>
<tr>
<th>Temperature regime (°C day/night)</th>
<th>Inoculated wounded</th>
<th>Inoculated no wound</th>
<th>Control wounded</th>
<th>Control no wound</th>
</tr>
</thead>
<tbody>
<tr>
<td>25/19</td>
<td>100</td>
<td>20</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>18/12</td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>12/8</td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Symptoms**

The fungus infects plants of all ages and all above-ground plant parts are susceptible to infection. Infected leaves and petioles wither and much of the foliage is cast. After infection a characteristic crooking of succulent stems and branches occurs which in the early stages is not associated with any visible lesions. Lenticular-shaped lesions up to 3 cm long develop on the branches and stems, particularly at branch axils. Salmon-pink gloeoid spore masses develop on these lesions when the weather is warm and wet. Breakage of branches at the lesion site is common. Death of plants which are infected at the cotyledon stage, or when only the first few leaflets have formed, is generally very rapid. Infection of the hypocotyl either below or above ground results in the formation of a glassy lesion which rapidly spreads and leads to collapse of the young plant. Plants with a woody stem frequently develop distorted cankers with external cracks and internal brown staining at ground level. The fungus also attacks developing seed pods which become either wholly or partly flattened and necrotic with prominent pink spore masses on the surface.

**MONITORING OF LUPIN POPULATIONS**

The potential seriousness of the effects of lupin decline on the nutrition of the sand dune forests and the threat to the stability of the dunes in some locations led to the installation of a programme to monitor the progress of the disease.

**Method**

Plots were established in Woodhill Forest in mid 1989 and in seven other sand dune forests (Table 2) in early 1990 in the stand types in which lupin growth was important, i.e., recently established stands (within 2 years of clearfelling of the previous crop), production-thinned stands (3–12 months after completion of the operation), and on the open dunes. Plots were distributed in a randomised block design with four blocks per stand type and 10 plots (each 1 m²) per block. Lupin numbers were monitored and individual plants categorised (Table 3) for size and disease level.

In mid-late 1991 new plots were established (few lupin plants remained in the original plots) in all but three of the original eight forests plus Taharoa Forest. Plot format and distribution altered at this time—ten 25-m² plots were established in each stand type or on the dunes. The stand types included and the monitoring period for each forest are listed in Table 2. With the exception of Woodhill Forest where all evaluations were carried out by FRI personnel, plot establishment and assessments were carried out by staff from the forests. For a variety of reasons (e.g., changing forest ownership and staff transfers) monitoring of plots
TABLE 2—Location of plots in the 1989–93 *Lupinus arboreus* monitoring programme.

<table>
<thead>
<tr>
<th>Forest</th>
<th>Stand type</th>
<th>Monitoring period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Recently established</td>
<td>Production thinned</td>
</tr>
<tr>
<td>Aupouri Forest</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bottle Lake Forest</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mangawhai Forest</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Pouto Forest</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Santoft Forest</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taharoa Forest</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Waitarere Forest</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Waiuku Forest</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Woodhill Forest</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE 3—Individual plant category and disease score used during the monitoring programme.

<table>
<thead>
<tr>
<th>Size category</th>
<th>Disease category</th>
</tr>
</thead>
<tbody>
<tr>
<td>1  cotyledons only</td>
<td>1   healthy</td>
</tr>
<tr>
<td>2  single stem plant, &lt;1 m in height</td>
<td>2   &lt;25% of plant died back</td>
</tr>
<tr>
<td>3  single stem plant, &gt;1 m in height</td>
<td>3   25–75% dieback</td>
</tr>
<tr>
<td>4  bush, &lt;1 m in height</td>
<td>4   76–99% dieback</td>
</tr>
<tr>
<td>5  bush, &gt;1 m in height</td>
<td>5   dead</td>
</tr>
</tbody>
</table>

Dead plants were removed after the observations had been made. The dieback assessment was made by estimating the percentage of the crown that had been killed by the fungus.

Results and Discussion

Data gathered from one North Island forest and the only South Island forest that was included in the monitoring programme (Bottle Lake) are presented in Fig. 1–10. The most regular assessments over the longest period of time were made at Woodhill Forest, and although plant numbers were extremely variable at the different sites in different forests the patterns of disease progress in the North Island forests were similar. In Fig. 1–10 changes in population structure at the Woodhill Forest and the Bottle Lake Forest sites are presented separately for each 2-year evaluation period because new plots were established in mid 1991. Because of the high mortality of the very young plants and the comparatively small numbers...
surviving to enter the higher size groups, the figures for the three most vigorous growth stages were combined and presented as "large plants". Periods of seed germination were clearly apparent and average disease scores (which included only live plants, i.e., those in disease categories 1–4) tended to be lowest at these times: the younger the population, the higher the proportion of plants recorded as healthy or with only a small amount of dieback.
FIG. 3—Woodhill Forest—production thinned plots. Number of plants per hectare and average disease score for all plants at each assessment.

Average disease scores (at 3-monthly intervals) for each different site at Woodhill Forest over the 4-year assessment period are shown in Fig. 11. Average disease scores for lupin in the thinned stands and on the open dune peaked each year towards the end of summer. The sharp drop recorded at the June assessment each year is due to two factors—ongoing mortality of badly diseased plants which removed them from the assessment and the influx of new seedlings during the late autumn and early winter germination period.
In only two forests (Aupouri and Pouto) apart from Woodhill and Bottle Lake had the same site types been monitored over the whole 4-year assessment period. Total numbers of plants per hectare have been plotted (Fig. 12–15) for these sites from the inception of the monitoring programme in 1989. With the exception of the production-thinned stand at Woodhill, the second group of plots in each type was located in the same compartment or
dune area as the first. These figures should be viewed separately as scales on the X axis are not comparable. Maximum numbers of plants per hectare ranged from 66 000 at Pouto Forest to 1.6 million on the dune at Woodhill Forest. The peak numbers recorded at the beginning of the monitoring programme have not been repeated at any of the locations during the succeeding years.
FIG. 9—Bottle Lake Forest—forest areas. Number of plants per hectare and average disease score for all plants at each assessment.

FIG. 10—Bottle Lake Forest—dune plots. Number of plants per hectare and average disease score for all plants at each assessment.

Data collected prior to June 1991 were analysed using a Box-Cox transformation (Box & Cox 1964) to determine whether there were differences
(a) between the same stand type in different forests,
(b) between different stand types in the same forest,
(c) between assessments at the same sites on different dates.
FIG. 11–Average disease scores of lupin plants in plots on the dune and in thinned *Pinus radiata* stands at Woodhill Forest. Results are from assessments made 3 months apart from April 1989 until March 1993.

FIG. 12–Number of *Lupinus arboreus* plants per hectare in the recently established areas at Bottle Lake Forest. The first monitoring period ran from July 1989 until September 1990. New plots established in September 1991 were monitored until December 1992.

Differences were compared using a Least Significant Difference test. On any particular date highly significant differences occurred between forests and also between stand types within each forest. However, relationships were not consistent over time. There were no consistent relationships with compartment type in different forests, nor within a forest when comparing different sites.

Structure and size of the lupin population can change very rapidly in response to a range of physical and environmental influences. Factors affecting population structure include
insect attack, competition from other herbaceous ground cover, and time of year of silvicultural practices which stimulate seed germination. Population structure at different sites varied considerably at the time of plot establishment. Marked variation occurred even between blocks within a single stand—partly as a result of the recent silvicultural history of the stand. The age and size of lupin plants in a thinned stand varied in relation to the length of time since the thinning operation. Germination of lupin seed, as for many legumes, is governed not only by environmental temperature and moisture but also by disturbance of the soil/sand which results in seed scarification, and mass germination usually follows any silvicultural operation where there has been a previous lupin cover. The movement of cattle into a compartment for grazing (an integral part of weed control in many sand dune forests) also stimulates germination of lupin seed.
Plots established in April 1989 in Compartment 63 of Woodhill Forest, which had been thinned over a period of 10 months finishing approximately 3 weeks previously, had mass germination of *L. arboreus* seed in the most recently thinned area. In those parts of the compartment thinned first (June 1988) there were no seedlings and the few plants surviving all showed signs of disease. Some new plant recruitment was recorded in all parts of the compartment (peaking in July 1989) every month until October 1989. Of a total of 798 plants only 18% remained in October and by June 1990 all of these plants were dead. Similarly, on the open dune 28% of the 503 plants recorded (all as newly germinating seedlings) since June 1989 remained alive in October 1989, 5% in January 1990, and 0.2% (one plant) in June 1990. At Woodhill Forest, all lupin plants died before flowering in all 10 plots established in the recently thinned compartment in June 1991. Two out of 10 plots in a newly-planted *P. radiata* stand and one out of 10 plots on the open dunes were similarly affected.

Because of these factors which affect the size and vigour of local populations, comparisons between sites are of limited value.

**SEED TESTS**

**Viability Tests**

One hundred seeds collected in April 1989 from the sand beneath three dead lupins on a Woodhill sand dune were germinated in the laboratory to check viability. To ensure even and prompt germination the testa of each was pierced with a needle before it was placed on moist filter paper in a closed petri dish. All the seed germinated and 14 days after germination the seedling lupins showed no visible signs of infection by *C. gloeosporioides*. This test was repeated in 1990 with seed collected from three locations (Woodhill Forest, Rotorua, and Pukehina Beach in the Bay of Plenty) and results were identical.

**Tests for *C. gloeosporioides***

Subsequent to the seed collection for the viability test outlined above, the susceptibility of immature seed pods to infection by *C. gloeosporioides* became apparent. Although many
infected seed pods wither and die, some carry a reduced number of viable seeds to maturity. As the potential for seed to carry spores of *C. gloeosporioides* appeared high, mature pods were collected from three locations (Aupouri Forest, Woodhill Forest, and Rotorua) in 1993 to test seed for presence of the fungus.

**Method**

Seed pods from the Rotorua collection were separated into two groups—those with lesions caused by *C. gloeosporioides* (although still producing some mature seed) and those appearing quite healthy. One hundred mature seeds from each of the four groups (Aupouri, Woodhill, Rotorua-healthy, Rotorua-diseased) were selected, surface sterilised in 10% hydrogen peroxide for 5 minutes, plated on to a 3% malt agar medium, and incubated at 25°C. Another 100 seeds per group were extracted individually from the seed pods using forceps sterilised between each transfer, and placed directly on to the agar medium. Plates were examined for development of fungal colonies after 12 days.

**Results**

There was a high degree of variability between the different seed sources (Table 4) which probably reflects quite localised differences in fungal activity at the time of seed formation and maturation. The isolation of *C. gloeosporioides* from the surface of seed extracted from healthy pods from the Rotorua collection indicates that a healthy looking seed pod is no guarantee of freedom from the fungus. Only a small proportion of seed carried the fungus internally—those seeds selected from the diseased Rotorua pods showed 15% infection whereas surface sterilised seed from other collections had 0, 1, and 2% infection.

<table>
<thead>
<tr>
<th>Source</th>
<th>Treatment</th>
<th>Seed bearing <em>C. gloeosporioides</em> (%)</th>
<th>Seed bearing other fungi (%)</th>
<th>Clean seed (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rotorua (healthy pods)</td>
<td>Not treated</td>
<td>19</td>
<td>67</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>Seed surface-sterilised</td>
<td>0</td>
<td>1</td>
<td>99</td>
</tr>
<tr>
<td>Rotorua (diseased pods)</td>
<td>Not treated</td>
<td>50</td>
<td>50</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Seed surface-sterilised</td>
<td>15</td>
<td>5</td>
<td>80</td>
</tr>
<tr>
<td>Woodhill Forest</td>
<td>Not treated</td>
<td>4</td>
<td>96</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Seed surface-sterilised</td>
<td>1</td>
<td>11</td>
<td>88</td>
</tr>
<tr>
<td>Aupouri Forest</td>
<td>Not treated</td>
<td>40</td>
<td>60</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Seed surface-sterilised</td>
<td>2</td>
<td>25</td>
<td>73</td>
</tr>
</tbody>
</table>

**Effect of Seed Pod Infection on Seedling Survival**

Although results from plating of *L. arboreus* seed on artificial growth media indicated that a high proportion of seed could be carrying *C. gloeosporioides*, the effect of the seed-borne fungus on pre- and post-emergence damping-off was unknown. Seed pods were collected from lupin planted in the FRI Longmile experimental area at Rotorua for evaluation of seedling survival.
Dick—Blight of *Lupinus arboreus*

Method

Seed pods were separated into two groups—those which had at least one lesion attributable to *C. gloeosporioides* and those which appeared quite healthy. One hundred and sixty-five seeds from each group were extracted with sterilised forceps, care being taken that no seed came into contact with any other. Many more diseased pods than healthy ones were needed to obtain the required number of seeds from each group—frequently only one or two seeds survived to maturity in the diseased pods. The seedcoat was pierced with a sharp sterile needle, the seeds were placed on moist filter paper and kept at 20°C in the dark until the radical emerged. The developing seedlings were then planted into pots of sand, 20 seedlings/pot. Dying seedlings were regularly removed for isolations to be made from diseased tissues, and survival was recorded at fortnightly intervals.

Results

Some seeds developed fungal growths whilst germinating on the filter paper and were discarded. Survival of seedlings 4 weeks after planting is recorded in Table 5. Subsequent mortality occurred but may have been associated with spread of fungi sporulating on above-ground plant parts.

TABLE 5—Survival of *L. arboreus* 4 weeks after sowing of germinated seed. Seed extracted from diseased and healthy seed pods

<table>
<thead>
<tr>
<th>Source</th>
<th>Initial number of seeds</th>
<th>Number of seeds planted</th>
<th>Number surviving at 4 weeks</th>
<th>Survival of initial number (%)</th>
<th>Survival of number planted (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy pods</td>
<td>165</td>
<td>140</td>
<td>80</td>
<td>50a</td>
<td>59a</td>
</tr>
<tr>
<td>Diseased pods</td>
<td>165</td>
<td>109</td>
<td>44</td>
<td>27b</td>
<td>40b</td>
</tr>
</tbody>
</table>

Differences in survival between seed sources were evaluated using a $X^2$ test, and are given at the 1% level of confidence.

The site of initial tissue collapse of emergent seedlings varied—cotyledons, hypocotyl, or the radical at the soil/air interface. *Colletotrichum gloeosporioides* was isolated from 49% of dying seedlings, *Fusarium* spp. from 15%, and primarily a suite of bacteria from the other 36%. Although there was a highly significant difference in survival between seedlings from the two sources, there was no significant difference in the proportions of dying plants from which *C. gloeosporioides* was isolated. No sporulation had been observed on plants in the first 4 weeks after planting (death of the young plants rapidly followed the first visible signs of infection) and so above-ground cross-infection was unlikely.

There is a clear disparity between the results obtained in 1989 and again in 1990 when germinating seed was free of fungal infection and those from the 1993 test. Seed collected in 1989 and again in 1990 was probably deposited in the sand in pre-blight years and had not been in contact with the fungus to the same extent as seed collected in 1993 had.

DISCUSSION

The origin of the fungus affecting tree lupins in New Zealand is not known. A review of world literature revealed that the only records of a member of the *C. gloeosporioides*
complex on *L. arboreus* were from the USA where *C. gloeosporioides* f. sp. *aeschnymone* has been utilised commercially as the mycoherbicide “Collego” to control the leguminous weed *Aeschynomene virginica* Link in rice and soybean fields. In one test examining the host range of the fungus, 82 species representing 48 genera within the Leguminosae were inoculated (at the two-leaf stage) with spore suspensions (Weidemann et al. 1988). Only the target weed, *A. virginica*, and *Lupinus arboreus* were rated as highly susceptible. The relationship between this fungus and the strain of *C. gloeosporioides* affecting *L. arboreus* in New Zealand has not yet been examined but could be worthy of study.

Although there is no certainty that the demise of tree lupin noted by correspondents to the New Zealand Botanical Society Newsletter (June 1988 and March 1989) and in 1987 by Molloy et al. (1991) was caused by or contributed to by *C. gloeosporioides*, the fungus does offer a possible explanation for what was clearly an out of the ordinary decline. At the time of the first nationwide survey in early 1989 lupin populations were affected throughout the country and no single area could be identified as a disease centre. In light of the subsequent partial revival of lupin populations, and anecdotal reports of lupin vigour in 1988 from some locations, it seems likely that 1987, 1988, and 1989 were the years of primary impact and spread of the disease.

Separating the impact of the disease from other factors influencing the health and vigour of the lupin has not always been possible. *Lupinus arboreus* has a long history of periodic defoliation by larvae of the kowhai moth. Repeated defoliations during a single summer occur at some locations in some seasons and this may result in widespread mortality. Larvae show a marked preference for plants that are more than 1 year old, though they will feed on younger plants—especially when the food source becomes depleted (Gadgil 1971c; M.Kay pers. comm.). Although larval populations appeared to be low in 1989 and 1990 (observation only, no data were collected), in subsequent years numbers built up and the impact of the kowhai moth defoliation was additional to that of the disease. The decline in moth populations may have been the result of the reduced food source during the initial period of disease-induced lupin mortality.

Mortality of young plants (less than about 10 cm high) sometimes occurs in spring/summer as a result of infection by the fungus *Pleiochaeta setosa* (Kirchner) Hughes. Lesions on the succulent stems may spread and coalesce, leading to total plant collapse. On older plants the discrete brown-black lesions on the foliage, pods, and green stems expand and coalesce under conditions of high moisture and warm temperatures, resulting in foliage loss and dieback. Other pests and diseases cause lesser amounts of damage from time to time. Snails may feed on the foliage and weevils (*Cecyropa* spp.) have been known to attack young plants at ground level (R.L.Gadgil, M.Kay pers. comm.). Larval populations of the omnivorous *Heliothis armigera conferta* Walker occasionally cause defoliation. Some cotyledon mortality is undoubtedly due to competition—a tight mat of plants develops under seed-producing plants (live or dead) after events which stimulate germination. Damping-off fungi (*Fusarium* spp. and Pythiaceous fungi) have been isolated from dying cotyledons and seedlings. The effect of the lupin blight is thus overlaid on numerous other influences.

Observations have been made by many interested persons that south of latitude 38° (approx.) individual plants or very small groups of plants can be found throughout the year with little or no evidence of disease. However, in the sand dune forests of the North Island where extensive areas were expected to have a cover of healthy lupin at certain stages of the
forest rotation the picture is different. To date, the lupin cover, although often appearing healthy early in each summer season, starts to decline and become patchy as the season progresses. No data exist to allow direct comparisons of the size of lupin populations and the vigour and longevity of plants in pre- and post-blight years although there is a considerable body of qualitative information published.

The life span of tree lupin overseas has been variously reported as up to 5–6 years in an Irish afforestation programme (O’Carrol 1982), up to 7 years in California (Davidson & Barbour 1977) and Great Britain (Palaniappan et al. 1979), with one record of plants up to 11 years old (Dancer et al. 1977). In New Zealand Gadgil (1971a) suggested that plants in Woodhill Forest did not survive for more than 5 years. In the absence of pests and diseases, decline of the lupin in newly planted stands generally sets in between the third and fourth year after planting, and similarly in thinned stands. This shade-induced decline is thought to be related not only to the direct effect of light conditions which are suboptimal for a plant which naturally grows in open conditions, but also to increased competition from other understorey communities as shading increases. In open areas lacking competition, a longer lifespan could be anticipated. Sandberg (unpubl. data) examined a lupin population in Rotoehu Forest over a 2-year period (September 1982–October 1984) where no plants were more than 5 years old and 79% were less than 3 years old. During the 4-year monitoring period at Woodhill Forest no plant in the plots survived for more than 2.5 years, few survived for 2 years, and no plant set seed more than once.

Results from the monitoring programme indicate that the lifespan of *L. arboreus* in the northern sand-dune forests and on the dunes is up to 3 years shorter than that expected in pre-blight years. Seed production is reduced not only because many plants do not survive to seed-bearing age but also because infection of flowerheads and green seed pods on surviving plants is common. *Lupinus arboreus* will continue to have a place in the ecosystem of the sand dune forests but its productivity can no longer be relied on for provision of a large part of the nitrogen requirements.

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REFERENCES


