

Control Measures for Ergot in the Paspalum (*Paspalum dilatatum* Poir.) Seed Crop¹

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ABSTRACT

A number of methods for controlling ergot (*Claviceps paspali* Stev. and Hall) infection of paspalum (*Paspalum dilatatum* Poir.) were investigated. Partial field control of sclerotial germination was achieved through application of triadimefon (1.8 kg a.i. ha⁻¹) and sodium azide (12.5 kg a.i. ha⁻¹) to the soil surface prior to anthesis and by burning of stubble and trash after harvest. Although control of primary ascospore inoculum was demonstrated, ergot infection of harvested seed still occurred as a result of secondary conidial infection from outside sources.

Some recommendations for reducing ergot contamination of paspalum seed crops are made.

Additional index words: sclerotia, clavae, conidia, burning, triadimefon, sodium azide, soil incorporation.

INTRODUCTION

Claviceps paspali Stev. and Hall, the ergot of *Paspalum* spp. is widespread throughout the North Island of New Zealand (Hopkirk, 1936) and the northwest South Island on *Paspalum dilatatum* Poir. Paspalum is present as a major species in large areas of grasslands in the Auckland province and as a minor species through much of the remainder of the North Island and north-west South Island (Percival, 1977), its importance as a pasture component being its summer production ability (Lambert, 1967).

Paspalum seed production in New Zealand has been complicated by ergot infection which has delayed the release of seed of the improved cultivar "Grasslands Raki" paspalum (Lancashire, et al., 1980). Similar problems with seed production have also been reported from the U.S.A. (Wells, et al., 1958; Luttrell, 1977), and Australia (Lancashire et al., 1980). As sclerotia are the structure of survival for ergot fungi and the source of primary inoculum for infection of grasses (Cunfer and Seckinger, 1977), control measures should be aimed at removal of ascospore inoculum through destruction of the sclerotia (Hardison, 1977). This paper reports investigations of various measures

for control of *C. paspali*. A brief report of results from the 1978/79 season has been previously published (Lancashire et al., 1980).

MATERIALS AND METHODS

A stand of *Paspalum dilatatum*, set out in 1974 as spaced plants on a 0.6 m grid in a Kairanga sandy loam soil near Palmerston North, New Zealand was used as a trial site for field work, and as a source of *C. paspali* sclerotia.

Laboratory

The effects of fungicides and nitrogen on sclerotial germination were investigated, following the work of Hardison (1975, 1977) on *C. purpurea* (Fr.) Tul., Nuzum (1976) on *C. phalaridis* Walker, and Hampton and Scott (1980) on *Gloeotinia temulenta* (Prill. and Del.) Wilson, Noble and Gray. Sclerotia which had overwintered in the field were collected (4 October 1976) by washing them out of soil clumps, drying, air blowing to remove trash, and sorting from pure seed over a diaphanoscope. Sclerotia were then placed on 1 cm of damp tamped perlite in a 15 cm petri dish, 100 sclerotia per dish, and the fungicide and nitrogen treatments (Table 1) sprayed on by means of an atomizer. Four replicates of each treatment were set up, with two replicates per treatment being covered (petri dish lid on) and two uncovered (petri dish lid off and dish in a perspex tank) (Hampton and Scott, 1980). All dishes were placed under N.U.V. light with a 12 hour photoperiod at 20 ± 2°C, and the number of sclerotia bearing clavae counted at 18, 31 and 40 days.

Field

Control of sclerotial germination.
1978/79

The paspalum block of approximately 0.3 ha was divided into two after the seed harvest in March 1978, and the accumulated dead plant material on the ground surface in one half was burnt. Crop debris was left on the ground in the other half of the area. In November of 1978, the two areas were divided into plots 15 × 4 m, and treatments (benomyl 3.3 kg a.i. ha⁻¹, triadimefon 1.8 kg a.i. ha⁻¹, urea 112 kg ha⁻¹, sulphate of ammonia, 112 kg ha⁻¹) were applied by knapsack sprayer to the soil surface on 22 November 1978. These treatments were each applied to six plots per block, three of which were rotary hoed to a depth of 6-8 cm after the chemical application. Each block had two check

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Table 1. The effect of laboratory chemical treatment on the viability of sclerotia of *C. paspali*.

Treatment mg product/dish	Percentage of sclerotia bearing clavae after forty days incubation at 20C			
	dish covered ¹	dish uncovered ¹	treatment mean	
nil	-	15.0	17.0	16.0
benomyl	5 mg ²	0.5	4.0	2.3
benomyl	10 mg	0.5	0.5	0.5
triadimefon	1.7 mg ³	0.0	0.0	0.0
triadimefon	3.4 mg	0.0	0.0	0.0
urea	8.6 mg ⁴	9.5	17.0	13.3
urea	17.3 mg	7.0	13.5	10.3
ammonium sulphate	8.6 mg	15.0	12.5	13.8
ammonium sulphate	17.3 mg	8.0	10.0	9.0
L.S.D. 0.01 ⁵				3.1

¹mean of 2 replicates. No significant differences between covered and uncovered dishes.

²equivalent to 1.7 kg a.i. ha⁻¹

³equivalent to 0.3 kg a.i. ha⁻¹

⁴equivalent to 5.6 kg a.i. ha⁻¹

⁵L.S.D. only on treatments with clavae. All analyses on arc sine transformed data.

plots in the north-west corner, one of which was rotary hoed. On 3 December 1978, approximately 1 kg of soil was collected from each plot. Sclerotia were obtained as previously described, and the effects of the treatments on sclerotial germination were assessed.

1979/80

Sodium azide (12.5 kg a.i. ha⁻¹) and triadimefon (1.8 kg a.i. ha⁻¹) were applied to the soil surface of 3 m² plots, the former on 5 December and 21 December 1979, and the latter on 5 December 1979. Chemical treatments were replicated four times in a randomized block design. The remainder of the 0.3 ha block was treated with triadimefon (0.6 kg a.i. ha⁻¹) on December 1979, with the exception of one 3 m² plot on the north-western corner of the block which served as a check. Within each plot, an area of 0.5 m² was marked out and the number of clavae within the area assessed weekly from 21 December 1979 to 25 January 1980. At seed harvest (30 January 1980), heads from four 0.1 m² quadrats in each plot were cut and hand threshed by rubbing the seed from the head. Seed samples were then purity tested (ISTA, 1976).

RESULTS

Laboratory

The first clavae appeared ten days after the experiment was set up. Both fungicide treatments and the higher rates of nitrogen significantly reduced the numbers of clavae formed (Table 1), with triadimefon giving complete control. No significant differences in clavae numbers were recorded between sclerotia in restricted and non-restricted air spaces.

Field

Fewer viable sclerotia were recovered from plots which had been burnt the previous autumn (Table 2), although the differences recorded could not be statistically analysed because there were no treatment replicates. However, there were no significant differences between plots which had been rotary hoed and those which had not (Table 2). In the non-burnt plots, triadimefon at 1.8 kg a.i. ha⁻¹ was the only treatment to significantly reduce the number of viable sclerotia recovered (Table 3). However, there were no differences in the ergot level in harvested seed (Lancashire et al., 1980).

In the following season both triadimefon and sodium azide at both application times significantly reduced the number of clavae found per unit area (Table 4), but at harvest, differences in the ergot level in harvested seed, although lower for sodium azide, were not significant.

Table 2. The effect of post harvest burning and spring rotary hoeing on viability of sclerotia recovered from the soil.

Treatment	Percentage of sclerotia bearing clavae after forty days incubation at 20C		
	Burnt ¹	Not Burnt	Mean
Rotary Hoed ²	0.3	4.2	2.3
Not Rotary Hoed	0.5	5.3	2.9
Mean	0.4	4.8	

L.S.D. (rotary hoe) 0.01 = 3.9

¹burnt 18 March 1978

²rotary hoed 22 November 1978

³sclerotia collected 3 December 1978

Table 3. The effect of chemicals applied to the soil surface on the viability of sclerotia recovered from the soil in non-burnt plots.

Treatment ³	Percent sclerotia ¹ bearing clavae after 40 days incubation at 20 C ²
nil	5.3
benomyl 3.3 kg a.i. ha ⁻¹	3.7
triadimefon 1.8 kg a.i. ha ⁻¹	0.2
urea 112 kg ha ⁻¹	3.6
ammonium sulphate 112 kg ha ⁻¹	3.3
L.S.D. 0.05	4.6

¹sclerotia collected 3 December 1978

²data means for rotary hoed and non rotary hoed plots

³applied 22 November 1978

DISCUSSION

In the laboratory, triadimefon completely prevented *C. paspali* clavae formation, while benomyl significantly reduced the number of clavae formed. However, in the field, only triadimefon spring applied as a ground-cover

Table 4. The effect of application of triadimefon and sodium azide to the soil surface on sclerotial germination and ergot infection of harvested seed 1979/80.

Treatment	No. of clavae 0.5 m ² removed between 21.12.79 and 25.1.80	% ergot in harvested seed
nil	32.0	24.5
triadimefon ¹ 1.8 kg a.i. ha ⁻¹	8.1	24.7
sodium azide ¹ 12.5 kg a.i. ha ⁻¹	3.2	17.9
sodium azide ² 12.5 kg a.i. ha ⁻¹	1.1	14.8
L.S.D. 0.05	7.5	11.6

¹applied to the soil surface 5 December 1979

²applied to the soil surface 21 December 1979

fungicide, significantly reduced the number of clavae produced, although control was not complete in either 1978/79 or 1979/80. Sodium azide applied four and two weeks prior to peak anthesis of *P. dilatatum* also significantly reduced the number of clavae recorded in the crop, but again, control was not complete. Although the higher rates of nitrogen reduced the number of clavae formed in the laboratory, the field results were consistent with those reported for *G. temulenta* (Hampton and Scott, 1981), where urea failed to prevent apothecial germination.

It was not possible, using replicated plot trials, to obtain either ergot free or low ergot levels in harvested seed at Palmerston North, because of the spread of inoculum from (i) untreated check plots where they were included, (ii) from plots where treatments (fungicide, burning or rotary hoeing) did not completely prevent clavae formation, (iii) from wind-borne inoculum from outside the trial area. In the 1977/78 season, when triadimefon applied in early November appeared to have prevented clavae formation the honeydew phase of the disease was observed on the western boundary of the crop (1 February 1978) and over the next ten days spread towards the center of the crop so that up to 10% of heads were infected. Hampton (1977) showed that fungicide application at early and mid anthesis to prevent secondary conidial infection significantly reduced the amount of ergot in harvested seed compared with untreated checks (Table 5), but did not control the pathogen. In 1977/78 and 1978/79, a foliar spray with triadimefon at early and mid anthesis checked the spread of *C. paspali*, but did not totally halt it.

Seasonal effects on the incidence of ergot were observed, in that less ergot was present in seed in a dry season than in a season where moisture was not limiting (Lancashire et al., 1980). Luttrell (1977) suggested that environmental effects on *C. paspali* were most significant with primary inoculum, as exposure to low temperatures is required to break dormancy in sclerotia and adequate moisture for germination of sclerotia and production of ascospores is required for initiation of the primary disease cycle. Conidia, however, can infect the head during and after anthesis under dry conditions, presumably because of the protected micro-environment within the floret.

Non-chemical control measures were not successful at this site. Although stubble burning after harvest reduced the number of viable sclerotia per unit area, ergot infection of harvested seed did not differ significantly because of inoculum movement from the untreated plots (Lancashire et al., 1980). However, Wells, et al., (1958) successfully used stubble burning to inactivate sclerotia of *C. paspali*, and recorded a 29% increase in seed yield associated with a 33% decrease in ergot contamination. The importance of burning to destroy sclerotia of *C. purpurea* has also long been recognized (Hardison, 1976).

Bretag (1978) showed that when *C. purpurea* sclerotia were buried at a depth of 15 cm beneath the soil surface, less than 3% were able to germinate, compared with 66% of sclerotia left on the soil surface for the same period of time. He recommended deep plowing of infested areas as a control measure. The paspalum block at Palmerston North was rotary hoed to place sclerotia beneath the soil surface, but a depth of only 6-8 cm could be achieved, and results showed no differences between hoed and non-hoed plots. In an established crop, it would be difficult to place sclerotia deep enough under the soil surface so that germination was prevented. However, rotary hoeing in spring between spaced plants, apart from aiding weed control, may have a role in helping to incorporate chemicals into the soil.

Although this work did not successfully provide a complete control program for *C. paspali*, a number of recommendations can be made for paspalum seed production.

Table 5. The effect of fungicide application at anthesis on ergot contamination of harvested paspalum seed, 1976/77¹

Treatment	percent ergot	percent pure seed
nil	46.8	27.0
triadimefon ² (early anthesis)	44.9	28.5
triadimefon ² (early and mid anthesis)	40.9	31.4
benomyl ³ (early and mid anthesis)	38.4	35.5
L.S.D. 0.05	4.4	4.3

¹Data from Hampton (1977)

²0.6 kg a.i. ha⁻¹

³3.3 kg a.i. ha⁻¹

A. Established stands

1. Burn off all trash left after seed harvest.
2. Apply a ground cover fungicide spray* (e.g. triadimefon at 1.5-2.5 kg a.i. ha⁻¹) in spring (November-early December) and incorporate the fungicide into the soil.
3. Monitor the crop during anthesis and spray with triadimefon (1.5-2.5 kg a.i. ha⁻¹) or benomyl (3.3 kg a.i. ha⁻¹) at the first sign of the honeydew phase.
4. Eliminate volunteer plants adjacent to the seed production area.

*sodium azide, at current costs, is too expensive to use at the rates required.

B. New stands

1. Grow in an area with a reliable dry spell in early to mid summer, in a position preferably isolated from volunteer plants.
2. Treat seed to be sown with triadimenol at 0.01% a.i., by weight to prevent germination of sclerotia present in the seed line (Hampton, unpublished data).
3. Follow the procedures for established stands.

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