THE EFFECTS OF CULTIVATION ON DISEASE INCIDENCE IN GRASS GRUB POPULATIONS

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SUMMARY

Grass grub *(Costelytra zealandica)* populations under younger pastures generally have a lower incidence of disease than those in older pastures. It was investigated whether this was due to the cultivation itself or low host numbers for the first 2 years after cultivation. Four cultivation treatments were studied: paraquat and direct drilling, shallow rotary hoeing, deep rotary hoeing and mould board ploughing. Insecticide was used to effect low host numbers. There were no significant differences in disease levels between cultivation treatments but shallow rotary hoeing results in a less aggregated distribution of pathogens. There is some indication of reduced disease incidence where host numbers have been kept low for 1 and 2 years.

INTRODUCTION

Two or three years after cultivation and pasture renewal grass grub can become a serious problem with high populations causing severe damage to pasture. Observations made at the Takapau Research Station in central Hawkes Bay have shown that populations in young pastures have a much lower incidence of protozoan and milky disease than pastures which have been down 5 years or more.

A similar situation has been shown to occur in porina populations. Young pastures harbour high porina populations which have a low incidence of virus disease (Kalmakoff 1979). Kalmakoff suggested that cultivation removed virus from the soil surface where it is readily accessible to porina, thereby reducing levels of infection.

This paper reports on a trial carried out to determine if low disease levels in grass grub populations in new pasture are directly attributable to the effects of cultivation or are a consequence of low host numbers for one or two generations following cultivation.

METHOD

Cultivation treatments

The four treatments were as follows:

1. No cultivation — plots were treated with paraquat and then direct drilled.
2. Shallow rotary hoeing — plots were rotary hoed to a depth of approximately 8 cm.
3. Deep rotary hoeing — soil was thoroughly mixed to a depth of approximately 16 cm.
4. Mouldboard plough — the plough was used to invert the turf and the surface was lightly harrowed to provide a seed bed.

Cultivation was done in September 1977 and immediately sown with ryegrass *(Lolium perenne)*/white clover *(Trifolium repens)*. Plots were 5.5 x 7.3 m with 8 replicates of each treatment in a randomised block design.

Low host numbers

Each main cultivated plot was subdivided into six small subplots and each of three treatments applied to two of these randomised plots. The treatments were —

(a) grass grub beetles enclosed on plots in November 1977 to ensure the development of a population immediately after cultivation.

(b) Insecticide applied to plots in February 1978 to eliminate grass grub from plots for 1 year. Grass grub beetles were then enclosed on these plots in November 1978.

(c) Insecticide was applied to these plots in February 1978 and 1979.

The insecticide used was fensulfothion, applied with a hand shaker at the rate of 2.2 kg/ha.

Sampling

In July 1978 and May 1979, two-four 15 cm spade squares were taken from each subplot excluding those treated with insecticide. More spade squares were taken where grass grub numbers were low. The insecticide treated areas were sampled by taking four spade squares from treated areas within each main plot. In May 1980 and 1981 five 10 cm diameter soil cores were taken from each subplot.

All grass grub sampled were examined microscopically for the presence of pathogens.

RESULTS

The pathogens recorded were three protozoan species, *Nosema takapauensis,* the most common infection, *Nosema costelytrae* and a *Mattesia* sp, and a milky disease bacterium *Bacillus* sp.

Effects of cultivation on disease incidence

The results were very variable between plots. Although for all years except 1981, the shallow rotary hoed treatment had the highest incidence of disease, this was not statistically significant (Table 1). By 1981 there was very little difference between the plots but the mouldboard plough treatment remained the lowest. In 1980 and 1981 the distribution of disease incidence was examined by comparing the proportion of those core samples containing grass grub, which had diseased larvae in them with the percentage of grubs with disease in those samples (Table 2). The shallow rotary hoed treatment had significantly ($P < 0.01$) more samples with diseased larvae, and a lower incidence of disease in those larvae than the other treatments. In 1981 the distribution of disease was similar in the direct drilled and both rotary hoed plots but in the mouldboard plough treatment there were significantly fewer samples containing diseased larvae and a greater level of disease in them ($P < 0.01$).

TABLE 1: Percent disease for each cultivation treatment, 1978-1981.

Treatment	1978	1978	1980	1981
Direct drill	7.8	12.8	12.9	13.9
Shallow rotary hoe	13.1	20.0	16.4	13.0
Deep rotary hoe	10.4	15.6	12.1	15.8
Mouldboard plough	11.6	11.4	11.5	7.9

TABLE 2: Distribution of disease under different cultivation treatments, 1980-1981.

	% grass grub samples with diseased larvae		% grass grub diseased in samples with diseased larvae	
	1980	1981	1980	1981
Direct drilled	21.7	18.8	51.5	56.8
Shallow rotary hoed	32.3	23.9	38.7	45.4
Deep rotary hoed	19.0	21.2	55.2	50.0
Mouldboard ploughed	18.2	13.9	55.9	70.0

Effect of low host number

In 1978 mean density of grass grub in the insecticide treated plots was 26/m^2 compared to 194/m^2 in the untreated plots. In 1979 there was significantly less disease in these treated plots (Table 3). Similar effects were obtained for all cultivation treatments, but the difference was not significant for the mouldboard plough treatment in which only low disease levels were found. In 1979 the densities in the different treatments were: 281/m^2 in plots with no insecticide treatment, 117/m^2 in plots with one insecticide treatment and 9.5/m^2 in plots where insecticide was applied for 2 successive years ((a), (b) and (c) in methods). In 1980 disease incidence was variable and there were no differences between these treatments but in 1981 there was again an indication that low host numbers for 1 and 2 years reduced infection in the population (Table 3). These differences were significant for the shallow rotary hoed ($P < 0.05$) and the mouldboard plough treatments ($P < 0.01$).

TABLE 3: Percent disease in each cultivation treatment after 0, 1 and 2 years of insecticide application.

Treatment	Years of insecticide treatment	% disease 1979	% disease 1981
Direct drilled	0	11.7	7.4
	1	7.8	9.1
	2		15.4
Shallow rotary hoed	0	11.2	16.3
	1	9.8	13.3
	2		8.9
Deep rotary hoed	0	13.1	18.9
	1	8.8	16.6
	2		10.7
Mouldboard plough	0	5.1	4.3
	1	3.6	3.4
	2		0

DISCUSSION

It was expected that little disturbance of the soil, such as in the direct drilled treatment, would result in the highest levels of infection in subsequent generations of grass grub. That it did not may partly be due to the clover dominant sward resulting from application of paraquat which is thought to reduce the susceptibility of grass grub to disease (Miln unpublished). Although there is a suggestion of higher disease levels under a shallow rotary hoed treatment there is insufficient evidence to prove this. This aspect may warrant further study. In 1978, at the beginning of this trial a severe drought considerably reduced incidence of disease in grass grub populations at Takapau. It is likely that in the direct drilled and shallow rotary hoed treatments the protozoan spores were higher in the soil profile than in the other treatments and more vulnerable to lack of moisture and high temperatures which affect their survival. There is evidence, here, to show that cultivation will redistribute pathogens in the soil. Thorough mixing of the soil at a level where it is still accessible to grass grub, such as in the shallow rotary hoed treatment, results in a more even distribution of disease rather than the aggregated 'hotspots' which occur in other treatments.

Little is known of the persistence of protozoan spores in the soil but it is suspected that viability declines considerably after a year. Hence maintaining reasonable levels of infection in a population requires regular renewal of pathogens through new infections. Low numbers of grass grub will mean the addition of few 'new' pathogens to the soil and subsequently a lower disease incidence. There is an indication here that host suppression through the use of insecticide will cause lower disease levels in the next generation of grass grub.

In conclusion the low incidence of disease in grass grub populations after cultivation which is implicated as a reason for the build up of those populations to damaging levels is probably due more to low grass grub numbers than to the physical effects of the cultivation. Shallow cultivation, however, may help to conserve pathogens and will assist in distributing them more evenly through the soil.

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REFERENCES

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