ETIOLOGY AND CONTROL OF SOME FRUIT DISEASES OF AVOCADO (*PERSEA AMERICANA* MILL.) AT WESTFALIA ESTATE

by

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Submitted to the Faculty of Agriculture
Department of Microbiology and Plant Pathology
University of Pretoria,
in partial fulfilment of the requirements
for the degree of
D.Sc. (Agric.)

PRETORIA

November 1982

ACKNOWLEDGEMENTS

The author is indebted to his promoter, Professor J.M. Kotzé, Head of the Department of Microbiology and Plant Pathology, University of Pretoria, for his guidance and encouragement throughout this study.

Grateful acknowledgement is made to the Management of Westfalia Estate, in particular to Mr. F.J. Lourens, Dr. D.L. Milne and Mr. J.C. Toerien as well as the South African Avocado Growers' Association for the financial support that made this work possible.

The kind co-operation of the Section Managers at Westfalia Estate and the assistance of Mr. J.T. Ramathoka is greatly appreciated.

Grateful appreciation is expressed to Dr. D.L. Milne for his valuable help during the writing-up of this thesis.

CONTENTS

GENERAL INTRODUCTION

Avocado growing in South Africa is a relatively young and a rapidly expanding industry. The number of avocado trees planted up to 1970 totalled 320 000 and increased to 845 000 in 1980 (Burelli, 1981). Production has also increased considerably in the past few years. In 1974, 3 040 tons of avocados were exported from South Africa while 9 500 tons were shipped to overseas markets in 1979 (Lourens, 1979). In 1980, the export totalled 11 632 tons (Kotzé, 1981).

The introduction of new rootstocks resistant to Phytophthora root rot and the registration of modern chemicals for the control of the disease have given impetus to an increase in plantings. Further increases in production are expected and greater efforts are therefore needed to ensure the arrival of high quality fruit on a competitive overseas market.

An important aspect of the South African avocado export industry is the great distance from the European market. Fruit is transported under refrigeration and the exceptionally long cold storage period required for sea transport further increases and complicates South Africa's post-harvest problems. Heavy losses caused by post-harvest diseases have occurred in the past, but most of them remained unreported and uninvestigated. It is only in the past two years that the South African Avocado Growers' Association has been conducting a detailed survey of the problems experienced with fruit arriving in Europe.

The purpose of this study was to identify the major pre- and post-harvest fruit diseases, to investigate their characteristics and to devise effective control measures. The investigations were carried out at Westfalia Estate (Pty) Limited. The estate is situated in the North Eastern Transvaal (longitude 30° 10' and latitude 23° 45') and is the largest single avocado grower in South Africa, with about 500 hectares planted mainly with the cultivars Fuerte and Hass and to a lesser extent with Edranol and Ryan.

The average rainfall at Westfalia Estate between 1914

and 1981 was 1 291mm, most of which fell in the summer between October and March. This warm, humid climate creates conditions conducive to a wide variety of unique disease problems on avocados.

PART ONE

CERCOSPORA SPOT DISEASE OF AVOCADOS

1 - INTRODUCTION

In South Africa, Cercospora spot disease of avocados was first described from the Tzaneen area by Brodrick, Pretorius and Frean (1974) and at that time it was thought to be caused by a Phomopsis species. Growers named it "black spot". According to Mr. W.E. Maddison, Section Manager of Westfalia Estate, who has over 30 years of practical experience with avocados, the disease was first observed in the mid-sixties as a problem of increasing economic importance.

According to Brodrick et al. (1974) the disease spread alarmingly throughout the Northern Transvaal following the heavy rains during the 1971/72 season and today it is found in all major avocado centres of the Lowveld.

2 - LITERATURE REVIEW

Cercospora spot of avocados was first described in Florida by Stevens (1922), who reported that the disease was responsible for a large percentage of inferior quality fruit. Subsequently, Zentmyer (1953) referred to it as the most important disease of avocados in Florida. Cercospora spot is also prevalent in Martinique and Cameroon (Gustafson, 1976) and in Mexico, where it is the second most common disease of avocados (Turu, 1969).

Various descriptions of the symptoms of the disease have been published. Stevens (1922) described Cercospora spot or blotch disease of avocados as a surface spott · of fruit from seedling trees which first appears when t uit is less than semi-developed, but is more pronounced a. ng late maturity. Fully developed spots are 3 - 6 mm in diameter, slightly irregular, usually black in colour and often with white areas of sporulation in the centre. The spots may be scattered on infected fruit or they may coalesce to form irregular black patches. Normally the disease is confined to the rind of the fruit, although the flesh may be invaded during the advanced stages of disease development. The interior of the spots consists of brown, spongy tissues made up of dead collapsed rind cells interwoven with dark mycelium of the fungus. According to Zentmyer (1953), Cercospora spot is manifested as small, brown, slightly sunken lesions with a definite margin, but irregular in shape, scattered on the fruit. The spots later develop cracks through which other fungi may penetrate the fruit. Small angular spots are caused by the pathogen on leaves (Ruehle, 1943b; Zentmyer, 1953). In South Africa, Cercospora spot is characterised by minute, raised, shiny, black spots, 1 - 3 mm in diameter which are frequently associated with cracking and corking of the lenticels.

Stevens (1922) found Cercospora spot disease only on seedling avocados and never on grafted cultivars. Ruehle (1943b) reported that the most susceptible cultivars in Florida were Waldin, Booth 7, Booth 8, Taylor, Linda, Lula, Nabal, Trapp and Wagner, while Collinson, Fuchsia and Pollock were moderately susceptible.

Traditionally, Cercospora purpurea Cke has been implicated as the causal organism of Cercospora spot (Cooke, 1878). However, in the revision by Deighton (1976), C. purpurea was renamed Pseudocercospora purpurea (Cke) Deighton. P. purpurea is characterised by dark to black, globular to irregular stromata, 15 - 125 µm in diameter in fruit spots. Fascicles are fairly to extremely dense, divergent to compact. Conidiophores are pale to medium dark olivaceous brown, dark in mass, uniform in width and colour, multi-septate, not or rarely branched, slightly geniculate, straight to undulate with a small spore scar at the rounded tip, 3 - 4,5 x 20 - 200 µm. Some isolates show only short divergent conidiophores and others only long ones, appearing especially long when conidia are persistent. Conidia are obclavato-cylindric, pale olivaceous, with long obconically truncate bases, obtuse to subacute tips, indistinctly 1 - 9 septate, straight to curved, 2 - 4,5 x 20 - 100 µm. Hosts include Persea americana Mill., P. carolinensis Nees. and P. palustris Sarg. (Chupp, 1953).

Saccardo (1901) stated that Cercospora perseae Ellis & Martin is a synonym of Pseudocercospora (Cercospora) purpurea. According to Chupp (1953) this is incorrect. The type of C. perseae has effuse fruiting with conidiophores in a true coremium, while P. purpurea has distinct spots and conidiophores in divergent fascicles.

Stevens (1922) believed that another spore form of P. purpurea exists and that this is probably produced in pycnidia on the bark of dead twigs. Some of his cultures produced small black bodies, similar to immature pycnidia a number of months after having been inoculated on sterilised avocado twigs. However, these bodies never contained spores.

P. purpurea grows readily on ordinary laboratory media and produces a typical growth which is first greyish in colour and later becomes brown or blackish-brown. Round, raised, tufted grey colonies which are hemispherical in outline are produced on Cornmeal Agar. The surface growth is composed of short, thickly tufted hyphae and the colony has a tough or leathery consistency (Stevens, 1922).

A number of reports have been published on the chemical control of Cercospora spot. Stevens (1922) recommended preventative sprays with two or three applications of Bordeaux mixture. The first application was to be made after fruit set, followed by one or two sprays at three-weekly intervals. He further pointed out that the control of Cercospora spot is more important than the control of anthracnose since elimination of Cercospora spot would result in less wound openings for the anthracnose organism to enter. Ruehle (1940; 1941; 1943a; 1953) evaluated various Bordeaux and cuprous oxide mixtures for the control of Cercospora spot on different avocado cultivars. Three applications sprayed at monthly intervals resulted in a tenfold and more decrease in the percentage infected fruit, while a fivefold decrease was observed following two applications. McMillan (1976) reported that Cercospora spot in Florida could be controlled by timely applications of copper sprays to developing leaves and fruit. An application of copper in early May, followed by another in early June gave effective control on cultivars maturing in summer and autumn. On cultivars maturing in winter a third application in mid-July was necessary for effective control of the disease. McMillan (1976) further stated that Cercospora spot can be controlled by monthly field sprays with benomyl at 1,7 - 2,2 kg/ha. In South Africa, Brodrick et al. (1974) reported that promising results were obtained with three, four and five applications of an unspecified organic systemic fungicide.

Brodrick (1978) discussed various methods for fungicidal control experiments on avocado fruit diseases. He recommended that fruit on the tree be enclosed in paper bags which are tied around the fruit pedicel for a certain period of time to investigate natural infection. This technique was also employed successfully by Kotzé (1963) and Viljoen, Steyn and Kotzé (1972) to establish infection periods for other fruit diseases.

3 - MATERIALS AND METHODS

The importance of Cercospora spot was determined by recording the percentage losses in export fruit for the past six years (1976 - 1981) at Westfalia Estate. In this packhouse survey, fruit with more than five mature Cercospora spots was classified unsuitable for export. Fuerte, Edranol, Hass and Ryan avocado cultivars were also included in the study to obtain information on the relative susceptibility of these cultivars to the disease. It was conducted throughout the picking season in order to study the seasonal distribution of Cercospora spot. All the fruit used in the investigation was commercially sprayed with benomyl at a concentration of 0,025 percent active ingredient (a.i.) twice every summer season, the first spray being applied in November and the second in January. In total about 150 000 fruits were examined.

To confirm the positive identification of the causal organism, artificial inoculations were made to reproduce typical Cercospora spot symptoms under controlled conditions (Koch's postulates). Axenic cultures of P. purpurea were grown on sterile avocado pieces placed on Water Agar in Petri dishes. The agar consisted of 10g technical agar added to one litre distilled water and sterilised at 121°C for 15 minutes. Sterile avocado pieces were obtained by thorough flaming of the surface of hard fruit and then cutting it aseptically into pieces. Conidia from sporulating P. purpurea colonies were suspended in sterile distilled water and applied to Fuerte fruit with an atomizer. Fruit on the tree was closed in paper bags prior to inoculation to prevent natural infection and was then closed in polythene bags for five days immediately after inoculation. Finally the polythene bags were removed and the fruit was enclosed in paper bags again until harvest. At harvest time symptoms were analysed and re-isolations of the fungus were made on Potato Dextrose Agar containing 39g Merck PDA suspended in one litre water. Notes were made on the appearance and development of symptoms under natural orchard conditions and on fruit inoculated artificially.

Observations were made on the pathogen and its cultural

characteristics during the laboratory work. Cultures of the fungus were submitted to the Commonwealth Mycological Institute in England to confirm its identity.

During the isolation studies of P. purpurea and associated organisms, a number of artificial media were evaluated, viz. Czapek-Dox Agar, V-8 Juice Agar, Malt Extract Agar, Cornmeal Agar and Potato Dextrose Agar (PDA). In later work, mainly PDA was used. In the isolation procedure the surface of the fruit was sterilised with 96 percent ethanol for five seconds and a thin layer was cut from the epidermis over the disease spots and small pieces of the subepidermal brownish tissues were transferred to PDA in Petri dishes. Cultures were placed under near ultra violet light (Philips TL 40 W/08 RS) at ambient temperature to induce sporulation. Isolations were made from both young and old lesions (50 fruits each) as well as from sunken and raised Cercospora spots (50 fruits each).

To investigate the distribution of Cercospora spots on the various aspects of the trees, four Fuerte trees were chosen which were not shaded from any direction, at block 34A of Westfalia Section. An average of about 75 fruits were picked from each aspect of each tree on 26 May, 1982 and the mean number of Cercospora spots was established. In the same block, trees with different root rot severity ratings were also selected. The root rot disease index is a rating scale from 0 (healthy) to 10 (dead). On 5 May, 1982 an average of 75 fruits were picked from four trees in each disease category between 0 and 7 and the mean number of Cercospora spots per fruit was established and correlated to the disease index of the trees.

A Hirst spore trap was operated from October, 1977 until April, 1979 in a 14 year old Fuerte orchard at block 34 of Westfalia Section, to monitor the number of air-borne P. purpurea conidia. It was placed 1,5m above the ground and it was adjusted so as to draw in air at a rate of nine litres per minute. As trapping surface, microscope slides covered with vaseline on one side were used. Slides were changed and examined daily. Three stains were initially used to facilitate the identification of spores, viz.

Methylene Blue, Rose Bengal and Malachite Green. Later in the study, Methylene Blue dissolved in water at 0,25 percent, was used as a standard stain. Conidia were identified by taking measurements and considering the number of septae as well as the degree of staining. Conidia which measured between 2 - 4,5 µm in width and 20 - 100 µm in length, having no more than nine septae and exhibiting more pronounced apical and basal cell staining were classified as P. purpurea conidia. In orchard 34 of Westfalia Section, close to the spore trap, a small weather station was established. Temperature, relative humidity and rainfall were recorded and correlated with the spore trap results.

Experiments on the critical infection period of avocado fruit by Cercospora spot disease were conducted at block 35 of Westfalia Section during the 1977/78 season, at block 34A of Westfalia Section during the 1978/79 season and at block 14 of Westfalia Section during the 1981/82 season. According to the method of Kotzé (1963) and Viljoen et al. (1972) trees were selected at random and fruits were closed in brown paper bags on the trees. Initially all fruits were bagged and then exposed to natural Cercospora spot infection according to a pre-determined time schedule and then closed again to prevent contaminating infections. In the 1977/78 summer the experiment was started with fruit exposures from October until March in a cumulative way on a monthly basis. The rainfall experienced during that period was 1 503 mm and assessment of results took place on 20 June, 1978. The experiment in 1978/79 started in November and ran until March with both monthly and cumulative time exposures. The rainfall during these five months was 773 mm and results were evaluated on 5 June, 1979. The 1981/82 experiment also included monthly and cumulative time exposures from November until March and the rainfall during this period was 612 mm and the results were analysed on 13 May, 1982. There were four single tree replicates in each treatment and 200 fruits were closed in paper bags on each tree at the commencement of the experiments.

The following chemicals were evaluated in experiments on the control of Cercospora spot disease at Westfalia Estate:

benomyl, in a 50% a.i. WP form
thiophanate-methyl, in a 65% a.i. WP form
thiabendazole (TBZ), in a 45% a.i. flowable formulation
fosetyl-Al, in a 80% a.i. WP form
glyodin, in a 30% a.i. EC form
captafol, in a 80% a.i. WP form
captan, in a 50% a.i. WP form
copper oxychloride, in a 80% a.i. WP form
copper hydroxide, in a 77% a.i. WP form
etaconazole, in a 10% a.i. EC form
propiconazole, in a 10% a.i. EC form
prochloraz, in a 40% a.i. EC form
bitertanol, in a 19,5% a.i. EC form
PP 296, experimental fungicide
B 77, experimental fungicide
Nu Film 17 (pinolene), film forming agent
Agridex, experimental additive
Plyac, experimental additive
Solvaid, experimental additive
Biofilm, experimental additive

The chemical control experiments were initiated in 1976/77 season, when three systemic fungicides were tested, namely benomyl, thiophanate-methyl and fosetyl-Al. The site of the experiment was at block 34A of Westfalia Section, with four single tree replicates in each treatment and trees in all treatments being sprayed twice with high volume ground sprayers to run-off point. The first spray was applied on 19 November, 1976 and the second on 3 February, 1977.

In another intensive investigation during the 1977/78 season, the optimum timing and the number of benomyl sprays needed for the best Cercospora spot control was tested. The site of the experiment was again at block 34A of Westfalia Section, where four single tree replicates selected at random were used in each treatment. The concentration of benomyl was 0,025% a.i. with 0,02% Nu Film 17. Results were evaluated twice, first at the peak of the Fuerte picking season, on 12 April, 1978 and again on 7 June, 1978. 100 Fruits were harvested from each tree in each treatment of each assessment and evaluated for the severity of Cercospora spot infection.

In the 1978/79 season the two-spray treatments were further investigated. Applications were made in mid-November and mid-January. The effect of Biofilm and Solvaid experimental additives were compared against the standard Nu Film 17, in mixtures with benomyl. Experimental fungicides etaconazole and propiconazole were also tested. To one of the benomyl treatments TBZ was added in the second spray. There were eight single tree replications in each treatment and 100 fruits were harvested for evaluation from each tree. Assessment of the results was carried out on 25 May, 1979.

In the 1979/80 season the Cercospora spot control experiment was continued in block 34A of Westfalia Section. Plyac and Solvaid experimental additives were tested against Nu Film 17. New experimental fungicides were also evaluated, these included: PP 296, B 77 and glyodin. Other fungicides used in the experiment were: copper oxychloride, copper hydroxide, captafol, fosetyl-Al and benomyl. Two sprays were applied for all treatments, the first in mid-November and the second in mid-January, with the exception of treatments 9 and 11 in which a third spray was applied in mid-December. The number of single tree replicates was eight in each treatment and 100 fruits from each tree were used for evaluation. Assessment of results was made on 3 May, 1980.

Chemical control experiments were continued in the 1980/81 season and captafol was tested at various concentrations and in spray programmes with benomyl and copper oxychloride with the addition of Nu Film 17. Eight randomly selected trees at block 34A of Westfalia Section were used in each treatment and in all treatments trees were sprayed twice (mid-November and mid-January). An average of 50 fruits were harvested and evaluated from each tree in each treatment on 14 April, 1981.

In the 1981/82 season the site of the Cercospora spot control experiment was transferred to block 34B of Westfalia Section. Fuerte trees in this block were six years of age at the commencement of the experiment. Six randomly selected trees were used in each treatment and the test fungicides included benomyl, captafol, captan, copper oxy-

chloride, copper hydroxide and prochloraz. In all treatments trees were sprayed in mid-November and mid-January. An average of 60 fruits were harvested from each tree in each treatment and evaluated on 8 April, 1982.

Data collection in the experiments was based on methods recommended by Brodrick (1978). Each fruit was examined for the presence of Cercospora spot immediately after harvest. The number of spots was established on an evaluation scale with five categories. The number of spots in category one was zero, in category two 1 to 5, in category three 6 to 10, in category four 11 to 20 and in category five 21 or more. Fruit in categories one and two were regarded as marketable whereas fruit in categories three, four and five were classified as non-marketable. Analyses of the results were carried out by using standard statistical methods and a level of probability for significance of 95 percent.

4 - RESULTS

4.1 LOSSES CAUSED BY CERCOSPORA SPOT AT WESTFALIA ESTATE

The importance of Cercospora spot on the commerically sprayed avocados at Westfalia Estate is illustrated in Table 1. The disease caused most damage to Fuerte and Ryan cultivars, while losses of Edranol and Hass were significantly less.

The difference in losses between the seasons followed the rainfall pattern measured from the beginning of October until the end of February. When losses of Fuerte fruits were correlated with the rainfall figures, it was found that a significant (r = 0,829749) correlation exists between Cercospora spot damage and rainfall and this correlation was best described with a linear regression model of y = 4,47 + 0,01 x (Fig. 1).

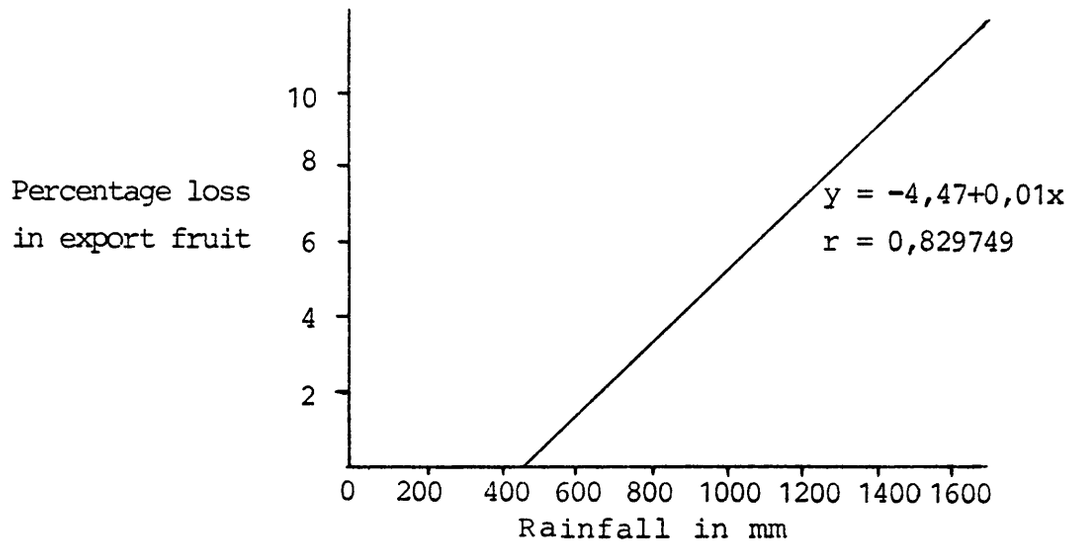

FIG. 1. - The correlation between rainfall and Cercospora spot losses on commercially sprayed Fuerte at Westfalia Estate.

The correlation between rainfall and Cercospora spot damage to Ryan best fitted the linear regression model of y = -3,43 + 0,007 x, with a significant correlation

coefficient of r = 0,899659 (Fig. 2).

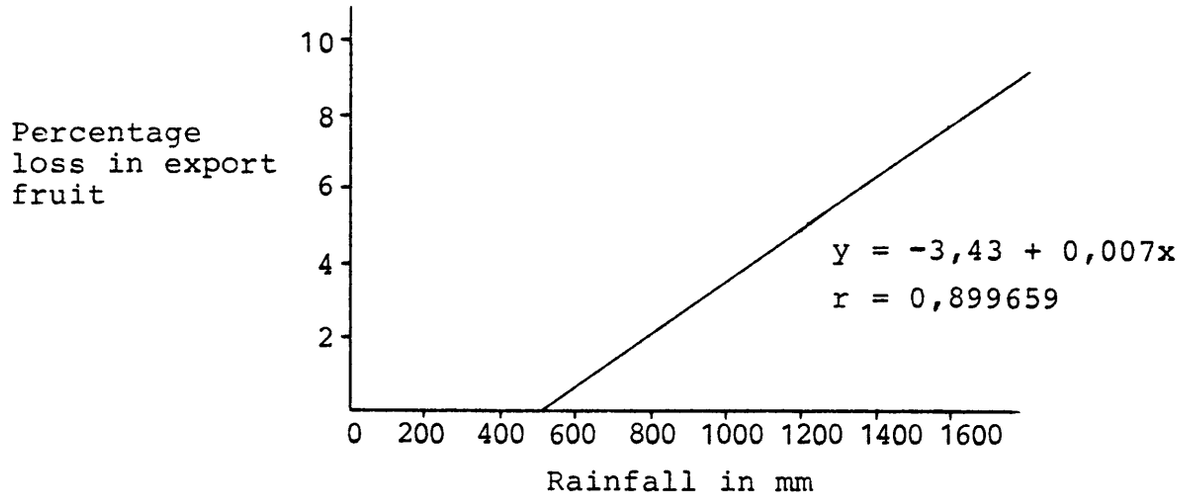

FIG. 2. - The correlation between rainfall and Cercospora spot losses on commercially sprayed Ryan at Westfalia Estate.

The statistical analysis of the losses in export fruit showed an increase in the severity of Cercospora spot at the beginning of the harvest season and a slight decrease towards the end of the picking season. The non-linear regression model to describe Cercospora spot incidence on Fuerte from February to August is $y = -2,25 + 4,44x - 0,46x^2$, with a highly significant correlation coefficient of R = 0,961865 (Fig. 3).

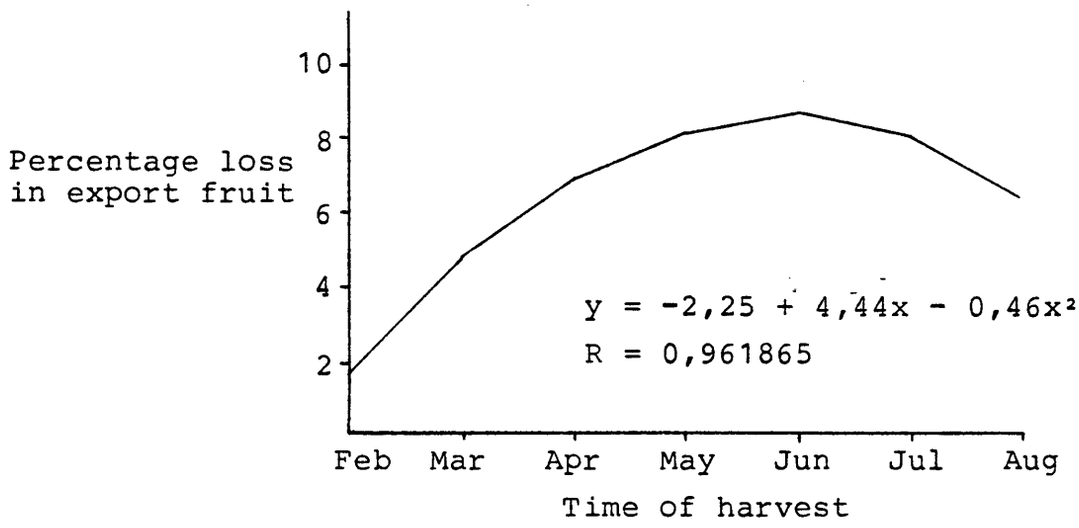

FIG. 3. - The losses in export fruit of commercially sprayed Fuerte in relation to harvest time at Westfalia Estate.

TABLE 1. - Percentage losses caused by Cercospora spot disease on commercially sprayed avocados at Westfalia Estate.

Cultivar	Season	February	March	April	May	June	July	August	September	Mean percent loss (N = 150 000 fruit)
Fuerte	1975/76	4,07	4,95	10,60	7,69	7,63	-	-	-	6,98
	1976/77	0,18	1,32	6,89	9,92	14,00	15,33	-	-	7,94
	1977/78	-	2,58	7,80	8,73	12,70	10,00	-	-	8,36
	1978/79	-	0,38	0,95	0,80	1,68	5,50	6,00	-	2,55
	1979/80	-	1,55	1,48	4,42	6,60	2,29	5,19	-	3,58
	1980/81	-	13,77	15,16	15,49	7,40	8,10	-	-	11,98
	Mean	2,12	4,09	7,14	7,84	8,33	8,24	5,59	-	6,89a
Edranol	1975/76	-	-	0	0,14	1,16	0,25	-	-	0,38
	1976/77	-	-	-	0	0,39	0	-	-	0,13
	1977/78	-	-	-	0	0,04	0	0,51	0	0,11
	1978/79	-	-	-	0	0	0	0	0	0
	1979/80	-	-	0,62	0	1,30	0,32	0,37	0,07	0,44
	1980/81	-	-	-	-	0,06	0,46	0	-	0,13
	Mean	-	-	0,31	0,02	0,49	0,17	0,22	0,03	0,19b
Hass	1975/76	-	-	-	-	-	0	0	2,00	0,66
	1976/77	-	-	-	-	0	0	0	-	0
	1977/78	-	-	-	-	-	-	0,38	0,40	0,39
	1978/79	-	-	-	-	-	-	0	-	0
	1979/80	-	-	-	-	-	-	0	0,25	0,12
	1980/81	-	-	-	-	-	0	0	-	0
	Mean	-	-	-	-	0	0	0,06	0,88	0,19b
Ryan	1975/76	-	-	-	4,00	-	1,00	4,00	12,33	5,33
	1976/77	-	-	-	-	-	7,52	7,50	-	7,51
	1977/78	-	-	-	-	-	-	6,66	-	6,66
	1978/79	-	-	-	-	-	0	2,27	-	1,13
	1979/80	-	-	-	-	-	-	3,80	-	3,80
	1980/81	-	-	-	-	-	6,60	1,60	-	4,10
	Mean	-	-	-	4,00	-	3,78	4,30	12,33	4,75a

Means with letters a and b differ statistically at 0,05 level (Duncan's multiple range test)

The increase in losses of Ryan export fruit for a five month harvest period fitted the regression line of $y = 0,58 + 1,69x$, with a non-significant correlation coefficient of $r = 0,697568$ (Fig. 4).

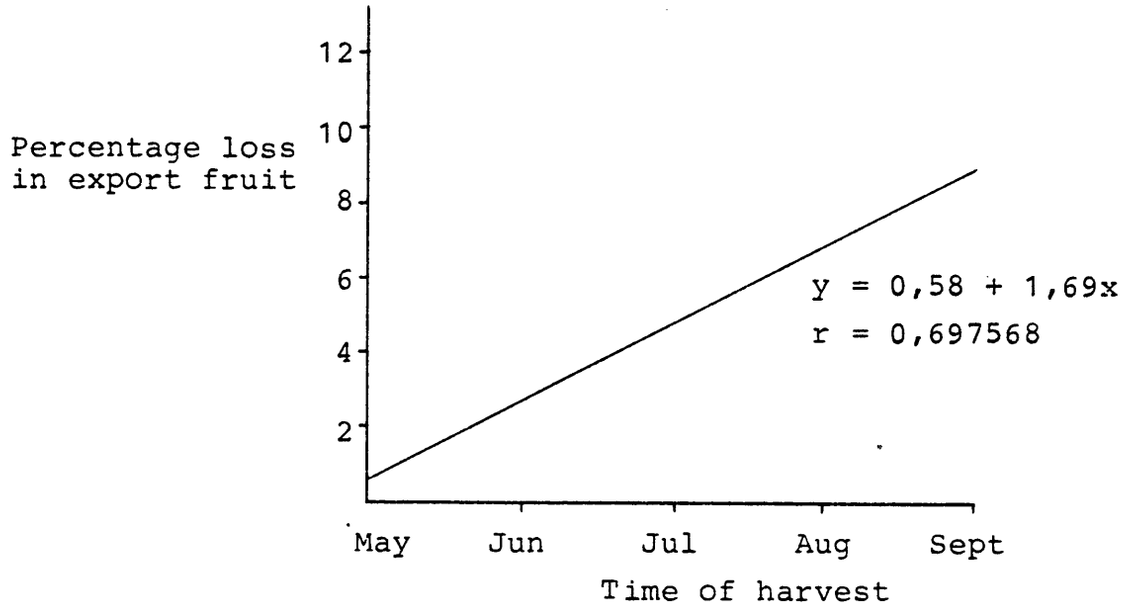

FIG. 4. - The losses in export fruit of commerically sprayed Ryan in relation to harvest time at Westfalia Estate.

4.2 OBSERVATIONS ON THE SYMPTOMS AND THE PATHOGEN

Fuerte fruit was artificially inoculated with the pathogen and it was reisolated from the spots, thereby fulfilling the requirements of Koch's postulates. Observations on symptoms made during the course of this study at Westfalia Estate correspond with descriptions given by earlier workers. The colour of the spots may vary from light brown to black, depending on the stage of development of the disease and the amount of corky cells in cracks associated with the spots. At first, the green epidermis of the fruit becomes slightly darker at the site of the infection and it turns darker as the fruit matures. It would appear that the disease initially causes a swelling of the tissues around the infection site causing the spot to rise above the level of the epidermis (Photos 1 and 2). However, at a later stage when epidermal cells are killed

and the tissues dry out, the spot becomes sunken and horizontal cracks appear, mainly on the perimeter of the spot. These cracks may serve as entrance sites for other fruit-rotting fungi, particularly Colletotrichum gloeosporioides (Photos 3 and 4).

Typical leaf symptoms of Cercospora spot infection were commonly seen on older attached leaves of Fuerte and Ryan cultivars (Photos 5 and 6).

Several isolates of the pathogen were sent to the Commonwealth Mycological Institute in England where taxonomists confirmed the identity of the fungus as Pseudocercospora purpurea (Cke) Deighton.

From a large number of isolations and sporulation studies it was established that most of the fresh P. purpurea isolations produce conidia on artificial media if cultures are kept under continuous near UV light. Sporulation begins about 10 days after isolation and a fair amount of conidia are produced for about 10 days, after which the fungus becomes sterile and previously produced conidia disappear. Such cultures regain the ability to sporulate if transferred to sterile, freshly cut avocado pieces, but only for a limited period. Sporulation under fluorescent light alternated with dark periods resulted in a weak sporulation by the fungus. No sporulation was observed in cultures grown in complete darkness.

The size, form and the number of septae of the conidia derived from colonies cultured on avocado pieces were identical to those collected in the orchard, using the spore trap (Photo 7). Some conidia of the cultures of P. purpurea grown on artificial media (PDA) exceeded the maximum length of 100 µm given by Chupp (1953) and in some conidia the number of septae was 13 instead of the described maximum of 9.

In some isolations, besides the Pseudocercospora conidial form, spermogonia-like bodies with abundant spermatia were found. They developed in a few direct isolations from-fruit-to-PDA and from-fruit-to-sterile avocado.

4.3 DISTRIBUTION OF CERCOSPORA SPOT

The distribution of Cercospora spots on fruit in the various aspects of the trees is presented in Table 2.

TABLE 2. - The distribution of Cercospora spots in various aspects of Fuerte avocado trees in 1981/82 season.

Aspect of tree	Mean number of Cercospora spot/fruit (N = 1200 fruit)
North	0,51 a
South	0,12 a
East	0,06 a
West	0,16 a

Means with the letter a do not differ statistically at p = 0,05 level

Although the number of Cercospora spots on fruit in the warm, sunny northern and western aspects of the trees appeared to be more prominent than on the southern and eastern sides, the difference was not statistically significant.

The distribution of Cercospora spots on fruit harvested from various disease rating trees is analysed in Fig. 5.

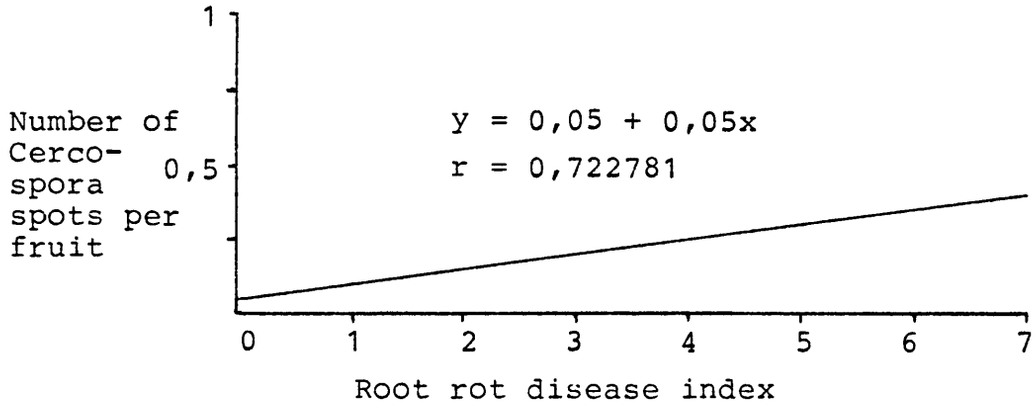

FIG. 5. - The correlation between root rot severity of Fuerte trees and the number of Cercospora spots on the fruit.

A significant correlation was found between the number of Cercospora spots on fruit and the root rot disease severity index of Fuerte trees. The linear regression model to determine the correlation is $y = 0,05 + 0,05x$, with $r = 0,722781$.

The incidence of P. purpurea and associated organisms was studied in young and old Cercospora spot lesions. In both cases spots were mature (Table 3).

TABLE 3. - Percentage incidence of P. purpurea and other fungi in young and old Cercospora spots of Fuerte fruit.

Organisms	Percentage incidence	
	Young spots in Apr. '78	Old spots in Dec. '78
Pseudocercospora purpurea	48,0	14,0
Colletotrichum gloeosporioides	7,5	24,5
Phoma spp.	0,5	2,5
Cladosporium spp.	0,5	2,5
Pestalotiopsis spp.	0,5	0
Phomopsis spp.	0	2,5
Thyronectria pseudotrichia	0	1,5
Fusarium decemcellulare	0	0,5
Drechslera spp.	0	0,5
Unidentified fungi	0,5	6,0
Sterile isolations	37,0	38,5

Pseudocercospora purpurea was more readily isolated from young spots than from old lesions. Furthermore, there were more secondary organisms present in old spots, with a marked increase in the incidence of Colletotrichum gloeosporioides. The incidence of unidentified fungi was also higher in old lesions, while the number of sterile isolations from the two spot groups was practically the same.

A comparison was also made of the incidence of P. purpurea in sunken and raised Cercospora spot lesions. Isolations were made from both types of spots at the same time, in April,

1978 (Table 4).

TABLE 4. - Percentage incidence of P. purpurea and associated organisms in sunken and raised Cercospora spot on mature Fuerte fruit.

Organisms	Percentage incidence	
	Raised spots	Sunken spots
Pseudocercospora purpurea	41,0	50,5
Colletotrichum gloeosporioides	2,5	24,5
Phoma spp.	0	1,5
Cladosporium spp.	2,0	2,5
Pestalotiopsis spp.	1,5	2,0
Unidentified fungi	0	1,5
Sterile isolations	53,0	17,5

The incidence of P. purpurea did not differ much in the two types of lesions. The occurrence of C. gloeosporioides, however, was considerably higher in sunken lesions. The latter also contained more secondary fungi, while a greater percentage of sterile tissue was present in raised spots.

4.4 SPORE TRAPPING OF P. PURPUREA CONIDIA

Spore trap results and weather data are presented in Table 5.

With the multiple regression analysis of the weekly number of conidia caught in the spore trap and the weekly rainfall and mean temperature values, a significant correlation was found. The equation for the regression line is Z (number of conidia) = -58,99 + 3,22x (temperature °C) + 0,18 y (rainfall mm), with a correlation coefficient of R = 0,543507. However, when this weekly conidia catch was compared to either rainfall or temperature respectively, no significant correlation was found. The equation of the linear regression line between conidia and temperature is $y = -48,84 + 3,05x$, with $r = 0,340300$ and between conidia and rainfall is $y = 15,31 + 0,15x$, with $r = 0,341572$. The number of P. purpurea conidia first reached significant

TABLE 5. - The effect of climatic factors on the incidence of air-borne conidia of P. purpurea during the summer of 1978/79.

Date	Number of P. purpurea conidia	Mean temperature °C	Mean relative humidity %	Rainfall mm
1978				
October 1st week	5	20,8	72,0	7,5
2nd week	9	20,6	69,5	8,5
3rd week	3	19,9	70,5	31,3
4th week	6	20,1	61,3	5,8
November 1st week	6	20,4	70,5	86,9
2nd week	24	22,5	64,8	52,4
3rd week	16	20,9	68,6	17,3
4th week	20	23,1	64,8	15,8
December 1st week	31	22,9	67,0	23,0
2nd week	15	21,9	63,3	40,8
3rd week	8	23,6	60,5	0
4th week	20	24,4	57,8	43,3
1979				
January 1st week	29	21,3	64,0	22,0
2nd week	26	24,0	61,2	19,3
3rd week	20	23,9	63,5	59,7
4th week	34	22,7	66,0	61,3
February 1st week	27	23,5	62,8	14,4
2nd week	3	25,9	59,6	0,2
3rd week	65	25,1	65,7	28,4
4th week	16	23,5	67,4	73,2
March 1st week	45	21,7	72,0	137,8
2nd week	7	22,1	61,4	4,0
3rd week	14	25,0	59,0	0,2
4th week	39	23,1	62,3	19,6

proportions with the onset of the rainy, warm summer period, which in this instance, started in November. Large numbers of P. purpurea conidia were detected in the orchard even as late as the last week of March, when commercial harvesting of the Fuertes had already started.

The daily analysis of typical spore trap results along with the weather data is presented in Table 6 to illustrate the role of the climatic factors in spore production.

TABLE 6. - The effect of climatic factors on the incidence of air-borne conidia of P. purpurea on a daily basis from 27 January until 6 February, 1979.

Date	Number of P. purpurea conidia	Mean temperature °C	Mean relative humidity %	Rainfall mm
1979 January 27	1	24,3	59,3	0
28	1	24,0	64,2	0
29	3	26,0	62,5	0
30	7	21,8	77,4	12,5
31	18	20,0	81,8	47,5
February 1	10	21,0	67,9	14,1
2	4	21,3	68,1	0,2
3	8	22,8	60,4	0,1
4	1	23,5	61,2	0
5	2	24,0	60,9	0
6	0	25,5	59,4	0

The multiple regression analysis of the data of the daily spore trapping in Table 6 indicated a significant correlation between the number of conidia and the rainfall and temperature figures. The resulting equation is Z (number of conidia) = 24,87 - 0,93x (temperature °C) + 0,25y (rainfall mm), with R = 0,875452. If analysed separately, the correlation between the number of conidia and temperature is illustrated with a linear regression model of y = 58,25 - 2,29x, with a significant correlation coefficient of r = -0,798131 (Fig. 6).

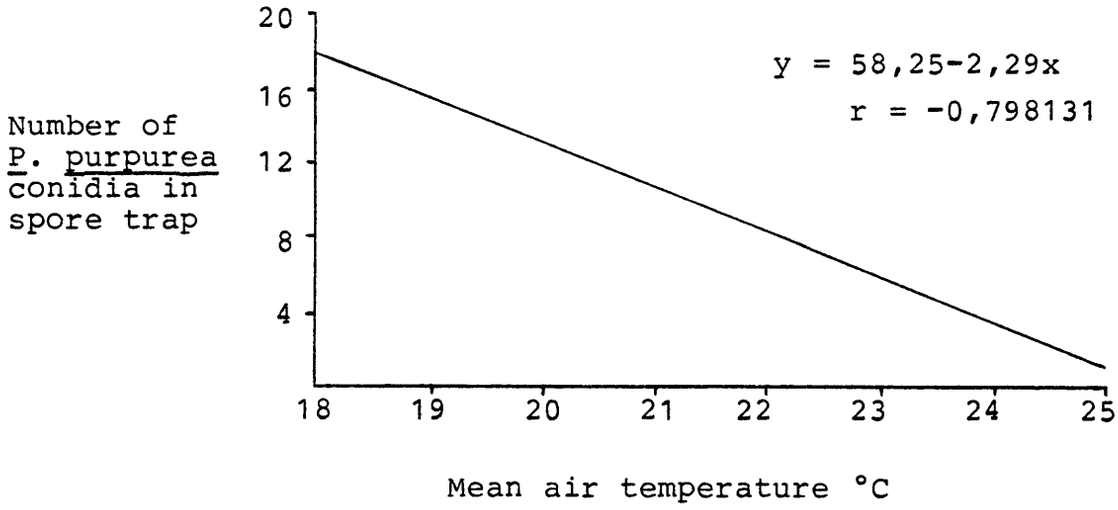

FIG. 6. - The effect of air temperature on the number of trapped P. purpurea conidia analysed on a daily data basis.

The correlation between the number of trapped conidia of P. purpurea and the mean relative humidity was also significant r = 0,798686 and the linear regression model to describe the correlation is y = -32,66 + 0,57x (Fig. 7).

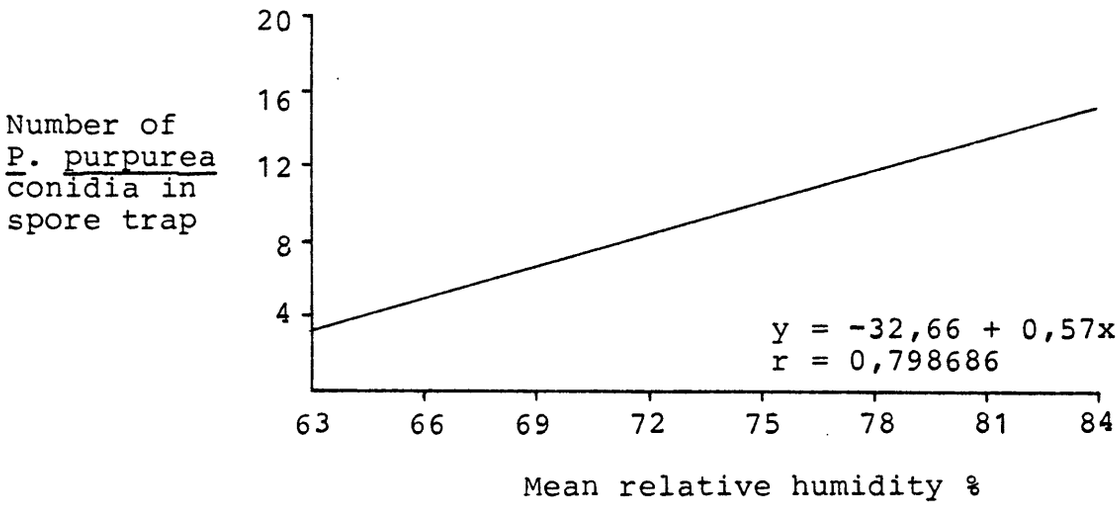

FIG. 7. - The effect of RH on the number of trapped P. purpurea conidia analysed on a daily data basis.

The most significant correlation (r = 0,905787) was found between the number of P. purpurea and rainfall. The linear regression model fitted to the data is y = 2,71 + 0,33x (Fig. 8).

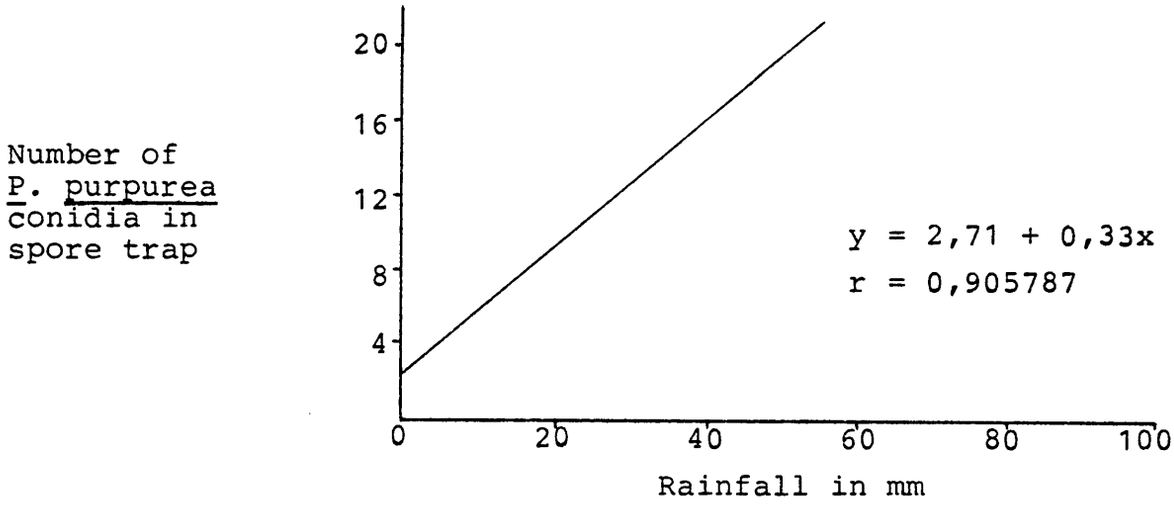

FIG. 8. - The effect of rainfall on the number of trapped P. purpurea conidia analysed on a daily data basis.

It is interesting to note that most of the P. purpurea conidia were trapped in the early morning from about 01h00 to 06h00.

4.5 CRITICAL INFECTION PERIODS FOR CERCOSPORA SPOT

A number of experiments were conducted to detect the critical period for fruit infection by P. purpurea under natural orchard conditions. During the 1977/78 summer the first experiment was undertaken with Fuerte fruit exposures from October until March in a cumulative way on a monthly basis. Treatments in this experiment included the exposing of fruit from the early summer period (October) and these exposure times were gradually extended by a month at a time up to March. The linear regression fitted to the number of Cercospora spots on the fruit and the number of months (exposure period) is y = 2,80 + 0,74x, with r = 0,403114. This indicates a non-significant increase of Cercospora spots on the fruit with the increase in the length

of the exposure time. If exposure time is decreased on a monthly interval from the full season's exposure to the end of summer (March), the correlation is significant r = -0,859188. The increase in Cercospora spot incidence on the fruit in relation to the length of the exposure time is described with the linear regression model of y = 6,11 - 0,79x (Fig. 9).

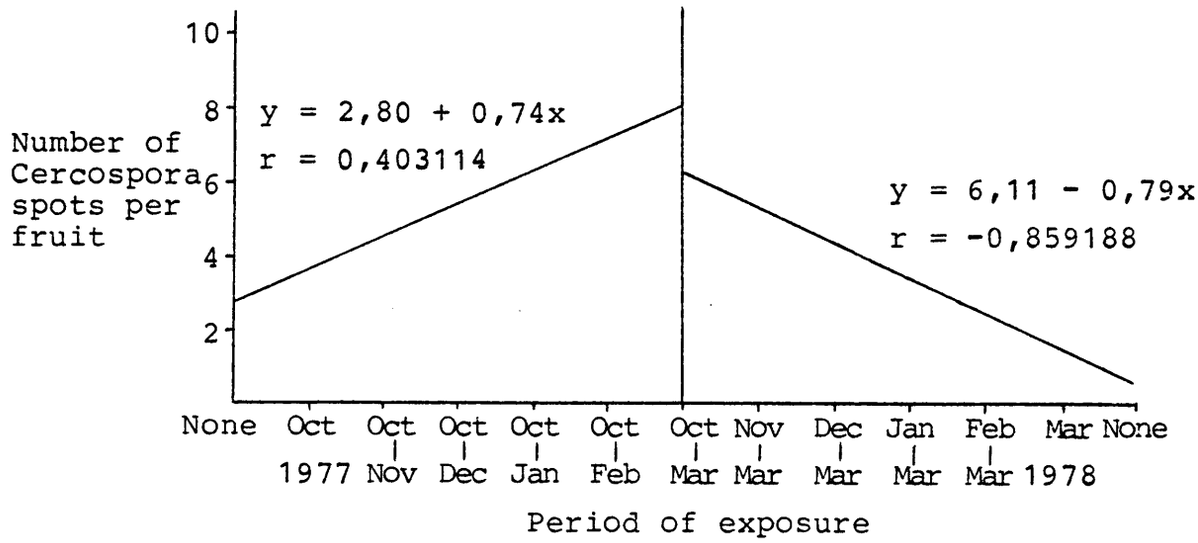

FIG. 9. - The effect of the length of the exposure time on the natural Cercospora spot infection in 1977/78.

The experiment for the detection of critical infection periods in the 1978/79 season was initiated in November and fruit was exposed at a monthly interval on a one monthly basis and also on a monthly cumulative basis. A good correlation, r = -0,899350 was found between the number of Cercospora spots on the fruit exposed on a monthly basis and timing of these exposures. According to this correlation the infection was more severe on fruit exposed to natural infection early in the season and it is described as y = 6,33 - 0,88x. A significant, r=-0,872669 increase in disease incidence was also found for the monthly cumulative exposures. The linear regression model for this increase is y = 7,46 - 1,17x (Fig. 10).

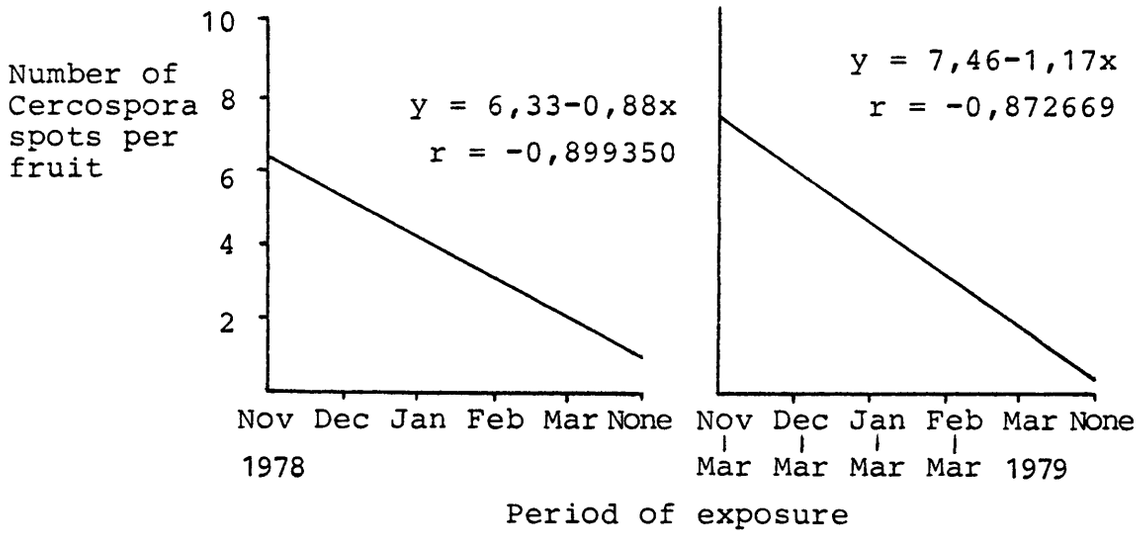

FIG. 10. - The effect of the length and timing of the exposure periods on the natural Cercospora spot infection in 1978/79.

Data of the 1978/79 fruit exposure experiment was also used to analyse correlations between the severity of Cercospora spot disease and rainfall and spore trap figures. The correlation between rainfall and Cercospora spot infection in the monthly exposure is y = 1,65 + 0,009x, with r = 0,319655 and the linear regression model for the correlation between rainfall and the monthly cumulative exposures is y = -0,10 + 0,008x, with r = 885196 (Fig. 11).

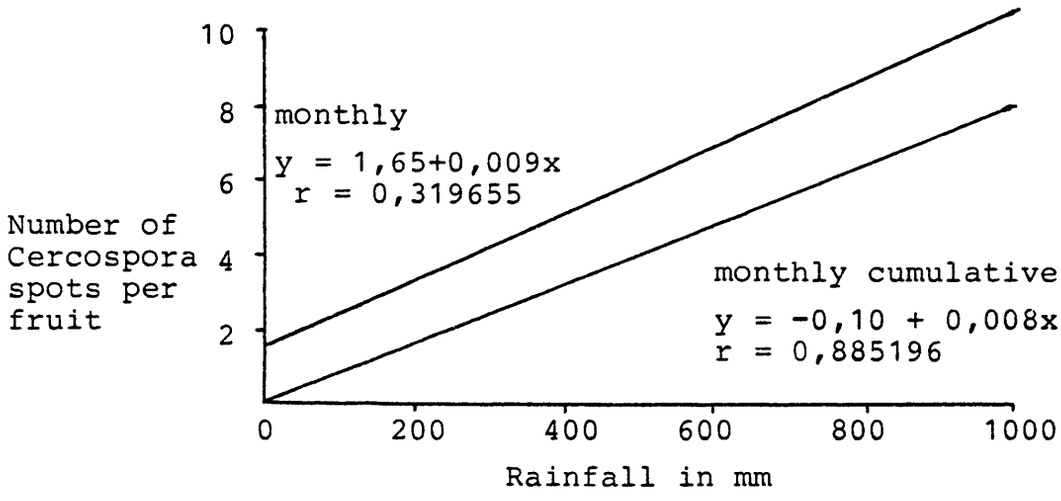

FIG. 11. - The correlation between Cercospora spot infection and rainfall in 1978/79.

The incidence of Cercospora spot infection in the 1978/79 fruit exposure experiment was correlated with the number of P. purpurea conidia in the spore trap on the monthly basis and the following regression line was obtained y = 2,84 + 0,003x, with r = 0,089442. The linear regression model for the correlation between infection and the number of conidia on the monthly cumulative basis is y = 0,06 + 0,01x, with r = 0,853691 (Fig. 12).

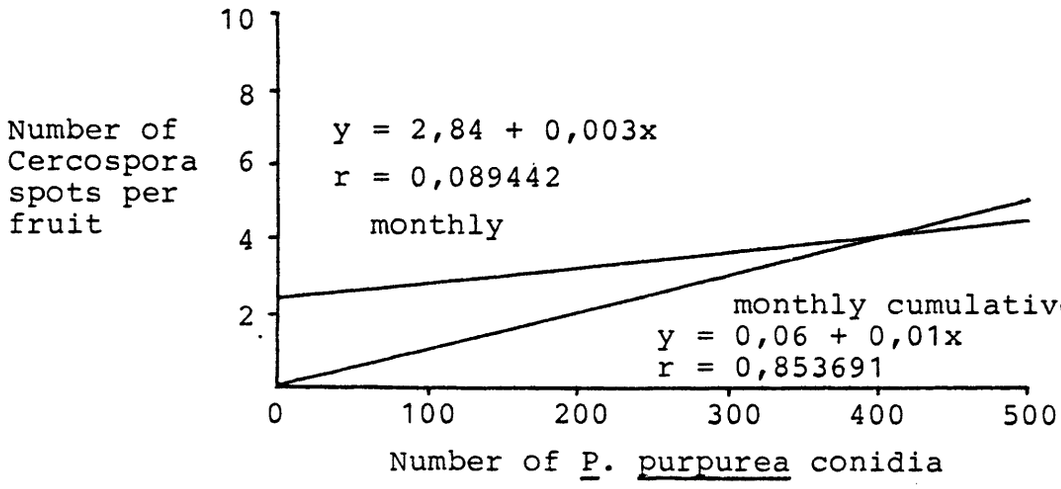

FIG. 12. - The correlation between Cercospora spot infection and the number of trapped P. purpurea conidia in 1978/79.

The third experiment to study critical infection periods under orchard conditions was conducted in 1981/82. The exposure periods were monthly and monthly cumulative. Cercospora spot infection correlated significantly with the exposure period on the monthly basis indicating an increase with early summer exposures. The linear regression model to describe the correlation is y = 4,48 - 0,69x, with r = -0,711963. The correlation between infection and the monthly cumulative exposures is also significant, r = -0,734053 and the equation is y = 5,04 - 0,74x (Fig. 13).

Cercospora spot infections recorded in the fruit exposure experiment of 1981/82 were also correlated with rainfall figures. The linear regression for the correlation between infection and rainfall in the monthly exposure is

y = -1,55 + 0,01x, with a non-significant r = 0,701418. The correlation between infection and rainfall in the monthly cumulative exposures is y = 0,21 + 0,006x, with a significant correlation coefficient of r = 0,786247 (Fig. 14).

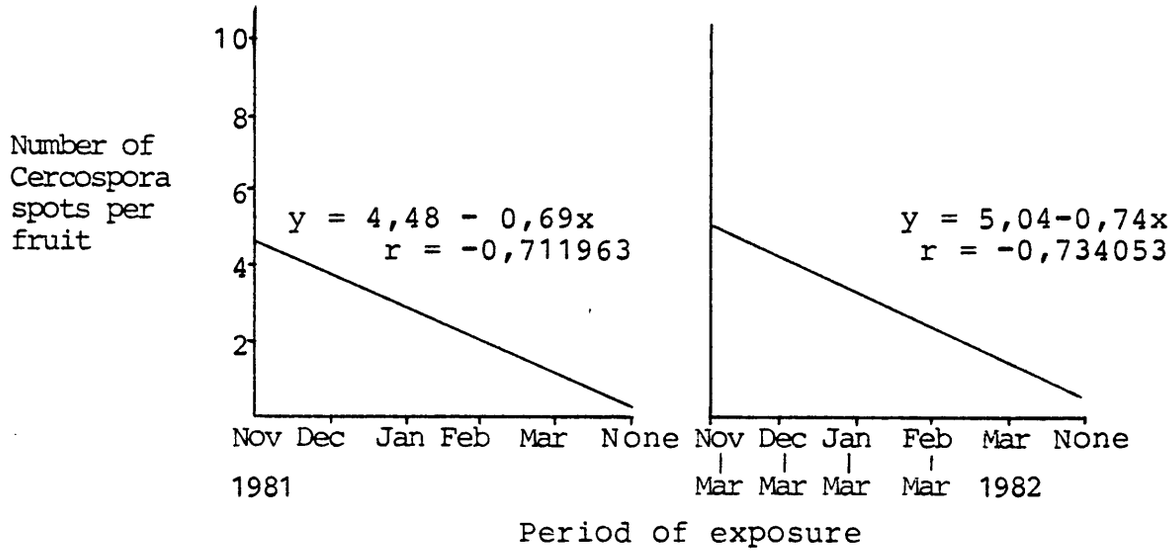

FIG. 13. - The effect of the length and timing of the exposure periods on the natural Cercospora spot infection in 1981/82.

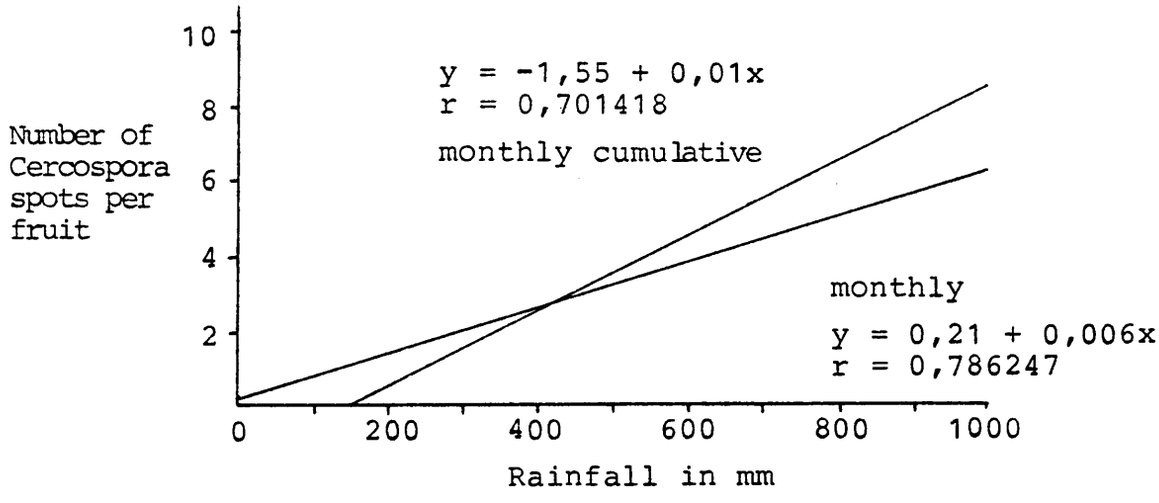

FIG. 14. - The correlation between Cercospora spot infection and rainfall in 1981/82.

4.6 CHEMICAL CONTROL OF CERCOSPORA SPOT

Results of the 1976/77 season's chemical control experiment are presented in Table 7. In the assessment made in April, all three chemicals at the various concentrations showed an equally significant control of Cercospora spot disease. Due to the increase in disease severity, differences between the treatments in June became significant and here benomyl proved to be the most effective at both rates. Thiophanate-methyl at the low rate and fosetyl-Al were statistically inferior to benomyl, but they significantly reduced disease in comparison with the control.

TABLE 7. - Control of Cercospora spot on Fuerte sprayed twice in the 1976/77 season.

Treatment number	Treatments	Mean number of Cerc. spots/ fruit (N = 4800)	
		Assessed on 22 Apr. 1977	Assessed on 15 June 1977
1	Fosetyl-Al 0,3% a.i.	2,5b	4,3b
2	Benomyl 0,02 % a.i.	0,9b	1,2c
3	Benomyl 0,025% a.i.	0,6b	1,3c
4	Thiophanate-methyl 0,05% a.i.	1,1b	3,3b
5	Thiophanate-methyl 0,07% a.i.	1,0b	2,1bc
6	Control	5,2a	8,3a

Means with letters a, b and c differ statistically at 0,05 level (Duncan's multiple range test)

The experiment in 1977/78 investigated the optimum timing of benomyl sprays and results are given in Table 8. In this benomyl timing experiment the severity of Cercospora spot infection increased greatly during the two months which elapsed between assessments. The disease was generally better controlled with frequent, short interval sprays. Late applications slowed down the increase of disease incidence between assessments. Good control was achieved by two benomyl sprays when one was applied in the first week of November and the second around mid-January (treatment No. 14). The first spray

was applied prior to an appreciable amount of rain at the end of November, while the second was in the middle of the very rainy January. Another two-spray treatment (No. 15) applied about four weeks later was inferior at the time of the first assessment, but showed a good residual effect in the second assessment.

In the 1978/79 experiment new additives and fungicides were tested against Cercospora spot, all in two-spray treatments and results are presented in Table 9. The various stickers added to the benomyl mixture did not significantly influence the efficiency of the fungicide against the disease. At low rates, propaconazole gave significantly less effective control than the benomyl and Nu Film 17 combination, while etaconazole and propaconazole at the high rates significantly increased the disease incidence compared to the control. TBZ was added to benomyl with the aim of enhancing post-harvest Dothiorella fruit rot control but it did not significantly improve Cercospora spot control compared to the benomyl plus Nu Film 17 mixture.

Additives were tested again in the 1979/80 season together with some new fungicides (Table 10). The best Cercospora spot control was obtained by using captafol at a concentration of 0,16 percent a.i. in two- and three-spray applications. There were no appreciable differences between benomyl in combination with various additives and results were consistently good. Cu-hydroxide and Cu-oxychloride tended to be less effective than benomyl, however, the differences were not significant. Treatments which failed to check the disease were fosetyl-Al, PP 296, B77, glyodin and bitertanol.

In the 1980/81 experiment captafol was tested at various concentrations and in spray programmes with other fungicides and compared to the standard benomyl spray (Table 11). The statistical analyses of the data obtained from the experiment showed that all chemical treatments ensured an effective reduction of Cercospora spot. There were no significant differences between the various chemical treatments.

Results of the experiment in 1981/82 on Cercospora spot control are presented in Table 12. All treatments in the experiment controlled the disease significantly, except captan

which failed to reduce it to a statistically lower level when the chemical was sprayed alone.

Lastly, the percentage disease control by benomyl has been calculated for the past six years by using data from Table 7 (assessment in April, treatment Nos. 2 and 6), Table 8 (assessment in April, treatment Nos. 14 and 1), Table 9 (treatment Nos. 1 and 9), Table 10 (treatment Nos. 1 and 13), Table 11 (treatment Nos. 1 and 8), and Table 12 (treatment Nos. 1 and 10) and statistically analysed results are presented in Fig. 15. There was a statistically significant decline in the efficacy of benomyl measured in terms of percentage disease control. This reduction is described by the linear regression line of $y = 92,58 - 4,86x$, with a correlation coefficient of $r = -0,787595$.

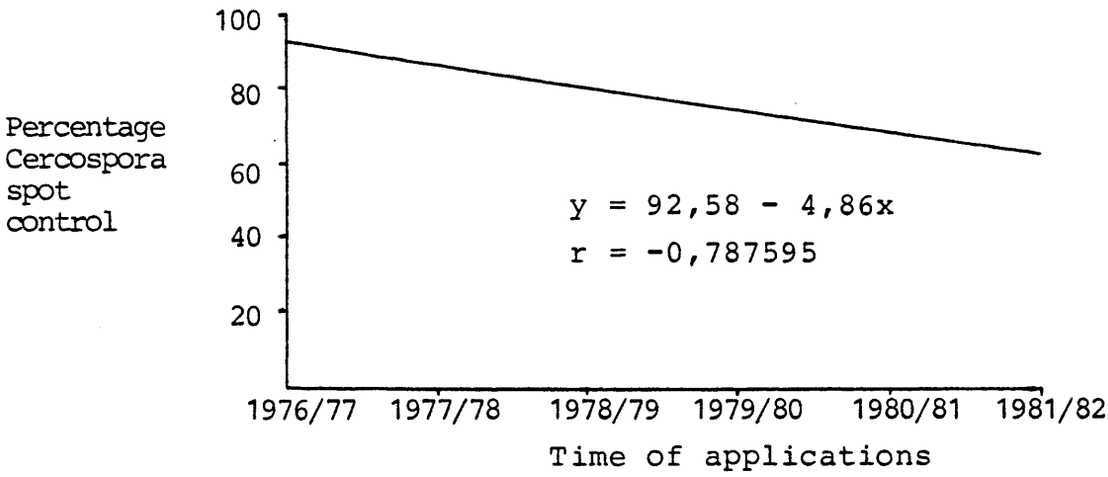

Fig. 15. - The control of Cercospora spot disease by benomyl in experiments over the past six years.

TABLE 8. - The effect of the number and timing of benomyl sprays on Cercospora spot of Fuerte in 1977/78.

Treatment number	No. of applications	Time interval weeks	Date of applications	Mean number of Cerc. spots per fruit (N = 12 000)	
				Assessed on 12 Apr. 1978	Assessed on 7 June 1978
1	-	-	Control	5,1ab	19,5a
2	1	-	6 Oct. 1977	4,0abc	10,4bc
3	2	8	6 Oct. 1977; 2 Dec. 1977	2,3abc	13,9ab
4	3	8	6 Oct, 1977; 2 Dec. 1977; 2 Febr. 1978	1,9abc	7,3bc
5	4	8	6 Oct. 1977; 2 Dec. 1977; 2 Febr. 1978; 6 Apr. 1978	1,0bc	3,5c
6	3	8	2 Dec. 1977; 2 Febr. 1978; 6 Apr. 1978	0,3c	3,4c
7	2	8	2 Feb. 1978; 6 Apr. 1978	3,0abc	6,4bc
8	1	-	6 Apr. 1978	3,4abc	10,3bc
9	1	-	11 Nov. 1977	0,9bc	9,0bc
10	2	9	11 Nov. 1977; 19 Jan. 1978	0,9bc	6,8bc
11	3	9	11 Nov. 1977; 19 Jan. 1978; 27 Mar. 1978	2,7abc	6,5bc
12	2	9	19 Jan. 1978; 27 Mar. 1978	6,0a	12,3bc
13	1	-	2 Febr. 1978	5,6a	10,7bc
14	2	10	2 Nov. 1977; 19 Jan. 1978	0,5c	6,1bc
15	2	10	28 Nov. 1977; 15 Febr. 1978	2,7abc	5,4c

Means with letters a, b and c differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 9. - The evaluation of fungicides and additives for the control of Cercospora spot on Fuerte sprayed twice in 1978/79.

Treatment number	Treatments	Mean number of Cerc. spots per fruit (N = 7 200)
1	Benomyl 0,025% a.i. + Nu Film 17 0,02%	1,3c
2	Benomyl 0,025% a.i. + Biofilm 0,05%	1,9bc
3	Benomyl 0,025% a.i. + Solvaid 0,03%	2,1bc
4	Benomyl 0,025% a.i. + TBZ 0,05% a.i. + Nu Film	1,2c
5	Etaconazole 0,025% a.i. + Nu Film 17 0,02%	7,0a
6	Propiconazole 0,025% a.i. + Nu Film 17 0,02%	8,1a
7	Etaconazole 0,005% a.i. + Nu Film 17 0,02%	2,5bc
8	Propiconazole 0,005% a.i. + Nu Film 17 0,02%	3,0b
9	Control.	4,3b

Means with letters a, b and c differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 10. - The effect of two- and three-spray treatments on Cercospora spot control of Fuerte in 1979/80.

Treatment number	Number of applications	Treatments	Mean number of Cerc. spots/fruit (N=10 400)
1	2	Benomyl 0,025% a.i. + Nu Film 17 0,02%	2,6 cd
2	2	Benomyl 0,025% a.i. + Plyac 0,03%	2,6 cd
3	2	Benomyl 0,025% a.i. + Solvaid 0,03%	2,7 cd
4	2	B 77 150 ppm + Glyodin 0,125% + Nu Film 17	7,1 b
5	2	Bitertanol 0,01% a.i. + Nu Film 17 0,02%	7,8 b
6	2	PP 296 0,08% a.i. + Nu Film 17 0,02%	9,2 ab
7	2	Cu-hydroxide 0,15% a.i. + Nu Film 17 0,02%	3,9 c
8	2	Cu-oxychloride 0,25% a.i. + Nu Film 17 0,02%	3,1 cd
9	3	Cu-oxychloride 0,25% a.i. + Nu Film 17 0,02%	3,6 c
10	2	Captafol 0,16% a.i. + Nu Film 17 0,02%	0,6 d
11	3	Captafol 0,16% a.i. + Nu Film 17 0,02%	0,7 d
12	2	Fosetyl-Al 0,3% a.i. + Nu Film 17 0,02%	11,4 a
13	-	Control	9,6 ab

Means with letters a,b, c and d differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 11. - The effect of fungicide treatments on Cercospora spot of Fuerte in 1980/81.

Treatment number	Time of applications	Treatments	Mean number of Cerc. spots/fruit (N=2 000)
1	Nov. 1980 Jan. 1981	Benomyl 0,025% a.i. + Nu Film 17 0,02%	6,3 b
2	Nov. 1980 Jan. 1981	Captafol 0,16% a.i. + Nu Film 17 0,02%	5,3 b
3	Nov. 1980 Jan. 1981	Captafol 0,16% a.i. + Nu Film 17 0,02% Benomyl 0,025% a.i. + Nu Film 17 0,02%	4,6 b
4	Nov. 1980 Jan. 1981	Captafol 0,16% a.i. + Nu Film 17 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 17 0,02%	7,5 b
5	Nov. 1980 Jan. 1981	Captafol 0,08% a.i. + Nu Film 17 0,02%	5,6 b
6	Nov. 1980 Jan. 1981	Captafol 0,08% a.i. + Nu Film 17 0,02% Benomyl 0,025% a.i. + Nu Film 17 0,02%	4,1 b
7	Nov. 1980 Jan. 1981	Captafol 0,08% a.i. + Nu Film 17 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 17 0,02%	4,5 b
8	-	Control	15,9 a

Means with letters a and b differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 12. - Control of Cercospora spot of Fuerte with two-spray treatments in 1981/82.

Treatment number	Time of applications	Treatments	Mean number of Cerc. spots/fruit (N=3 600)
1	Nov. 1981 Jan. 1982	Benomyl 0,025% a.i. + Nu Film 17 0,02%	0,9 b
2	Nov. 1981 Jan. 1982	Cu-oxychloride 0,25% a.i. + Nu Film 17 0,02%	0,4 b
3	Nov. 1981 Jan. 1982	Cu-hydroxide 0,23% a.i. + Nu Film 17 0,02%	0,4 b
4	Nov. 1981 Jan. 1982	Captan 0,1% a.i. + Nu Film 17 0,02%	2,4 ab
5	Nov. 1981 Jan. 1982	Prochloraz 0,04% a.i. + Nu Film 17 0,02%	1,0 b
6	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 17 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 17 0,02%	0,5 b
7	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 17 0,02% Cu-hydroxide 0,23% a.i. + Nu Film 17 0,02%	0,7 b
8	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 17 0,02% Captan 0,1% a.i. + Nu Film 17 0,02%	1,3 b
9	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 17 0,02% Cu-oxychl. 0,25% + Bitertanol 0,0125% + Agridex 0,1%	0,5 b
10	-	Control	3,2 a

Means with letters a and b differ statistically at 0,05 level (Duncan's multiple range test)

5 - DISCUSSION

Cercospora spot is an important disease of avocados in areas where high rainfall and favourable temperature are found (Stevens, 1922; Turu, 1969; Brodrick et al., 1974; Gustafson, 1976). It is present in most of the avocado growing areas of the Transvaal Lowveld in South Africa and is undoubtedly the most important pre-harvest avocado fruit disease at Westfalia Estate. Losses in export fruit are appreciable (up to 12%) even on commercially sprayed fruit (Table 1) and the disease is significantly more severe on unsprayed fruit (Tables 7, 8, 9, 10, 11 and 12), (up to 69%). Fuerte and Ryan cultivars are considerably more susceptible to infections by the pathogen than Edranol and Hass (Table 1). Significant correlations were found between losses on Fuerte and Ryan in relation to the amount of rain, the higher the rainfall, the more severe are the Cercospora spot losses (Figs. 1 and 2). Regression analysis between losses and harvest time revealed that there was a highly significant increase during the harvest period of Fuerte which decreased slightly towards the end of the dry winter season (Fig. 3). The increase in losses with late harvest was non-significant in the case of Ryan (Fig. 4).

The majority of the symptom descriptions (Stevens, 1922; Zentmyer, 1953; Brodrick et al., 1974) agree with observations made at Westfalia Estate. Differences from earlier descriptions of disease symptoms on Fuerte include the recognition of development stages of the disease. A characteristic swelling of the epidermal tissues at the infection site is the cause of the raised form which is the early development stage. It appears that this is the stage that Brodrick et al. (1974) described. Cercospora spot lesions become sunken in the later stage of disease development and probably these mature spot symptoms compare with those described by Zentmyer (1953). Cracks are formed only in older spots and they do not necessarily develop in lenticels. It is true, however, that the pathogen may infect the fruit through cracks in lenticels caused by other

factors.

The causal organism was identified as Pseudocercospora purpurea (Cke) Deighton and this was confirmed by taxonomists at the Commonwealth Mycological Institute in England. Typical conidia of P. purpurea were readily produced in fresh isolations on artificial media and on sterile avocado pieces. Cultures which became sterile, regained the ability to produce conidia on sterile avocado if kept under near UV light. This finding is in contrast to all previous reports on the in vitro sporulation of the fungus and it is the first reported case of inducing sporulation of P. purpurea under laboratory conditions. This is also the first confirmed record of the occurrence of Cercospora spot disease and of the fungus, Pseudocercospora purpurea in South Africa. The fact that the pathogen is a very slow growing organism which becomes sterile easily in cultures on artificial media is probably the reason why it was not previously identified and why the disease in South Africa is referred to as fruit spotting caused by a sterile fungus (Gorter, 1977). Black fruiting bodies found in older cultures of P. purpurea by Stevens (1922) were also observed in this study but differed from his observations in that these fruiting bodies sporulated and appeared to be spermogonia and not pycnidia. Their role in the Cercospora spot disease is unknown. There is no known sexual stage of the fungus and no evidence of its existence was found in this study. Since the pathogen can be isolated from fruit and leaf spots all year round in the conidial form, it appears to be independent of a sexual overwintering stage.

The disease is distributed throughout the tree on Fuerte, though there was a non-significant tendency for more Cercospora spots to occur on fruit from the northern and western aspects (Table 2). The incidence of the disease increased significantly with the increase in root rot severity of Fuerte trees (Fig. 5). This may point to the secondary invading behaviour of the pathogen. Fruit produced on trees with less foliage are more exposed to factors which enhance lenticel cracking and consequently a higher Cercospora spot infection may occur through the minute wounds in the cracks. The age

of spots has a marked influence on the success of isolation of the pathogen, it being much more easily recovered from young lesions (Table 3). The incidence of secondary invaders, mainly Colletotrichum gloeosporioides was also high in old spots. A similar phenomenon has been described by Kotzé (1963) and is well known in sub-tropical fruit. Although it is suggested in this study that sunken spots are older than raised spots, the success of recovery of P. purpurea from the two types of lesion of the same age on Fuerte fruit was nearly equal. However, the incidence of secondary organisms was again higher in sunken spots where cracks were present (Table 4). The above findings confirm the statements of Ruehle (1943b) and Zentmyer (1953) that anthracnose is often associated with Cercospora spot.

Spore trapping was attempted in this study for the first time in the history of Cercospora spot investigations. Virtually the whole first year's effort (1977/78 season) was confined to finding appropriate techniques and gaining sufficient experience in the identification of trapped conidia. Of the three stains tested Methylene Blue proved to be the most suitable. It stained conidia of P. purpurea by giving a moderate blueish cast which was more dense in the basal and apical cells of the conidia. Other Cercospora-like conidia caught in the spore trap stained differently and of course shape, measurements and the number of septae in the conidia were also necessary for the positive identification. A significant correlation was found between the number of conidia trapped weekly and the weekly rainfall and mean air temperature values, but the correlation was not significant if rainfall and temperature were analysed separately in relation to the conidia figures (Table 5). In Table 6, not only the multiple regression analysis of the daily number of conidia and temperature and rain correlated significantly, but also the correlations between the number of conidia and temperature (Fig. 6), relative humidity (Fig. 7) and rainfall (Fig. 8) respectively were also significant. The negative correlation of the air temperature to the number of conidia can be explained by the actual cooling effect of rain. High relative humidity is coupled to an increase in the number of

conidia and this again is a function of the rainfall. Relative humidity evidently plays an important role in the spore release mechanism of the fungus, since most conidia were caught in the early mornings when high humidity prevailed. Rain is the most important factor that influenced the production of P. purpurea conidia, showing the highest level of significance (Fig. 8) and repeatedly exhibited a significant effect on Cercospora spot incidence (Figs. 1 and 2). Equations were obtained which may be used to forecast the number of conidia expected to be produced and released into a given orchard's atmosphere and which can be used as an indication of high risk infection periods. This could be of value in the chemical control of the disease by enabling one to choose the optimum timing of the first spray. It is, however, of little importance in the timing of the follow-up sprays because the time period favourable for spore production is too long and there are many high risk periods at Westfalia Estate.

Experiments on the critical infection period by Cercospora spot disease firstly proved that long exposures of Fuerte fruit to natural infection resulted in a higher disease incidence (Fig. 9). Exposures on a monthly basis from November until March showed that a significantly more severe infection occurred on fruit exposed early in the summer season (Fig. 10). Poor correlation was found between rainfall and disease incidence in the monthly exposures, but the correlation was significant between rainfall and disease in the monthly cumulative exposures (Fig. 11). The same conclusion is made from correlations between disease incidence and spore trap results analysed on monthly and monthly cumulative basis (Fig. 12).

In a third experiment on the detection of the critical infection period, the monthly exposures again pointed to the importance of the time of the infection in disease incidence by being significantly more prominent on fruit exposed early in the growing season, provided infection is taking place (Fig. 13). Comparisons between the incidence of the disease and rainfall again proved that time between infection and symptom development plays a decisive role (Fig. 14).

With regard to the critical infection period, it can be concluded that Cercospora spot disease severity is determined by two major factors. Firstly, the high risk infection periods or the availability of conidia and weather conditions favourable for infection and secondly, the time or latent phase which must elapse between infection and symptom development. The latent phase appears to be about three months in duration as deducted from the above experiments and artificial inoculation experiments undertaken at Westfalia Estate to establish Koch's postulates. This also agrees with the statement of Stevens (1922) that late season's fruit is less susceptible to Cercospora spot disease.

In the preliminary chemical control experiments, benomyl gave the best results at a rate of 0,025 percent a.i. applied twice in the summer season (Table 7). Further experiments proved that two-spray benomyl treatments provided acceptable control and that the timing with November and January applications was near to optimum (Table 8). It is unclear why some of the treatments yielded unexpected results. For example, the three-spray application of benomyl in treatment No. 11 was found to be inferior to the two-spray treatment of No. 10 at the time of the first assessment.

It appears that sprays relatively early in the summer season (November) are more effective than late sprays in controlling the disease. This underlines earlier findings that the critical period for Cercospora spot disease is the early rainy season. The addition of the additive Nu Film 17 to benomyl was found to be insignificantly more effective than the mixing of Biofilm and Solvaid additives (Table 9). TBZ in the second spray of a benomyl treatment, aimed mainly at post-harvest disease control did not show efficacy against Cercospora spot disease. Etaconazole and propiconazole were ineffective. Benomyl with the commercially used Nu Film 17 and benomyl with Plyac and Solvaid additives were equally effective in the 1979/80 season's experiment (Table 10). The fungicide that gave best results was captafol, while benomyl, Cu-oxychloride and Cu-hydroxide also provided significant control compared with the unsprayed fruit. B 77, glyodin, bitertanol, PP 296 and fosetyl-Al were ineffective

The discovery of effective treatments with non-benzimidazole type fungicides led to the extensive testing of captafol and Cu-formulations. It was proved that the two-spray treatments of captafol at the 0,08 percent a.i. rate is as effective as the 0,16 percent a.i. concentration and that it gave good control both alone and in spray programmes with Cu-oxychloride (Table 11). This was confirmed again by results of the 1981/82 season's experiment (Table 12). Cu-oxychloride and Cu-hydroxide used alone and in spray programmes with captafol equalled the efficacy of benomyl treatments. There was a non-significantly higher disease incidence on prochloraz sprayed fruit and captan failed to control the disease. Bitertanol was included in the second spray of a captafol plus Cu-oxychloride spray programme (treatment No. 9) to investigate its effect on post-harvest diseases and it showed no control against Cercospora spot.

With regard to the chemical control of the disease at Westfalia Estate, it must be added that benomyl has been used exclusively for the past 10 years in most of the producing avocado orchards. This raised the question of resistance of P. purpurea to benomyl. The matter was not fully investigated from the resistance point of view, but the finding on the progressively poorer control by benomyl (Fig. 15) may suggest an increased tolerance of the fungus to the chemical. For this reason, Westfalia Estate has adopted the policy of alternating fungicides with different modes of action in controlling the disease. The chemicals presently recommended to replace benomyl under such conditions are captafol and the Cu-formulations.

PART TWO

POST-HARVEST DISEASES OF AVOCADOS

1 - INTRODUCTION

Post-harvest diseases occupy an important place in the group of avocado diseases troubling the South African export industry. The nature of all major post-harvest avocado diseases is that they appear on fruit which has started to soften. Thus, the development of visible disease symptoms usually occurs on fruit that is already on the overseas market. As long as fruit is hard, post-harvest disease infections remain latent, thus making their removal at the packhouse impossible. Even severely infected fruit may therefore be passed during inspection at the packhouse. This places a great responsibility on the grower to produce good quality fruit, free of the latent post-harvest disease infections.

The production of disease free fruit is particularly critical to export avocados which are transported by sea and are subjected to long periods of cold storage. Conditions during transport, such as temperatures, the atmosphere in containers, humidity, means of packing etc. have certain effects on the fruit and they also influence the development of pathological post-harvest diseases. Interaction between the various factors may occur so that interpretation of the outcome can be a very complex problem.

The pathological post-harvest diseases which are caused by fungi, were investigated in this study. On the basis of symptoms and organisms involved, pathological post-harvest diseases of avocados at Westfalia Estate may be divided into three groups: stem-end rot, anthracnose and Dothiorella/Colletotrichum complex fruit rot.

2 - LITERATURE REVIEW

2.1 STEM-END ROT

In California, stem-end rot is widely distributed, being as common in fruit from inland districts as from fruit from the coast (Horne, 1934).

Schiffmann-Nadel, Cohen and Arzee (1970) estimated losses by the disease of up to 20 percent in Israel. Stem-end rot is also one of the important avocado fruit diseases in Queensland, Australia (Peterson, 1978).

In South Africa, Jacobs (1974) stated that stem-end rot disease is not normally a serious problem on commercially stored avocados.

Horne (1931) described eye rot (stem-end rot) as a disease that can be caused by several fungi of common moulds which start growth at the stem wound and cause gradual decay of the fruit. He described the decayed part as often showing small cavities lined with white or variously coloured fungal hyphae.

Zentmyer (1953) was the first to use the descriptive name of stem-end rot for the disease.

In 1934, Horne identified Alternaria, Cladosporium and species of Fusarium occurring in stem-end rot of avocados in California. A year later, also from California, Dothiorella gregaria Sacc. (Botryosphaeria ribis chromogena Grossenbacher and Duggar) was reported to be associated with stem-end rot (Horne and Palmer, 1935). Zentmyer (1953) restated that Dothiorella gregaria (Botryosphaeria ribis) occasionally induces stem-end rot and he added that Diplodia and Phomopsis species are also involved in Florida.

Schiffmann-Nadel and Lattar (1958) found Diplodia natalensis Pole Evans most frequently causing stem-end rot in Israel.

Stem-end and fruit rot was shown by Joffe and Schiffmann-Nadel (1967) to be caused by Fusarium spp. amongst them F. solani, in Israeli avocados.

Muirhead (1977) identified stem-end rot fungi in

Australia and concluded that Dothiorella aromatica (Sacc.) Petr. and Syd. is the most common and that other fungi which can cause stem-end rot are Phomopsis perseae Zerova, Colletotrichum gloeosporioides (Penz.) Sacc. and Pestalotiopsis species. Peterson (1978) also in Australia, referred to Dothiorella aromatica as the main cause of stem-end rot.

In South Africa, Jacobs (1974) believed that stem-end rot is mainly due to a longer shelf-life, extended artificially by waxing or irradiation. In an index of South African plant pathogens, Gorter (1977) listed Botryodiplodia theobromae Pat. as the cause of avocado stem-end rot.

Schiffmann-Nadel et al. (1970) cultured Diplodia natalensis on PDA to produce conidia for stem-end rot inoculation studies.

Stem-end rot and fruit rot pathogens were isolated by Peterson (1978) as follows: avocado fruit with at least 30mm long peduncle attached was surface sterilised by immersion in 70 percent alcohol for 10 minutes and then allowed to dry. Sections of fruit were cultured on PDA and the incidence of fruit infecting fungi recorded.

Regarding the source of inoculum, stem-end rot pathogens reproduce in abundance on many above ground parts of the tree. Infected fruit which drop on the ground and mummify and also dry infected leaves and branches which drop or persist on the trees, are sources of inoculum for primary infection in nature by Colletotrichum gloeosporioides in stem-end rot, anthracnose and fruit rot (Ocfemia and Agati, 1925).

Horne and Palmer (1935) showed that when Dothiorella infected fruit becomes old and dark, the surface develops numerous small pimples at the tops of which minute spore masses appear as drops or coils. These spore masses emerge from pycnidia and similar structures are formed in the dead areas of tip-burned leaves and in the bark of dead twigs. During rainy or wet periods spores emerge from these pycnidia and are freely washed and spattered about. Spores of another sort are also formed which may be shot up to a distance of about 12mm. It is believed that spores on fallen leaves and twigs probably are not readily spread upwards in the tree.

Phomopsis perseae was also reported from dead avocado

leaves which may serve as an infection source (Zerova, 1940).

Avocado fruit was often found to be attacked by Diplodia natalensis through the stem of the fruit and it was observed that fruit harvested with short pedicels were severely rotted at softening, whereas the amount of rot was low in fruit picked with long pedicels (Shiffmann-Nadel, 1968).

In a follow-up investigation, Schiffmann-Nadel et al. (1970) established that while fruit having long pedicels soften before the beginning of fruit rot and escape infection, short pedicel fruit soften concomitantly with the development of rot.

Anatomical studies by Arzee, Cohen and Schiffmann-Nadel (1970) revealed that mycelium of D. natalensis was present in most tissues of the pedicel, but the conducting vessel elements seemed to serve as the main route for fungal penetration. In the vessels the mycelium advances via perforation plates and to some extent through pits.

Schiffmann-Nadel et al. (1970) observed a faster growth rate for the mycelia of D. natalensis in the pedicel of avocado fruit which was inoculated late in the season.

Peterson (1978) found that Fuerte is susceptible to infections by stem-end rot fungi at all stages of fruit development.

Zauberman, Schiffmann-Nadel, Fuchs and Yanko (1974) reported that five pre-harvest sprays with Cu-sulphate or TBZ during the growing season controlled stem-end rot caused by Colletotrichum and Diplodia.

Horne (1931) anticipated that good results would probably be obtained in controlling stem-end rot by dipping the cut stems of avocado fruit in alcohol and in melted paraffin.

Muirhead (1977) referred to unpublished results of Burden and Stone, according to which 2,4-D dips delayed ripening and increased the incidence of stem-end rot on Fuerte, even though similar treatment of citrus reduced stem-end rot.

Descriptions are given below for the five most

commonly encountered stem-end rot pathogens at Westfalia Estate.

Thyronectria pseudotrichia (Schw.) Seeler. It is generally seen in its conidial stage, Stilbella cinnabarina (Mont.) Wollenw., where coremia are single or in groups of 2 - 6 form basal subiculum. Coremia are orange-red to dark brown at the base changing to straw colour higher up and they measure 150 - 300 µm in length. Conidia of 4 - 7 x 2 - 3 µm are formed in the globular head which, when dry, with its mass of straw coloured conidia measures 125 - 500 µm in diameter. The name Thyronectria pseudotrichia is designated as the sexual stage of the fungus. Perithecia usually develop in cespitose clusters of 3 to 20 or more and they measure 200 - 590 µm in diameter. Their colour is bright orange-red, weathering to dark brown and finally almost black. Perithecia are erumpent through outer bark, collapsing and pezizoid when dry. Asci are clavate when young, tapering towards the apex, later broad, closely following contours of the spores, finally avenescent and measure 50 - 100 x 10 - 25 µm. No true paraphyses are found in the perithecium, but pseudoparaphyses pendent from roof. There are approximately 8 ascospores in an ascus. Ascospores are muriform, broadly bulging ellipsoid, sometimes curved and slightly tapered terminally, their colour is hyaline to pale yellow or light brown. The basically 3-septate ascospores are constricted with many other, often conspicious, transverse and longitudinal septae and they measure 15 - 40 x 7 - 15 µm. The above description was furnished by Seeler (1940).

Colletotrichum gloeosporioides (Penz.) Sacc. A detailed morphological description of the fungus from avocados was given by Ocfemia et al. (1925). The mycelium of the imperfect stage grows abundantly in pure cultures. The septate hyphae are hyaline when young, but become compact and dark in colour with age. The acervuli of the fungus are pustular. They are frequently arranged concentrically, both in artificial culture and on the host tissues. The acervuli are moist and pink at first due to the mass of conidia, but they turn dark later. Black sclerotia-like

bodies of irregular shape are formed around the acervuli in cultures. Conidia are thin walled with granular contents, hyaline, ellipsoid to elongate, rounded at the ends. Conidia produced in the field vary from 12,5 - 19,5 x 3,5 - 5,5 µm. Perithecia of the sexual stage Glomerella cingulata (Ston.) Spauld. and Schrenk are elongate or pear-shaped, dark brown, shortly rostrate and more or less hairy. Each ascus contains 8 hyaline, elliptic and slightly curved ascospores which measure 12,5 - 19,5 x 4,5 - 5,0 µm.

Dothiorella aromatica (Sacc.) Petr. and Syd. Pycnidia are produced sub-epidermally in groups or scattered, often covering large areas of the leaves. The lower halves of the pycnidia are embedded in the mesophyll and the upper halves are at first covered by the epidermis, but erumpent later. They are usually round or irregular in shape and measure 200 - 300 µm in diameter with a flat, indistinct ostiole which opens through an irregular round pore. Pycnidial walls usually measure between 15 - 25 µm, but in some cases are up to 30 µm thick consisting of several layers of indistinct and irregularly shaped dark cells of 5 - 8 µm in size. Conidia are oblong or spindle shaped at both ends being more pointed on the side of the attachment. They are straight or slightly curved, one celled, hyaline with indistinct fine grained plasma and measuring 16 - 23 x 5 - 7 µm. Conidiophores are simple, pointed at the end with an average length of 6 - 16 µm, seldom up to 20 µm and 2 µm wide at the base. This description of the fungus was given by Petrak and Sydow (1927) from specimens found on dry avocado leaves originating from the island of Malta.

Phomopsis perseae Zerova. A brief Russian and Latin description was given by Zerova (1940) to the species which he isolated from leaves of Persea gratissima (americana). He proved in glasshouse inoculation studies that the fungus is pathogenic to avocados. Pycnidia are produced under the epidermis, they become erumpent later and their colour slowly turns black. Pycnidia measure 400 - 500 x 200 - 225 µm

and they consist of one, seldom two or three cavities, the upper wall is thick, while the lower wall is thin, first yellow or indistinct, tapering with a round pore on top. Conidiophores are indistinct and conidia are fusiform with two lipid fragments at the two ends measuring 7 - 10,2 x 2,3 - 2,5 µm.

Fusarium decemcellulare Brick. A thorough description of the fungus was published by Booth and Waterston (1964). Cultures of the conidial form are initially pale with white to cream floccose mycelium. After a few days a rose pigmentation appears and this becomes darker with age. Microconidia are formed in chains, they are ellipsoid and measure 10 - 12 x 3 - 4 µm with a flat circular scar at the base on each end. Macroconidia formed typically on sporodochia where they give rise to characteristic wedge-shaped mass. Macroconidia measure 50 - 65 x 5 - 7 µm and they are 7 - 10 septate, cylindrical, curved and narrowing apically to a point. Diurnal fluctuations of light and temperature with an optimum of 12 hours light and 12 hours darkness and 25°C to 30°C favour sporulation in culture. Perithecia of the sexual stage, Calonectria rigidiuscula (Berk. and Br.) Sacc. are formed on the surface of stroma which arise from below the periderm of the host. Perithecia are globose, cream to yellow in colour, roughly warted, 200 - 300 µm high and 190 - 300 µm in diameter. Asci clavate, generally 4-spored and measuring 70 - 100 x 12 - 14 µm. Ascospores are hyaline, ellipsoid to reniform with 3 transverse septae and faint longitudinal striations when mature, they measure 22 - 28 x 7 - 10 µm. C. rigidiuscula is bisexual and self-fertile and perithecia are occasionally formed in culture from single ascospores or single conidia.

2.2 ANTHRACNOSE

Rorer (1919) reported that anthracnose is the most serious disease of the avocado in Trinidad and Tobago.

Stevens (1922) wrote that anthracnose was first brought to his attention in 1917 in Florida. In previous years some growers had observed it, but as the years passed the disease

became more prevalent and caused serious losses to the crop when it was well established. In some cases 90 percent or more of the fruit on a tree developed symptoms of the disease and most of the infected fruit was unfit for marketing.

In California, anthracnose is one of the most frequent rots (Horne, 1934). Export losses due to this disease have at times reached 80 percent in South Africa (Jacobs, 1974).

Brodrick et al. (1974) reported cases where losses as high as 70 percent was experienced following a very wet summer.

Ocfemia and Agati (1925) described the disease from leaves, twigs, blossoms and fruit of the avocado. The fungus produces a rusty blight on the foliage, often starting from the margin. From this point the leaves turn rusty brown. Infected leaves fall prematurely. Succulent young branches are attacked at any point and irregular brown to purplish coloured lesions are formed on them. On blossoms the disease causes reddish brown discolouration. The colour turns dark brown later. Infected flowers die and fall off. The fungus infects peduncles of the fruit causing the dropping off of young fruit. Fruit infection has been found to cause die-back of twigs due to the mycelium entering the twigs by way of the peduncles. On fruit the fungus produces brown spots of varying sizes. These spots increase in size, coalesce, gradually covering the entire fruit and becoming sunken. Infected fruit fall and are finally covered with bright coloured moist masses of conidia of the fungus.

Stevens (1922) also described symptoms of anthracnose at length. The infection appears as definite spots scattered over the surface of the fruit. These spots are round, brown to dark brown or black in colour and vary from 3 to 12mm in diameter. They are composed of hard, dry, corky tissue which penetrates the skin of the fruit into the flesh. The surface of the spot is slightly sunken, often cracked or fissured and in some cases a zonated effect is observed. When once formed, the spots do not appear to increase in size on the surface of the skin, but a decay of the flesh below may follow, particularly in mature fruit. Affected fruit may show a few or many spots of various sizes. Frequently, spots merge to

form irregular patches, the surfaces of which are deeply cracked or broken. Severe attacks on fruit that is not fully matured may misshape or dwarf them. Spots also appear in the bark of young shoots and on fruit stems and are somewhat similar to the spots on fruit. Infections on the fruit stems generally appear some time in advance of those on the fruit.

According to Horne (1934) the disease on green coloured fruit begins as a dark area, while on dark coloured cultivars the colour tends to become somewhat lighter. In early stages it resembles Dothiorella rot. With the advance of the decay in a moist atmosphere minute pustules develop on which pink spore masses appear. In a moderately humid environment the spore masses spread out in a pink layer, but with more moisture the colour is obscured. Anthracnose rot of avocados advances more slowly on the surface of the fruit than Dothiorella rot, but it grows deeper.

Zentmyer (1953) characterised anthracnose as sunken black spots on the fruit. The spots are nearly circular in outline. As the fruit ripens, the fungus invades the flesh to a greater degree until most of the fruit is rotted.

The causal agent for anthracnose was described by Higgins (1911) and Higgins, Hunn and Holt (1911) under the generic name of Gloeosporium.

In his study on avocado diseases, Stevens (1922) reported it as a Colletotrichum species.

Horne (1934) described anthracnose from California and identified the pathogen as Colletotrichum gloeosporioides Penz.

In Australia, Simmonds (1966) gave Colletotrichum gloeosporioides Penz. var. minor Simmonds and Colletotrichum acutatum Simmonds as the causes of fruit rots of avocados in the Queensland area.

In South Africa, Doidge (1924) found C. gloeosporioides for the first time on avocados.

Description of the pathogen, Colletotrichum gloeosporioides (Penz.) Sacc. is given in 2.1 of Part Two.

Ocfemia and Agati (1925) made single spore cultures from ascospores of Glomerella of avocados on Cornmeal Agar in Petri dishes. Their study on the growth of the fungus included the use of common media and sterilised plant tissues. Sterilised unpolished hulled rice was prepared by putting approximately the same amount of rice grains in test tubes and covering them with water about three cm deep. The test tubes were plugged and sterilised in an autoclave. The sterilised plant tissues were prepared by making five cm cuttings of the materials used. Moist absorbent cotton was placed at the bottom of the test tubes before the cuttings were introduced. The test tubes were then plugged and autoclaved. They summarised the results obtained on the various media, some of which are presented below. Water Agar: the fungus produced slow and poor growth, with little mycelium. Masses of conidia appeared 12 days after inoculation. Cornmeal Agar: growth was slow, mycelium scanty, but an abundance of pinkish masses of conidia was produced. Acervuli were setose and perithecia-like bodies were noted occasionally with no asci in them. Potato Dextrose Agar: the fungus produced very good growth. Abundant mycelium covered the entire surface of the slant in four to six days. Pinkish masses of conidia were produced abundantly in 8 to 11 days. Sterilised avocado stem: abundant mycelial growth was observed with large numbers of conidia.

Binyamini and Schiffmann-Nadel (1972) produced conidia of C. gloeosporioides for their inoculation studies on avocado fruit.

From results obtained in several inoculations by Stevens (1922) the fungus appeared to be a secondary invader, unable to penetrate the epidermis of avocados except through wounds. He induced typical anthracnose spots by introducing the fungus into punctures made on the fruit.

Zentmyer (1953) shared this opinion and wrote that Colletotrichum gloeosporioides is unable to enter unwounded fruit and it usually becomes established in lesions caused

by Cercospora spot disease or Sphaceloma.

Horne (1926a) stated that penetration of avocado fruit by the fungus is possible without wounds and that it penetrates the rind as well as the flesh.

Wardlaw, Baker and Crowdy (1939) isolated C. gloeosporioides from the skin of uninjured hard avocado fruit, suggesting that the pathogen may be present in the fruit in a latent from.

Ruehle (1943b) reported that the anthracnose fungus is unable to grow actively in healthy, uninjured, growing fruit, but may establish latent infections on them. The sites of latent infections are in the lenticels. Latent infections remain inactive until the fruit is developing and begin to grow actively when fruit reaches maturity and starts to soften. Latent infections are generally of little importance on fruit harvested at the right age and maturity and handled properly until it reaches the consumer. The pathogen readily establishes itself in an active condition in developing avocado fruit as it approaches maturity, through dead areas of cracks in the rind caused by other fungi or through mechanical injuries. Cercospora spot is the most common avenue of entrance.

The presence of latent anthracnose infection in avocado fruit was demonstrated by Binyamini and Schiffmann-Nadel (1972). Anatomical studies of artificially infected fruit showed appressoria of the fungus on the fruit while still on the tree and after picking until softening. The appearance of decaying spots on fruit that had been inoculated as much as three months prior to harvest, but showed no visible infection symptoms in the meantime, was further evidence of latent infection. The germ tube of the fungal conidia penetrated the wax layer and formed appressoria. During fruit softening, the appressoria germinated and this infective hyphae penetrated into the fruit. No wounds were needed for the infection to take place. They also suggested that hard avocado fruit may contain some inhibiting substances as indicated by unsuccessful artificial inoculations. Prusky (1981) isolated

an anti-fungal compound (diene) which inhibits the development of anthracnose in hard fruit and he showed that the breakdown of the compound occurs naturally as the fruit softens.

Field observations and laboratory examinations by Ocfemia and Agati (1925) showed that the mycelium of the fungus in the leaves enters the branches through the petioles. This type of infection causes die-back of twigs, sometimes resulting in total destruction of branches and in severe cases may involve the entire tree.

Stevens (1922) found that the period during which the fruit may become infected is perhaps comparatively short and lies between the time that the fruit is one-third to two-thirds mature. Young fruit shows no indication of the disease and spotting is rarely observed until after the fruit is half mature. As the fruit nears maturity it shows less tendency to spot. Most of the spots that appear on infected fruit are evident by the time the fruit is half grown.

Binyamini and Schiffmann-Nadel (1972) demonstrated by artificial inoculations that latent anthracnose infections occurred at all stages of fruit development (fruit from two cm in length to fully grown).

According to Peterson (1978) infections by C. gloeosporioides on Fuerte can take place at any stage of fruit development. The time of infection varied with the season and was closely linked to rainfall.

Stevens (1922) described severe anthracnose attacks on seedling trees and he also observed it on the Trapp cultivar.

Ocfemia and Agati (1925) recorded anthracnose in the nursery and found that the Mexican avocado type had 100 percent infection, while the West Indian type produced about 10 percent infected seedlings.

All cultivars grown in Florida are subjected to attacks by the fungus and the moderately susceptible cultivars are Collinson, Pollock and Fuchsia (Ruehle, 1943b).

In Australia, Allen (1977) found that Fuerte and Nabal are prone to severe losses caused by C. gloeosporioides,

whereas Hazzard and Sharwil are not nearly so susceptible.

In South Africa, Fuerte is exceptionally susceptible to anthracnose and losses due to this disease are usually high (Jacobs, 1974).

Stevens (1922) found that for the control of anthracnose no bloom spray is needed and that two or three timely pre-harvest applications of Bordeaux mixture is sufficient to check the disease. He stated that the first application should be made three or four weeks after the bloom has disappeared. A second application should follow from three weeks to a month later and possibly a third application three weeks after the second.

Copper fungicides, primarily aimed at Cercospora spot control were also demonstrated to be effective against anthracnose (Ruehle, 1943b).

McMillan (1976) recommended monthly field sprays to control anthracnose with benomyl.

Practical problems regarding anthracnose control in the field were discussed by Allen (1977) in Australia. He remarked that copper sprays at monthly intervals from flowering to harvest yielded promising results at one place, but the same treatment was unsatisfactory at another locality.

The spraying of copper fungicides at 14 and 28 day intervals from fruit set until harvest controlled avocado anthracnose and furthermore benomyl at 14 day intervals was also effective (Peterson and Inch, 1980).

Techniques for the evaluation of fungicides for the control of anthracnose of avocados were discussed by Kotzé and Kuschke (1979).

Kotzé, Kuschke and Durand (1981) evaluated fungicides in pre-harvest field sprays for the control of anthracnose and found that benomyl, captafol and Cu-oxychloride reduced disease incidence.

Brodrick (1978) outlined the technical requirements for experiments to evaluate fungicides against anthracnose and recommended methods for data collection.

On the post-harvest control of anthracnose, Brodrick et al. (1974) reported that dip treatments using hot water or fungicides failed to give satisfactory results.

Muirhead (1974) tested chemicals as post-harvest dips and concluded that benomyl was ineffective. This was thought to be due to the thickness of the cuticle rather than to resistance of C. gloeosporioides to benomyl. The post-harvest hot water-benomyl dip treatment proved to be also ineffective against the disease (Muirhead, 1976).

Muirhead (1974) also cited unpublished results of Burden and Stone, in which TBZ, captan, nabam and Na-salicylanilide were tested in a dip against anthracnose. None of these chemicals reduced the disease.

2.3 DOTHIORELLA FRUIT ROT

Dothiorella fruit rot occurs throughout California, but causes serious losses only in the moist coastal regions (Horne, 1934).

Horne and Palmer (1935) found that it is not widespread, and that it is not a rapidly destructive form of spoilage. However, at times serious losses occurred in the affected districts both by loss of fruit and through discrimination by buyers against fruit from certain districts, regardless of whether the fruit itself was infected or not.

According to Horne and Palmer (1935) the disease does not develop on fruit still hanging on the tree or on fruit after it is picked until softening begins. On Fuerte with first detectable softening, small, dark spots with vague boundaries appear. Not all Dothiorella spots develop at this stage, but new areas continue to make their appearance as softening progresses. Spots may first become visible to the naked eye when about three mm in diameter. Small spots are light umber, rather vaguely defined and not sunken or distinctly marked. As the spots become more distinct they darken slightly. Later spots spread rapidly, becoming somewhat sunken, while a watery rot spreads rather slowly into the flesh. An unpleasant odour develops. The surface settles

and becomes uneven. Later the whole fruit shrivels up and becomes dark. At a wound or at the stem attachment Dothiorella rot may appear earlier and penetrate deeper than on other parts of the fruit.

Horne (1934) stated that where fruit is taken from the tree in perfect condition and taken at the earliest permissible stage of maturity, Dothiorella rot appears to develop at a later stage in softening than with fruit held on the tree long after attaining adequate maturity for utilisation.

Muirhead (1977) found discrete lesions caused by Dothiorella which were similar to anthracnose and difficult to identify on the basis of symptoms only.

Dothiorella fruit rot was first reported from California by Horne (1931). The pathogen was subsequently identified as Botryosphaeria ribis chromogena (Dothiorella gregaria) (Horne and Palmer, 1935).

Four years earlier, however, a new Dothiorella species was described from avocado leaves by Petrak and Sydow (1927) and it was named D. aromatica.

Muirhead (1977) and Peterson (1978) found D. aromatica to be the main cause of stem-end rot and regarded it as an important fruit rotting pathogen.

In South Africa, Gorter (1977) pointed out that Botryosphaeria ribis is the responsible organism for a type of avocado fruit spotting disease.

The description of Dothiorella aromatica (Sacc.) Petr. and Syd. is found in 2.1 of Part Two.

Horne and Palmer (1935) studied the mode of infection by the fungus and found that once a spore is deposited on the moist fruit it germinates by sending out a tube-like thread which grows into the lenticel and penetrates the skin. The fungus makes considerable growth in the lenticel, but is unable to attack and destroy the living skin until the time of softening of the fruit. The latent infection through lenticels does not take place until some time after a fruit diameter of about 37 mm has been reached.

Dothiorella fruit rot is a particularly common disease

on Fuerte in California (McLean, 1931).

On the pre-harvest chemical control of the disease, Horne (1932) wrote that repeated sprays with Bordeaux mixture evidently restrained the disease. A single spray was not sufficient to check it effectively and Bordeaux dust was not promising.

Palmer (1932) in an avocado orchard where unsprayed fruit showed 80 percent infection, achieved 0 to 28 percent fruit infection with a Bordeaux mixture spray programme.

A very comprehensive spray experiment was conducted by Horne and Palmer (1935) for four years. They found that Dothiorella fruit rot can be effectively controlled by the use of fungicides and made the following statements: Liquid fungicides are more effective than dust formulations, copper sulphate is more effective than zinc sulphate and the fungicidal value of copper sulphate when used as Bordeaux mixture is greatly increased by the addition of sulphur. Their recommendation for control of the disease was a spray of Bordeaux mixture, wettable sulphur and spreader applied first when the fruit is about 37mm in diameter and sprayed again about two months later.

Dipping avocado fruit in various chemicals and in hot water for the post-harvest control of Dothiorella fruit rot were ineffective (Horne, 1931).

The effect of post-harvest waxing and cellophane wrapping of avocado fruit was investigated by Kotzé and Kuschke (1978).

3 - MATERIALS AND METHODS

Losses caused by post-harvest diseases to avocados at Westfalia Estate were monitored for two full seasons during 1978 and 1979. Samples from the commercially picked fruit were analysed. In each season fruit was sprayed twice with benomyl at 0,025% a.i. with 0,02% Nu Film 17 additive, in mid-November and mid-January, but received no post-harvest treatments. Fruit was wrapped in cellophane, packed in cardboard cartons, cold stored at 6°C for 28 days and then ripened at room temperature.

Fruit in the cartons was checked daily and evaluated for post-harvest diseases as soon as it reached the eating-ripe stage. Each fruit was first assessed for external disease symptoms and then cut longitudinally and assessed for internal symptoms. The extent of losses was measured by a rating scale of 0 to 10, where 0 represented uninfected fruit and 10 meant that the entire fruit was affected by the disease. This cold storage temperature and period and evaluation technique has been used as a standard in all post-harvest disease analyses described in this study. The oil content of the fruit was determined according to the method of Swarts (1976).

Symptoms of post-harvest diseases were studied on artificially inoculated fruit that was not exposed to natural infections. Fuerte fruit was closed in brown paper bags soon after fruit set in October, 1978, before the onset of the natural infection period. This fruit was inoculated by applying conidial suspensions of the various pathogens with an atomizer in November and December, 1978 and also in January, 1979. Immediately after inoculation, fruit was enclosed in polyethylene bags for five days to promote successful infection after which the polyethylene bags were removed and the fruit was again closed in paper bags to prevent further contaminating infections.

Fruit was harvested in April, 1979, ripened at ambient temperature and carefully evaluated for the post-harvest diseases which developed on them and isolations were made from infected tissues to establish the pathogens involved in the disease.

The isolation of fungi from post-harvest diseases was made on PDA and subsequent culturing for spore production was done on sterile avocado fruit pieces under axenic conditions. To induce profuse sporulation, cultures were kept under continuous near UV light.

The identity of all post-harvest pathogens used in this study was tentatively determined at Westfalia Estate and then sent for confirmation to the Commonwealth Mycological Institute in England.

The incidence of post-harvest diseases in relation to root rot severity symptoms of Fuerte trees was investigated by harvesting 300 fruits from each tree with a disease severity rating of 0 to 7 at block 34A of Westfalia Section. The occurrence of post-harvest diseases in respect to the position of fruit on Fuerte trees was also studied. An average of 75 fruits were picked from each aspect of the four test trees at the commercially sprayed block 34A of Westfalia Section on 26 May, 1982.

Observations were made on the infection source of these pathogens in commercial avocado orchards at Westfalia Estate.

The occurrence of various stem-end rot fungi and the effect of cold storage on their incidence was investigated on Fuerte fruit harvested in block 34A of Westfalia Section on 16 March,1978. The number of fruits in the experiment was 700, and these were divided into two groups. The one group was ripened at ambient temperature directly after harvest and the other group went through 28 days cold storage at 6°C to simulate export conditions after which fruits were ripened at ambient temperature. Fruit with stem-end rot symptoms was cut open and isolations were made on PDA to determine the causal organisms. The occurrence of stem-end rot fungi in Edranol fruit was also examined.

The pathogenicity of the fungi involved in post-harvest diseases as stem-end rot agents was tested in artificial post-harvest inoculations on four avocado cultivars. Inoculations were made with spore suspensions on the stem scar of debuttoned fruit and through the pedicel cut to 0,5cm in length. The extent of stem-end rot damage was evaluated

externally and internally on fruit ripened at ambient temperature 12 days after inoculation.

The pathogenicity of some of the more important stem-end rot fungi as well as Fusarium culmorum (Smith) Sacc. was tested in post-harvest inoculations to deterimine their virulence as fruit rotting agents through wounds. Inoculations were made to wounds inflicted by a sterile blade. Wounds extended just below the epidermis at the widest diameter of the fruit. Spore suspensions of the various fungi were administered into the wounds after which the inoculation sites were closed with a piece of masking tape and fruit was ripened at ambient temperature. The extent of damage was measured 11 days after inoculation by the diameter of the rot. F. culmorum was isolated earlier from a post-harvest fruit spot of Edranol. The oil content of the fruit used in this experiment was: Fuerte 12,2 percent, Edranol 10,2 percent, Hass 14,0 percent and Ryan 7,1 percent. There were 24 fruits in each pathogen/cultivar combination.

The critical infection periods for fruit infection by post-harvest pathogens were investigated on Fuerte by exposing the fruit to natural infection under orchard conditions. These experiments were done in conjunction with investigations on the critical infection period of Cercospora spot disease. Fruit was closed in brown paper bags on four trees selected at random at block 35 of Westfalia Section. The number of fruits used in the experiment was 800. Initially all fruits were bagged and later exposed to natural infection according to a time schedule and were then closed again. Fruit remained in the paper bags until harvest. Fruit was harvested on 20 June, 1978, wrapped in cellophane, packed in cartons and cold stored for 28 days at 6°C. Following cold storage, fruit was ripened at ambient temperature and evaluated for post-harvest diseases as soon as they reached the eating-ripe stage. A similar fruit exposing experiment was laid out in 1978/79 at block 34A of Westfalia Section. Fruit exposure started in November and continued until March on monthly and monthly cumulative basis. Fruit was harvested on 5 June, 1979.

An experiment, based on the techniques used by Peterson

(1978) was undertaken in the 1980/81 season. Six Fuerte trees were selected at random from block 14 of Westfalia Section. Ten fruits were picked from each tree at monthly intervals from November until April. Fruit was surface sterilised by immersion in absolute ethanol for one minute and was then allowed to dry. Fruit pedicels were sliced in one mm thick pieces and five of these round cross sections of the pedicel were placed on PDA in each Petri dish. Samples of the skin were taken by an eight mm diameter cork borer from five evenly spaced positions on the widest diameter perimeter of fruit. Skin samples were also cut in one mm thick pieces and with the epidermis side up, placed on PDA. Fungi isolated out of these pedicel and skin pieces were then identified and the frequency of the important species was recorded.

The incidence of post-harvest diseases on fruit sprayed pre-harvest with various fungicides was analysed. The first experiment on the pre-harvest control of post-harvest diseases was done in 1976/77. Four single tree replicates selected at random from Fuerte block 34A of Westfalia Section were sprayed twice with either benomyl, thiophanate-methyl or fosetyl-Al. The first spray was applied on 19 November, 1976 and the second on 3 February, 1977. Fruit was harvested on 22 April, 1977, cold stored for 28 days and evaluated for post-harvest diseases as described earlier. In the 1979/80 season, the experiment included the testing of copper fungicides, captafol, benomyl, fosetyl-Al and a number of new fungicides as well as various additives. The site of the experiment was block 34A of Westfalia Section. Eight trees were used as replicates in each treatment and there were two sprays in each treatment, the first in mid-November and the second in mid-January, but in treatment numbers 9 and 11 a third spray was applied in mid-December. Fruit was harvested on 3 May, 1980 and evaluated for post-harvest diseases after cold storage.

The 1980/81 season's pre-harvest fungidical spray experiment for the control of post-harvest diseases was conducted again at block 34A of Westfalia Section. Eight randomly selected Fuerte trees were used in each treatment and trees were sprayed twice in the growing season.

Fungicides tested in the experiment included benomyl, captafol and Cu-oxychloride. An average of 250 fruits were harvested on 14 April and 14 May, 1981 and evaluated for post-harvest diseases after cold storage.

The site of the pre-harvest fungicidal spray experiment in 1981/82 was at block 34B of Westfalia Estate, where six randomly selected Fuerte trees, six years of age, were used in each treatment. The following fungicides were tested: benomyl, captafol, Cu-oxychloride, Cu-hydroxide and prochloraz. An average of 360 fruits were harvested on 8 April 1982 and evaluated for post-harvest diseases after a 28 day cold storage period.

The effect of the length of the ripening time, that is, the time between harvest and eating ripe stage, on the incidence of post-harvest diseases of Fuerte was investigated in 1982. The site of the experiment was block 16B of Westfalia Section, where 16 trees were selected at random. The trees received no pre-harvest fungicide treatment. A total of 330 fruits were harvested from the selected trees on 20 April, 1982, wrapped in cellophane (EX 906778, 345 QMS) and packed in 26 cartons and cold stored for 28 days and then ripened at ambient temperature. The ripening time was determined by the number of days which elapsed between the date when fruits were taken out of cold store and the date when they reached eating ripe stage. The mean post-harvest disease severity was recorded for each carton and this was correlated to the ripening time.

For the experiment on the effect of the removal of the fruit pedicel on post-harvest diseases, 260 Fuerte fruits were harvested on 27 March, 1979 at block 34A of Westfalia Section. Half of the fruit was debuttoned after picking, still in the orchard and was then wrapped in cellophane and packed in cartons. The other half of the experiment fruit was packed with a 0,5cm long pedicel. Results were evaluated after a 28 days cold storage period.

The effect of rain on post-harvest diseases was investigated by picking Fuerte fruit at block 35 of Westfalia Section from commerically sprayed trees on 24 May 1978.

The first batch was harvested in the morning, while the fruit was still wet from the previous day's rain and a second batch was harvested from the same trees later the same day, when the fruit was dry. Fruit was passed through the commercially used wind-tunnel in which each fruit is exposed to ventilated air drying for about three minutes. The drying eliminated all visible water from the surface of the fruit. Fruit was wrapped in cellophane, packed in cartons and cold stored for 28 days.

Fuerte fruit intended for an experiment to determine the effect of washing, was harvested from block 35 of Westfalia Section on 24 May, 1978. The treatment of fruit consisted of washing it in tap water for about one minute, then drying it in the drying tunnel and packing and cold storing the fruit for 28 days.

Fuerte fruit harvested at block 34 A of Westfalia Section on 18 April, 1980 was used for the experiment to investigate the effect of moisture condensated on the fruit. Some of the fuit was put into pre-cooling at 10°C and kept there for eight hours to induce condensation. The wet fruit along with the control fruit, was put through the drying tunnel for rapid drying. It was then wrapped in cellophane and packed in cartons and cold stored for 28 days.

The waxing of fruit and the sealing of the cut end of the pedicel with wax plus benomyl 0,1 percent a.i., plus TBZ 0,2 percent a.i. was done on Fuerte fruit harvested on 26 April, 1979 at block 34A of Westfalia Section. The sealing of the pedicel took place immediately after picking, in the orchard, while the waxing was done at the packhouse. Fruit was wrapped in cellophane and cold stored for 28 days. During the evaluation of the experiment isolations were made from fruit showing stem-end rot symptoms to determine the pathogens involved.

Fuerte fruit harvested at block 36 of Evenrond Section on 26 May, 1980 was used for testing the effect of cellophane wrapping. Half of the fruits were wrapped in cellophane and along with the unwrapped fruit, were packed in cartons and cold stored for 28 days.

The effect of TAG wax applied by means of roller brushes was investigated on Fuerte fruit harvested from block 26 of Westfalia Section on 17 May, 1982. Fruit was air dried after waxing and packed in cartons and cold stored for 28 days.

Fuerte harvested from block 34A of Westfalia Section on 19 May, 1982 was used in an experiment to study the effect of waxing and the addition of benomyl 0,05 percent a.i. plus TBZ 0,2 percent a.i. in the wax solution. The wax and wax plus fungicide were applied by means of roller brushes and it was followed by air drying in the drying tunnel. Fruit was packed in cartons and cold stored for 28 days.

4 - RESULTS

4.1 LOSSES CAUSED BY POST-HARVEST DISEASES AT WESTFALIA ESTATE

Losses caused by the various post-harvest diseases at Westfalia Estate were investigated on commercially sprayed Fuerte fruit and the extent of damage was correlated to the maturity in terms of oil content of the fruit (Figs. 16, 17 and 18).

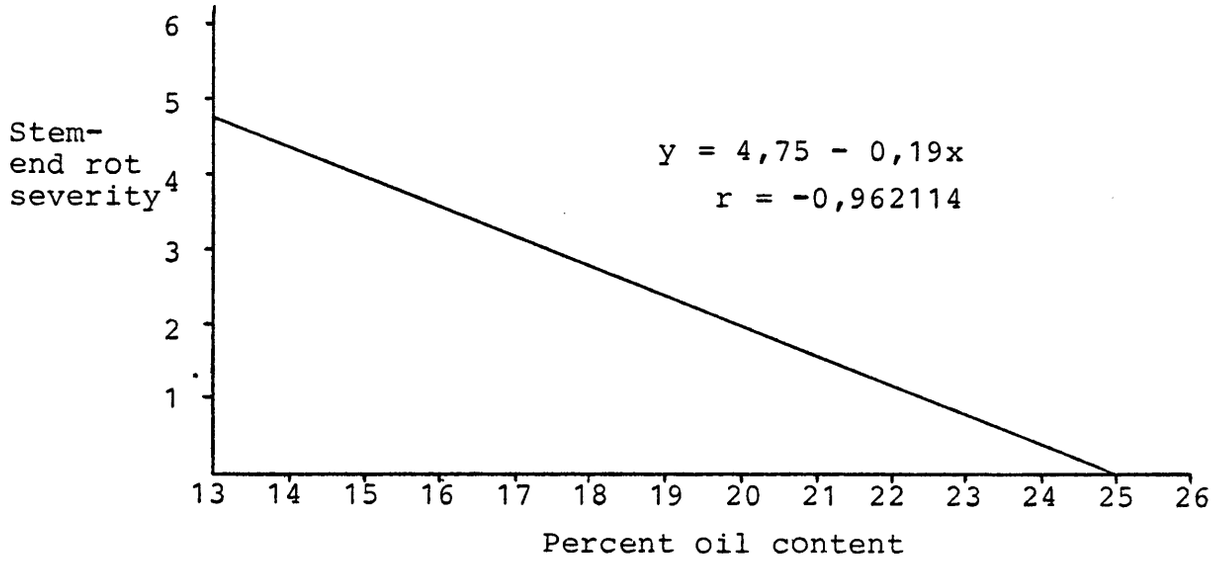

FIG. 16. - The severity of stem-end rot damage to Fuerte in relation to the oil content of the fruit in 1978 and 1979.

A highly significant correlation of r = -0,962114 was found between stem-end rot severity and the maturity, as indicated by the oil content of the fruit, which is described by the linear regression model of y = 4,75 - 0,19x. According to the model, stem-end rot severity decreases with the increase in oil content of the fruit. The oil content of avocado fruit correlates with the maturity of the fruit and it increases as the season progresses.

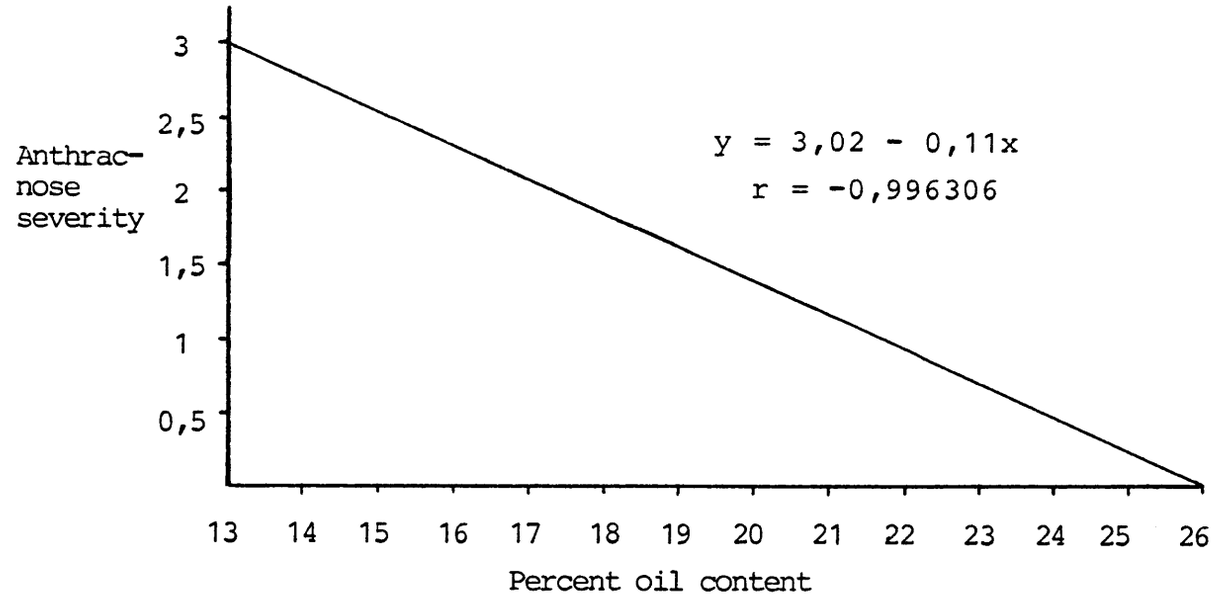

FIG. 17. - The severity of anthracnose damage to Fuerte in relation to the oil content of the fruit in 1978 and 1979.

Anthracnose damage showed a highly significant correlation to the oil content in the fruit with y = 3,02 - 0,11x and a correlation coefficient of r = -0,996306. This indicates a decreased anthracnose severity with the increase in oil content of the fruit.

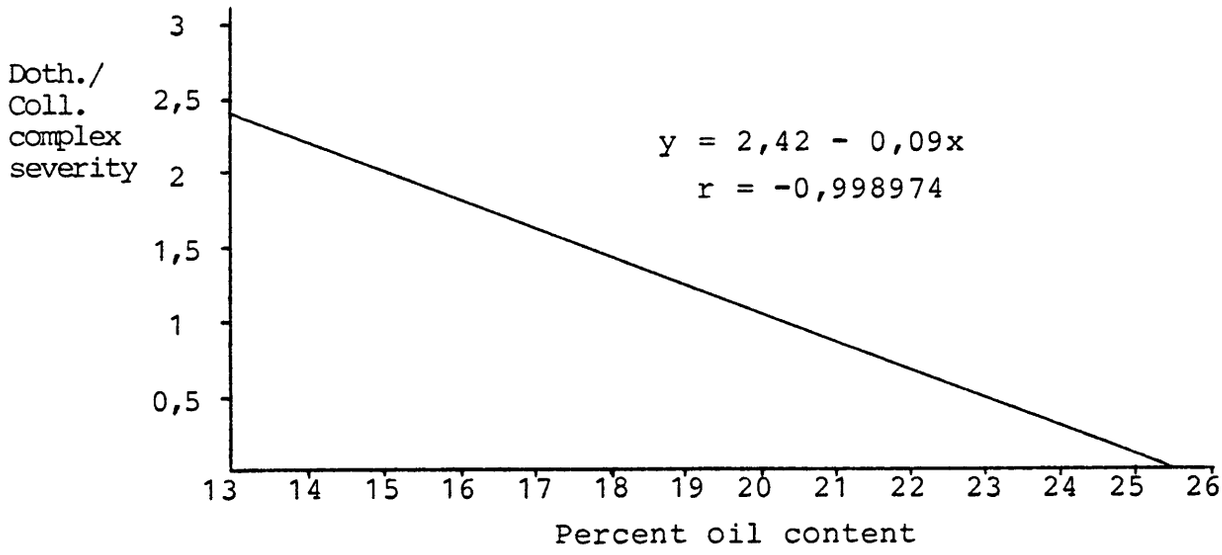

FIG. 18. - The severity of Dothiorella/Colletotrichum complex fruit rot damage to Fuerte in relation to the oil content of the fruit in 1978 and 1979.

Dothiorella/Colletotrichum complex fruit rot was also less severe on fruit with higher oil content and the linear regression equation to describe this decrease in disease severity is y = 2,42 - 0,09x, with a highly significant correlation coefficient of r = -0,998974.

Thus, all three post-harvest diseases, stem-end rot, anthracnose and Dothiorella/Colletotrichum complex fruit rot were found to be important on Fuerte fruit with low oil content and the severity of these diseases decreased with the increase in oil content as fruit matured.

4.2 OBSERVATIONS ON POST-HARVEST DISEASE SYMPTOMS AND ON THE PATHOGENS

Stem-end rot is a type of fruit decay which always starts from the pedicel end of the fruit and advances internally, causing rot of the fruit flesh. Externally, the infected fruit turns brown or black along with the advance of the rot internally. Internal symptoms include flesh rot and vascular bundle discolouration. It is often associated with an unpleasant odour. Sporulation of the invading fungus may be seen on the fruit surface in the advanced stages of the disease and mycelial growth may be observed in cavities inside the fruit. Internal cavities are seldom formed. Under dry conditions the mummification of infected fruit frequently occurs (Photo 8). Stem-end rot symptoms are often typical for the specific fungus species causing the disease (Photos 9, 10, 11, 12, 13, 14, 15, 16, 17, 18 and 19). Differences in stem-end rot symptoms caused by the various fungi were evaluated in respect of the colour of the internal flesh rot, the presence of vascular bundle discolouration and the sporulation of the fungus on the surface of infected fruit (Table 13).

It was found at Westfalia Estate that anthracnose occasionally shows up on hard Fuerte fruit prior to harvest and that it develops mostly in wounds caused by Cercospora spot or insects. Anthracnose on hard fruit, however, appears to be much less serious than that on softening fruit. In all the post-harvest work in this study, only the anthracnose symptoms which develop

TABLE 13. - Stem-end rot symptoms on Fuerte fruit caused by various pathogens in artificial inoculations

Fungi	Flesh discolouration			Vascular bundle discolouration	Externally visible sporulation
	Reddish brown	Brown	Black		
Colletotrichum gloeosporioides	-	+	+	+	+
Dothiorella aromatica	-	+	+	+	-
Drechslera setariae	-	+	-	+	-
Fusarium decemcellulare	+	+	-	-	+
Fusarium sambucinum	+	+	-	-	-
Fusarium solani	-	+	-	-	-
Lasiodiplodia theobromae	-	-	+	+	+
Pestalotiopsis versicolor	-	+	-	+	+
Phomopsis perseae	-	+	-	-	-
Rhizopus stolonifer	-	+	-	+	+
Thyronectria pseudotrichia	-	+	-	+	+

on soft fruit with the typical symptoms of circular rot with an early sporulation in the centre and deep flesh penetration on soft fruit, were classified as anthracnose (Photo 20). The superficial form of Colletotrichum gloeosporioides rot which does not usually penetrate the flesh and in which sporulation appears only in the late stage of the disease, was classified as part of the Dothiorella/Colletotrichum complex fruit rot.

Symptoms of Dothiorella fruit rot on Fuerte include the superficial form of rot with a reddish-brown colour at first which turns dark brown later. It develops only on softening fruit after the fruit has been picked. Artificial pre-harvest inoculations with Dothiorella aromatica produced symptoms often indistinguishable from the superficial form of anthracnose caused by C. gloeosporioides and it was therefore classified under the collective name of Dothiorella/Colletotrichum complex fruit rot. When isolations are made from the complex rot of Fuerte, a mixed population of the fungi D. aromatica and C. gloeosporioides is recovered.

Photo 21 illustrates symptoms of the post-harvest Dothiorella/Colletotrichum complex fruit rot which developed on Fuerte fruit inoculated three to five months before harvest with a mixed spore suspension of D. aromatica and C. gloeosporioides. Re-isolations from the encircled area proved the presence of both fungi (see Table 18).

The following fungi, which also exhibited some degree of virulence in follow-up pathogenicity studies, were isolated out of stem-end rot of avocados at Westfalia Estate: Thyronectria pseudotrichia (Schw.) Seeler, Colletotrichum gloeosporioides (Penz.) Sacc., Dothiorella aromatica (Sacc.) Petr. and Syd., Phomopsis perseae Zerova, Lasiodiplodia theobromae (Pat.) Griff. and Maubl., Fusarium decemcellulare Brick, Fusarium sambucinum Fuckel, Fusarium solani (Mart.) Sacc., Drechslera setariae (Sawada) Subram. and Jain, Pestalotiopsis versicolor (Speg.) Steyart and Rhizopus stolonifer (Ehr. ex Fr.) Lind. These fungi were tentatively identified at Westfalia Estate and subsequently submitted for confirmation to the Commonwealth Mycological Institute in England.

The fungus causing anthracnose was identified as Colletotrichum gloeosporioides (Penz.) Sacc. in the conidial form and Glomerella cingulata (Ston.) Spauld. and Schrenk in the ascogenous form.

The identity of the fungus involved in the complex fruit rot was determined by taxonomists as Dothiorella aromatica (Sacc.) Petr. and Syd.

T. pseudotrichia in fresh isolations produced fast growing mycelia mostly submerged in the medium with some aerial parts on which conidia were produced on simple phialides forming a spore ball on top. In older isolates Stilbella coremia develop on the surface of the medium, (Photo 22). No perithecia were produced in axenic cultures.

C. gloeosporioides was readily isolated from infected plant material. It sporulated well on PDA after several transfers (Photo 23). Cultures first produced asexual conidia and later Glomerella perithecia appeared in many of the isolates grown under near UV light (Photo 24).

D. aromatica cultures showed a fair to poor pycnidial production in the from-fruit-to-PDA isolations if grown under near UV, but were inclined to produce sterile mycelia in subsequent transfers. The fungus remained in sporulation or sterile cultures regained the ability to sporulate if transfer was done onto aseptic avocado fruit pieces under near UV light (Photo 25).

P. perseae sporulated sparsely in fresh isolations, but became sterile after a few transplants. It could be kept or returned to sporulation on sterile avocado pieces under near UV light (Photo 26). Some of the from-fruit-to-PDA isolates yielded a hitherto unreported perithecial form with two-celled ascospores.

F. decemcellulare formed colonies rich in red pigment on PDA and typical micro-and macroconidia were produced (Photo 27).

L. theobromae tends to lose the ability to sporulate after several subculturings and it can be restored by inoculating it onto avocado fruit under near UV light (Photo 28).

4.3 DISTRIBUTION OF POST-HARVEST DISEASES AND PATHOGENS

Results of the investigation on the occurrence of post-harvest diseases with reference to tree aspect are presented in Table 14.

TABLE 14. - The distribution of post-harvest diseases on Fuerte fruit harvested from various aspects of the tree in 1981/82 season.

Aspect of tree	Mean post-harvest disease severity (0 to 10 scale)(N = 1200)		
	Doth./Coll. complex	Anthracnose	Stem-end rot
North	1,05 a	0,07 a	0,12 b
South	0,51 a	0,00 a	0,04 c
East	0,87 a	0,04 a	0,27 a
West	0,71 a	0,02 a	0,04 c

Means with letters a, b and c differ statistically at 0,05 level (Duncan's multiple range test)

Dothiorella/Colletotrichum complex fruit rot and anthracnose appeared to be evenly distributed on fruit in the various aspects of the tree, but stem-end rot incidence was significantly the highest on fruit on the eastern side of the trees, followed by fruit on the northern side.

Correlations between post-harvest disease incidence and root rot severity of trees are given in Figs. 19, 20 and 21.

Stem-end rot showed a non-significant correlation to the root rot state of Fuerte trees and is described with $y = 1,00 - 0,08x$, with $r = -0,507888$. This indicates that stem-end rot incidence may decrease with an increase in root rot severity of the trees.

The linear regression model to illustrate correlation between anthracnose and root rot severity of trees is $y = 1,38 - 0,17x$, with a significant correlation coefficient of $r = -0,655084$. This proves statistically that anthracnose is less prevalent on trees more severely attacked by root rot.

The incidence of Dothiorella/Colletotrichum complex

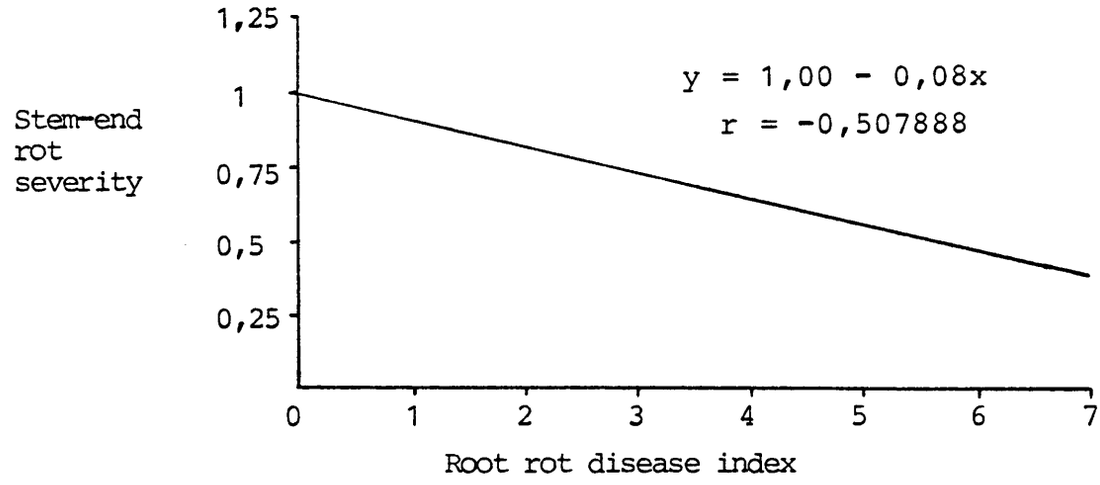

FIG. 19. - Stem-end rot incidence on Fuerte fruit harvested from trees at various stages of root rot in 1981/82.

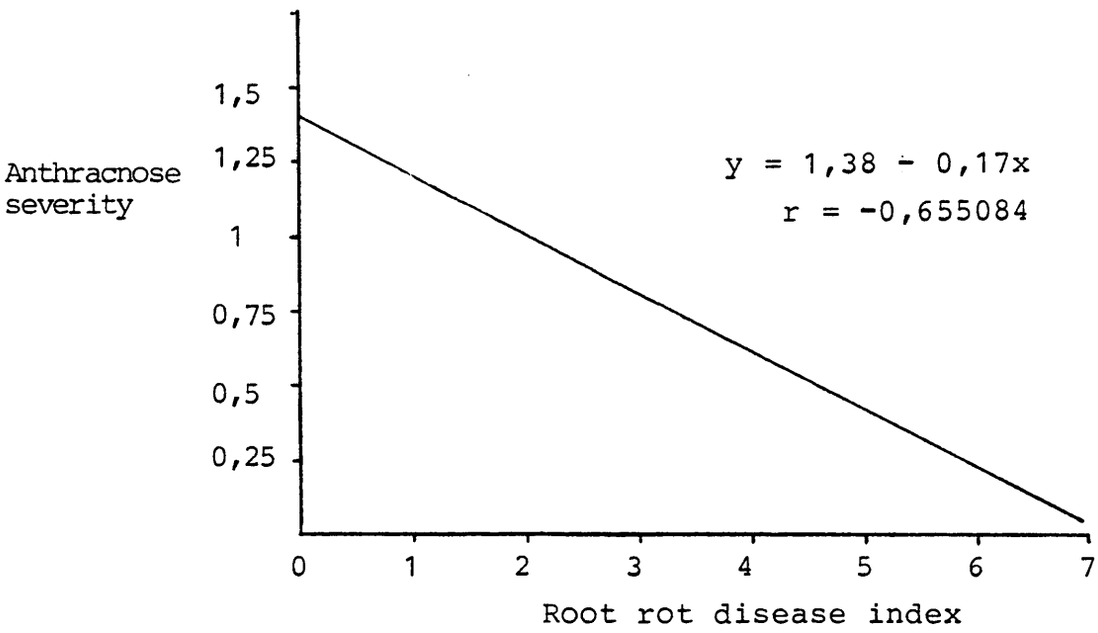

FIG. 20. - Anthracnose incidence on Fuerte fruit harvested from trees at various stages of root rot in 1981/82.

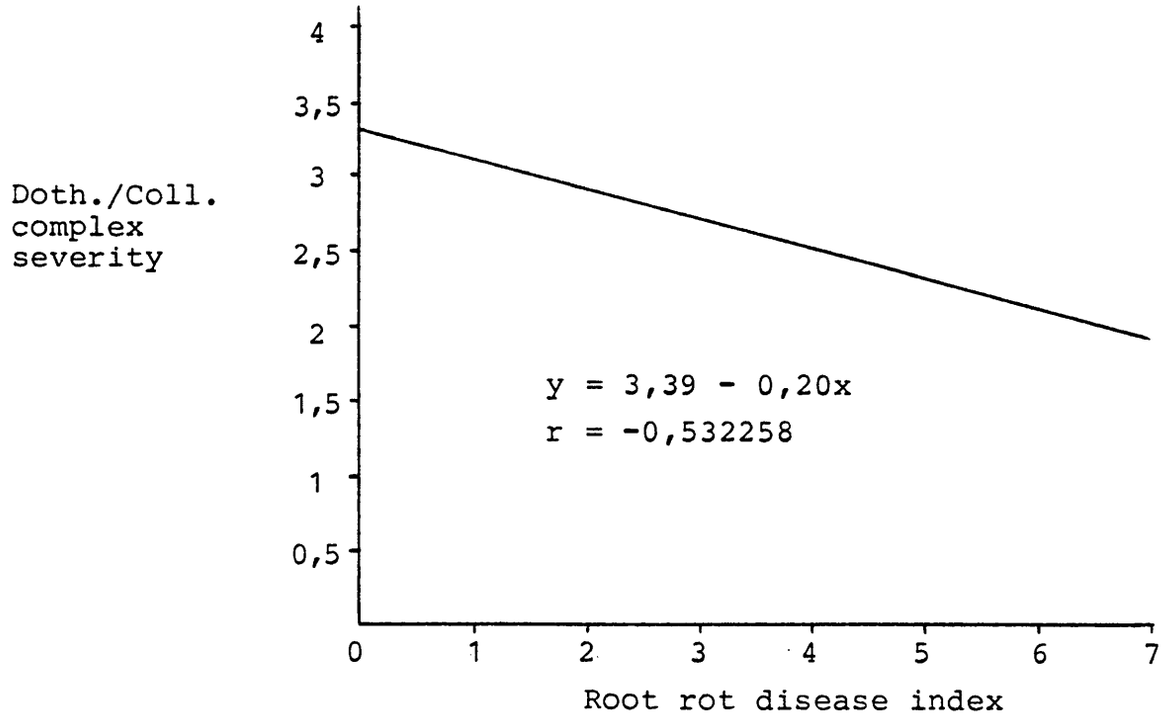

FIG. 21. - Dothiorella/Colletotrichum complex fruit rot on Fuerte harvested from trees at various stages of root rot in 1981/82.

fruit rot also tended to decrease with increased root rot severity of the trees. The equation for the correlation being y = 3,39 - 0,20x, with a non-significant r = -0,532258.

The occurrence of the various stem-end rot pathogens and the effect of cold storage on their frequency was investigated on Fuerte and the results are presented in Table 15.

By far the most common fungus in the stem-end rot of Fuerte ripened at ambient temperature was Thyronectria pseudotrichia and though its frequency was reduced considerably by cold storage it remained the most significant organism in stem-end rot of cold stored fruit. Colletotrichum gloeosporioides showed a marked increase in its relative frequency after cold storage. A similar increase was observed in the occurrence of Phomopsis perseae after cold storage. The frequency of Dothiorella aromatica stayed almost unchanged by cold storage. Rhizopus stolonifer appeared only in fruit which underwent cold storage. The Fusarium spp. and Drechslera setariae were occasionally seen in stem-end rot.

TABLE 15. - The occurrence of fungal pathogens in stem-end rot of Fuerte ripened at ambient temperature and after 28 days cold storage at 6°C in 1978.

Fungi	Percentage occurrence (N=700)	
	Ripened at ambient temp.	Ripened after cold storage
Thyronectria pseudotrichia	81	36
Colletotrichum gloeosporioides	7	28
Phomopsis perseae	3	12
Dothiorella aromatica	4	6
Fusarium decemcellulare	1	-
Fusarium sambucinum	-	2
Fusarium solani	-	2
Rhizopus stolonifer	-	8
Drechslera setariae	-	2
Unidentified fungi	4	4

The incidence of stem-end rot fungi in Edranol fruit is given in Table 16.

TABLE 16. - Occurrence of fungal pathogens in stem-end rot of Edranol ripened at ambient temperature in 1978.

Fungi	Percentage occurrence (N=200)
Dothiorella aromatica	64
Colletotrichum gloeosporioides	18
Thyronectria pseudotrichia	9
Pestalotiopsis versicolor	9

Stem-end rot in Edranol is much less of a problem than in Fuerte, so fewer diseased fruit were available for isolation. The major pathogen in Edranol stem-end rot appeared to be D. aromatica with C. gloeosporioides as the second most common, while only a few fruit showed T. pseudotrichia and P. versicolor infection.

4.4 SOURCE OF INOCULUM OF POST-HARVEST DISEASE PATHOGENS

T. pseudotrichia was the most prevalent fungus on avocado branches which were dying back or were already dried out. Invaded twigs or branches close to the point of attachment to larger branches are often covered with the macroscopically visible mass of coremia of the Stilbella stage. This gives a yellow to orange-red colour to the affected branches. Perithecia develop at the base of older coremia. Results of the survey in which the percentage of dead branches bearing T. pseudotrichia sporulation was investigated on the various avocado cultivars are presented in Table 17.

TABLE 17. - The frequency of branch infection by T. pseudotrichia on different avocado cultivars in 1982.

Cultivars	Percentage dead branches infected with T. pseudotrichia (N = 400)
Fuerte	51
Hass	32
Ryan	30
Edranol	22

T. pseudotrichia was most frequent on Fuerte branches and less common on Hass and Ryan branches and the least branch infection by the fungus was recorded for Edranol.

C. gloeosporioides was a very common fungus on all four cultivars observed virtually on all parts of the trees and it was present in both the conidial and perithecial form. During the spore trapping of Pseudocercospora purpurea, spores indistinguishable from the ascospores of Glomerella cingulata were seen in great numbers.

D. aromatica was often isolated from twigs and branches, but fruiting bodies were not frequently seen on them. Pycnidia were produced in abundance on fallen fruit.

P. perseae pycnidia were found on dead branches sunken in the bark of Fuerte trees and the fungus was often observed with abundant sporulation in the skin of overripe Hass fruit.

F. decemcellulare causes die-back of avocado branches and close to the base of the affected branches yellow sporodochia appear from under the bark in which mostly macronidia are produced and on the same stromae perithecia of the Calonectria rigidiuscula stage are formed. It was most common on Hass, but was also found on Fuerte and Edranol.

L. theobromae was widely distributed, but not often seen. Pycnidia appeared on dead branches and the fungus was isolated also from stem canker of Fuerte trees in association with Phytophthora cinnamomi Rands.

4.5 PATHOGENICITY OF POST-HARVEST DISEASE PATHOGENS

The pathogenicity of fungi isolated from stem-end rot of avocados at Westfalia Estate was investigated by pre-harvest inoculations. This was done in order to establish their pathogenicity as the active, primary cause of diseases and also to determine the types of diseases they cause (Table 18).

C. gloeosporioides and D. aromatica were found to be capable of directly attacking uninjured Fuerte fruit prior to harvest, on the tree and to cause post-harvest fruit spot, rot and stem-end rot. All symptoms developed on softening fruit only. L. theobromae caused stem-end rot only, but the fungus was recovered from under the skin of apparently symptomless Fuerte fruit. F. decemcellulare induced a few small brown skin lesions on the ripening fruit from which the fungus was readily re-isolated. None of the other organisms caused post-harvest disease development in this experiment. P. perseae inoculated fruit dropped before harvest, so no observations could be made on the symptoms caused by this organism.

The pathogenic potentials of these organisms as stem-end rot pathogens were studied in post-harvest inoculations on four avocado cultivars (Tables 19, 20, 21 and 22).

TABLE 18. - The types of post-harvest diseases developed on Fuerte fruit after pre-harvest inoculation with stem-end rot fungi in 1978/79.

Fungi	External fruit spot or rot symptoms	Stem-end rot	Re-isolation of fungi
Colletotrichum gloeosporioides	+	+	+
Dothiorella aromatica	+	+	+
Drechslera setariae	-	-	-
Fusarium decemcellulare	+	-	+
Fusarium sambucinum	-	-	-
Fusarium solani	-	-	-
Lasiodiplodia theobromae	-	+	+
Pestalotiopsis versicolor	-	-	-
Rhizopus stolonifer	-	-	-
Thyronectria pseudotrichia	-	-	-

TABLE 19. - Pathogenicity of stem-end rot fungi to avocados inoculated through the pedicel scar and evaluated externally.

Fungi	Mean external stem-end rot severity (0 to 10 scale) (N= 528)				
	Fuerte	Edranol	Hass	Ryan	Mean
Colletotrichum gloeosporioides	2,3	4,0	5,6	10	5,4 ab
Dothiorella aromatica	2,8	3,3	2,0	10	4,5 bcd
Drechslera setariae	0,5	0,3	0	0	0,2 d
Fusarium decemcellulare	0,8	0,5	0	1,3	0,6 d
Fusarium sambucinum	1,0	1,0	0	0,8	0,7 d
Fusarium solani	0,8	0	0	0	0,2 d
Lasiodiplodia theobromae	7,5	7,8	6,5	9,8	7,9 a
Pestalotiopsis versicolor	1,0	1,0	0,5	1,0	0,8 d
Phomopsis perseae	1,3	1,3	0	4,5	1,7 cd
Rhizopus stolonifer	6,0	10	0	3,0	4,7 abc
Thyronectria pseudotrichia	1,5	1,3	0,8	3,5	1,7 cd
Mean	2,3 a	2,7 a	1,4 a	3,9 a	

Means with letters a, b, c and d differ statistically at 0,05 level (Duncan's multiple range test)

L. theobromae, C. gloeosporioides and R. stolonifer were the most pathogenic fungi to the four avocado cultivars in inoculations through the pedicel scar. D. aromatica was the next most pathogenic species. P. perseae and T. pseudotrichia were moderately pathogenic, while D. setariae, F. decemcellulare, F. sambucinum, F. solani and P. versicolor possessed even less pathogenic potential to cause stem-end rot. Statistical analyses showed that all four avocado cultivars were equally susceptible to infections by these fungi through the pedicel scar.

In inoculations onto 0,5cm long pedicel, L. theobromae, C. gloeosporioides and D. aromatica were the most pathogenic species on the four avocado cultivars, while P. perseae and T. pseudotrichia were significantly less virulent. R. stolonifer and D. setariae inoculations induced no external stem-end rot symptoms on any of the cultivars. No statistical difference was obtained between the various cultivars as to their susceptibility to stem-end rot.

Internal stem-end rot damage in the inoculations through the pedicel scar was most severe in fruit with L. theobromae, C. gloeosporioides and D. aromatica infections. The mean stem-end rot damage caused by R. stolonifer on the four cultivars was significantly less and though stem-end rot caused by P. perseae and T. pseudotrichia was even less severe, the difference was not significant. There was no appreciable difference in the susceptibility of the various avocado cultivars to stem-end rot infection through the pedicel scar.

Following inoculations onto 0,5cm long fruit pedicel the internal stem-end rot damage was most extensive with L. theobromae, C. gloeosporioides and D. aromatica. There were no statistical differences between the pathogenicity of the other fungi, though P. perseae and T. pseudotrichia tended to be more virulent than the others. The four avocado cultivars showed no significant differences in susceptibility to stem-end rot if inoculated through the pedicel and evaluated internally.

The ability of these fungi to cause post-harvest fruit rot on the four avocado cultivars was also investigated by

TABLE 20. - Pathogenicity of stem-end rot fungi to avocados inoculated onto half cm long pedicel and evaluated externally.

Fungi	Mean external stem-end rot severity (0 to 10 scale) (N=528)				
	Fuerte	Edranol	Hass	Ryan	Mean
Colletotrichum gloeosporioides	2,0	1,5	1,8	5,8	2,7 ab
Dothiorella aromatica	1,5	2,0	2,0	8,0	3,3 a
Drechslera setariae	0	0	0	0	0 d
Fusarium decemcellulare	0,5	0	0	0,5	0,2 d
Fusarium sambucinum	0	0,3	0	0	0,1 d
Fusarium solani	0,3	0	0	0,5	0,2 d
Lasiodiplodia theobromae	3,3	2,0	0	4,5	2,4 abc
Pestalotiopsis versicolor	0	0	0	1,0	0,2 d
Phomopsis perseae	0,8	1,0	0	1,0	0,7 cd
Rhizopus stolonifer	0	0	0	0	0 d
Thyronectria pseudotrichia	0,5	0,3	0	1,3	0,5 d
Mean	0,8 a	0,6 a	0,3 a	2,0 a	

Means with letters a, b, c and d differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 21. - Pathogenicity of stem-end rot fungi to avocados inoculated through the pedicel scar and evaluated internally.

Fungi	Mean internal stem-end rot severity (0 to 10 scale) (N= 528)				
	Fuerte	Edranol	Hass	Ryan	Mean
Colletotrichum gloeosporioides	3,5	8,8	10	10	8,0 a
Dothiorella aromatica	4,5	5,0	9,3	10	7,2 ab
Drechslera setariae	0,5	0,3	1,0	0	0,4 d
Fusarium decemcellulare	1,0	0,5	0,5	1,3	0,8 d
Fusarium sambucinum	1,3	1,3	2,5	1,0	1,5 d
Fusarium solani	0,8	0	0,5	0	0,3 d
Lasiodiplodia theobromae	8,8	9,3	10	9,8	9,4 a
Pestalotiopsis versicolor	1,0	1,0	1,8	1,3	1,2 d
Phomopsis perseae	2,8	2,0	2,0	5,8	3,1 cd
Rhizopus stolonifer	6,0	10	0,3	3,3	4,9 bc
Thyronectria pseudotrichia	2,0	1,8	3,5	4,0	2,8 cd
Mean	2,9 a	3,6 a	3,7 a	4,2 a	

Means with letters a, b, c and d differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 22. - Pathogenicity of stem-end rot fungi to avocados inoculated onto a half cm long pedicel and evaluated internally.

Fungi	Mean internal stem-end rot severity (0 to 10 scale) (N=528)				
	Fuerte	Edranol	Hass	Ryan	Mean
Colletotrichum gloeosporioides	2,5	3,5	9,3	9,5	6,2 a
Dothiorella aromatica	2,3	3,3	7,3	8,8	5,4 a
Drechslera setariae	0	0	0,5	0	0,1 b
Fusarium decemcellulare	0,5	0	1	0,5	0,5 b
Fusarium sambucinum	0	0,3	0	0	0,1 b
Fusarium solani	0,3	0	0	0,5	0,2 b
Lasiodiplodia theobromae	5,5	4,0	2,8	5,0	4,3 a
Pestalotiopsis versicolor	0	0	0,3	1,0	0,3 b
Phomopsis perseae	1,3	1,5	1,0	2,0	1,4 b
Rhizopus stolonifer	0	0	0	0	0 b
Thyronectria pseudotrichia	0,8	0,3	1,5	2,0	1,1 b
Mean	1,2 a	1,1 a	2,1 a	2,6 a	

Means with letters a and b differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 23. - Fruit rot caused after 11 days by some of the stem-end rot fungi in artificial post-harvest inoculations through wounds.

Fungi	Diameter of fruit rot in mm 11 days after inoculation (N=864)				
	Fuerte	Edranol	Hass	Ryan	Mean
Colletotrichum gloeosporioides	23	34	32	11	25 abc
Dothiorella aromatica	13	10	19	1	10 bc
Fusarium decemcellulare	19	20	12	12	15 bc
Fusarium culmorum	8	13	12	0	8 c
Lasiodiplodia theobromae	47	80	35	41	50 a
Pestalotiopsis versicolor	0	0	0	0	0 c
Phomopsis perseae	3	14	7	0	6 c
Rhizopus stolonifer	130	16	16	0	40 ab
Thyronectria pseudotrichia	20	4	5	6	8 c
Mean	29 a	21 a	15 a	7 a	

Means with letters a, b and c differ statistically at 0,05 level (Duncan's multiple range test)

inoculating them into wounds. The extent of fruit rot which developed 11 days after inoculation was measured to determine pathogenicity (Table 23).

The stem-end rot fungi that showed significant potential to cause fruit rot through wounds on the skin were L. theobromae, R. stolonifer and C. gloeosporioides. Moderate fruit rotting potential was found for F. decemcellulare and D. aromatica. Although Ryan was the cultivar least susceptible to rot caused by stem-end rot fungi through wounded epidermis, the difference was not significant.

4.6 CRITICAL INFECTION PERIODS FOR POST-HARVEST DISEASES

Results of the experiment exposing fruit to natural infection under orchard conditions in 1977/78 season are presented in Figs. 22, 23 and 24.

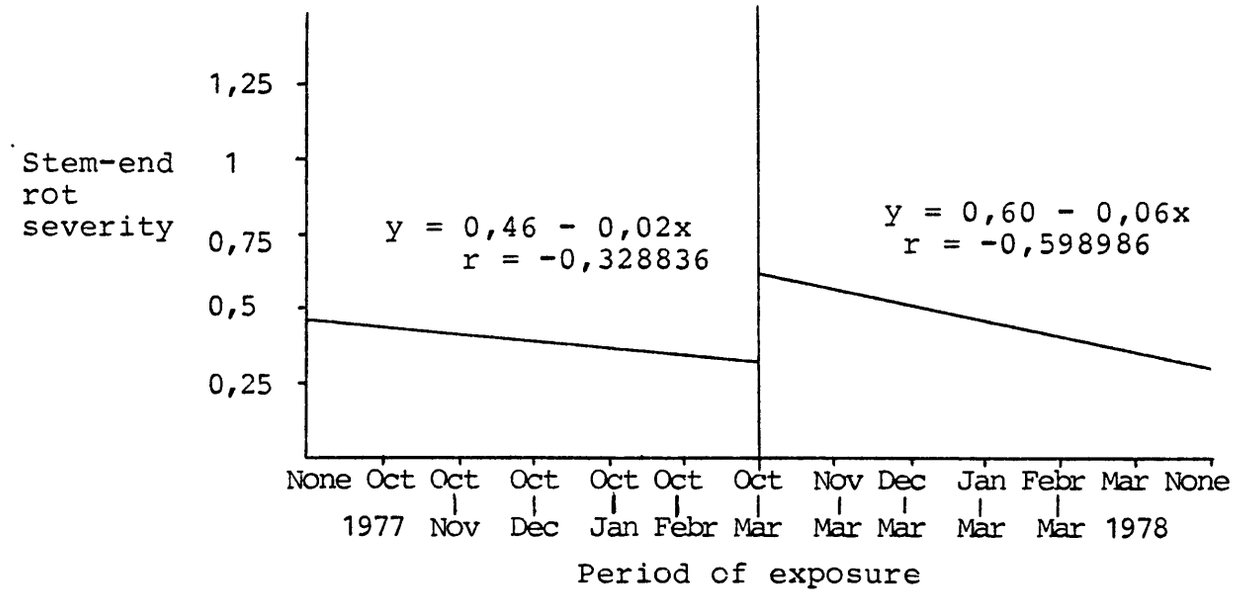

FIG. 22 - The effect of the length of the exposure time on the natural stem-end rot infection of Fuerte fruit in 1977/78.

A decrease in stem-end rot incidence was obtained in the monthly cumulative exposures early in the season with $y = 0,46 - 0,02x$, $r = -0,328836$ is non-significant. The monthly cumulative exposures, by shortening the length of

exposures towards the end of the season, indicated a decrease in stem-end rot incidence and the regression which described this correlation is y = 0,60 - 0,06x, with a non-significant r = -0,598986.

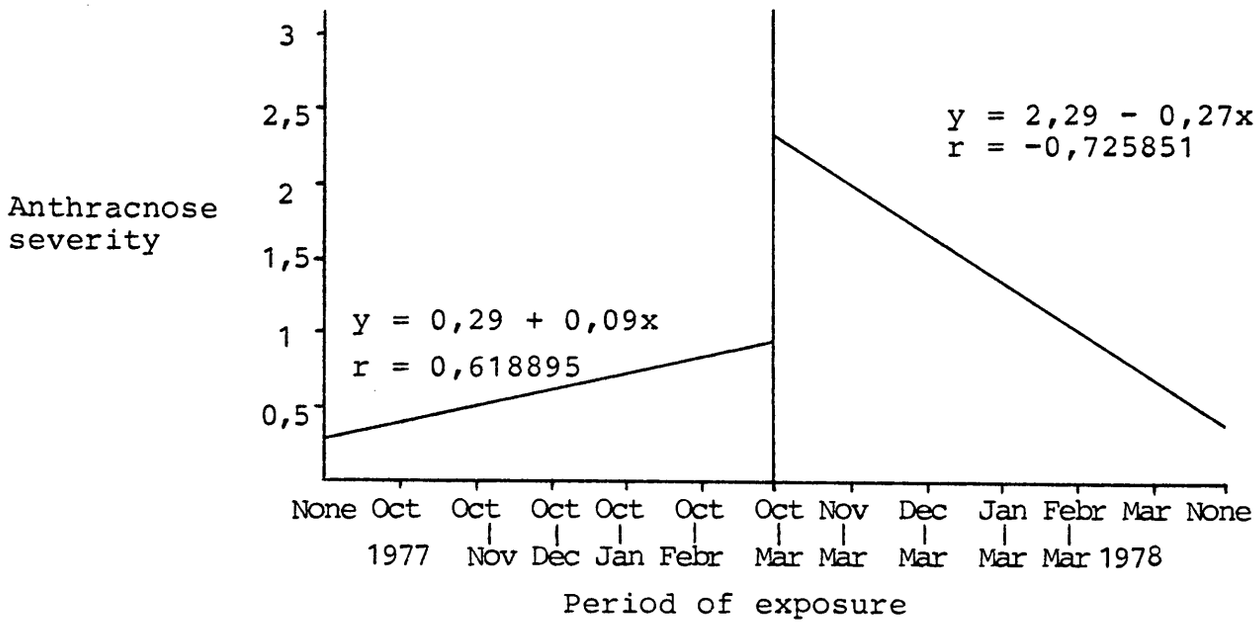

FIG. 23. - The effect of the length of the exposure time on the natural anthracnose infection of Fuerte fruit in 1977/78.

Anthracnose increased with the lengthening of exposure time on a monthly basis at the beginning of the summer season and it is described with the equation of y = 0,29 + 0,09x, with a non-significant r = 0,618895. The decrease in anthracnose incidence was significant if correlated to the cumulative exposures shortened progressively towards harvest time with y = 2,29 - 0,27x and r = -0,725851.

Dothiorella/Colletotrichum complex fruit rot increased with the progressive lengthening of exposure time early in the season and the linear regression model to describe the correlation is y = -0,62 + 0,38x, with a highly significant correlation coefficient of r = 0,926882. The decrease in the incidence of Dothiorella/Colletotrichum complex correlated to the shortening in exposure time towards the end of the

summer season with y = 2,74 - 0,37x, with a highly significant r = -0,970146.

The fruit exposure experiment was repeated in the 1978/79 season on Fuerte trees and the results are given in Figs. 25, 26 and 27.

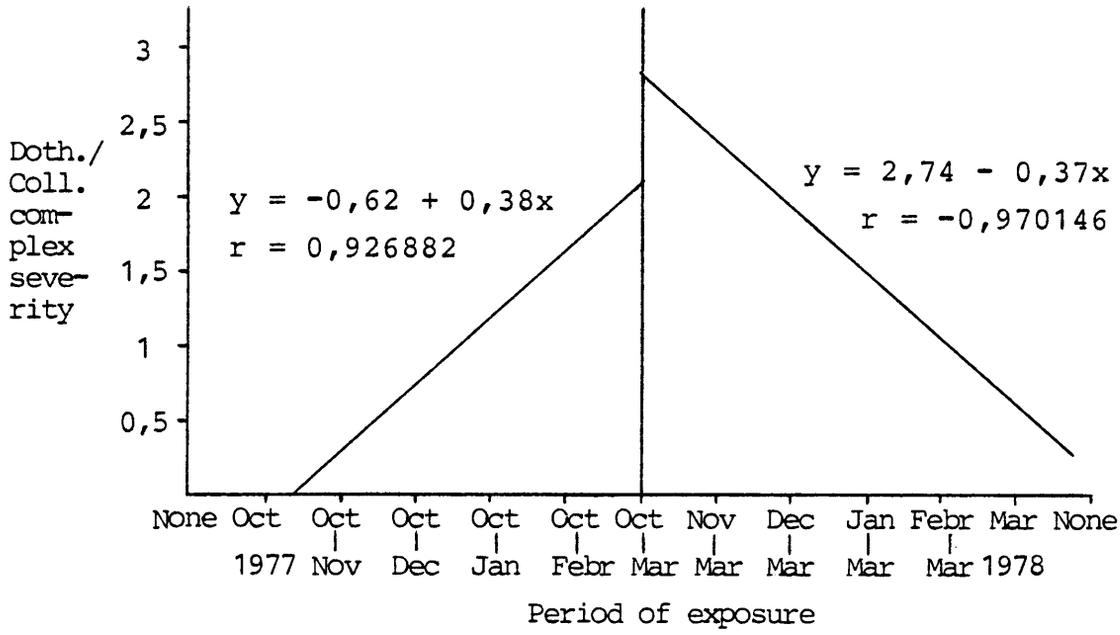

FIG. 24. - The effect of the length of the exposure time on the natural Dothiorella/Colletotrichum complex infection of Fuerte fruit in 1977/78.

Stem-end rot incidence appeared to be more severe on fruit exposed later in the season in the monthly exposures with y = 0,57 + 0,01x and r = 0,055884, which is not significant. Correlation of the disease with the monthly cumulative exposure periods is calculated as y = 1,41 - 0,21x, with a non-significant r = -0,692672 (Fig. 25).

Anthracnose incidence remained static in the monthly exposures and is described by y = 0,56 + 0,0005x with a non-significant correlation coefficient of r = 0,002608. There was a decrease in anthracnose with the shortening of the exposure time and the correlation is given as y = 1,10 - 0,13x, with a non-significant correlation coefficient of r = -0,582546 (Fig. 26).

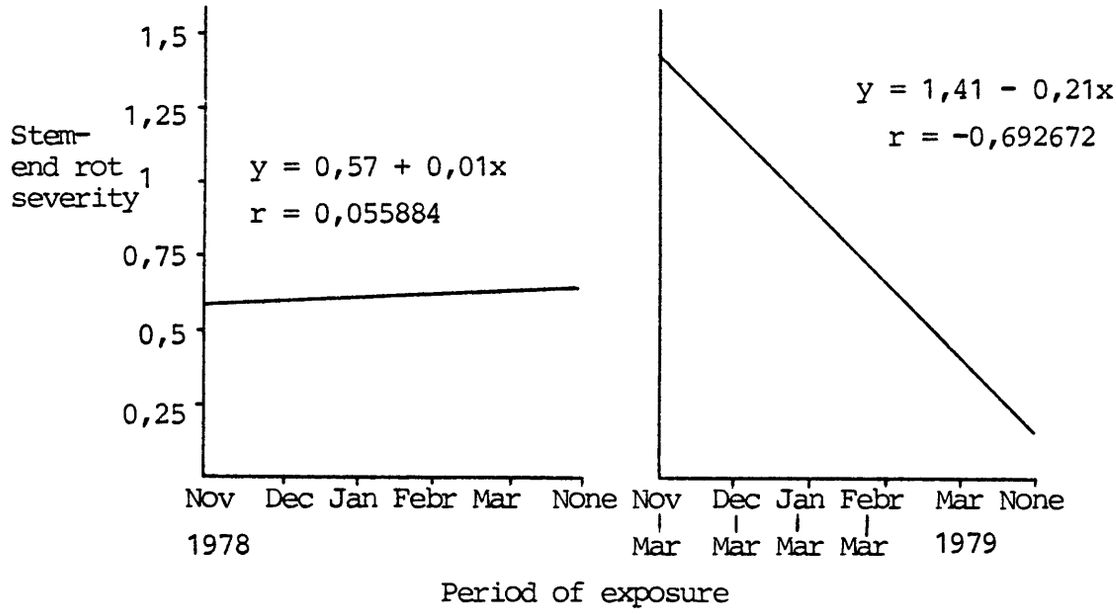

FIG. 25. - The effect of the length and timing of the exposure periods on natural stem-end rot infection in 1978/79.

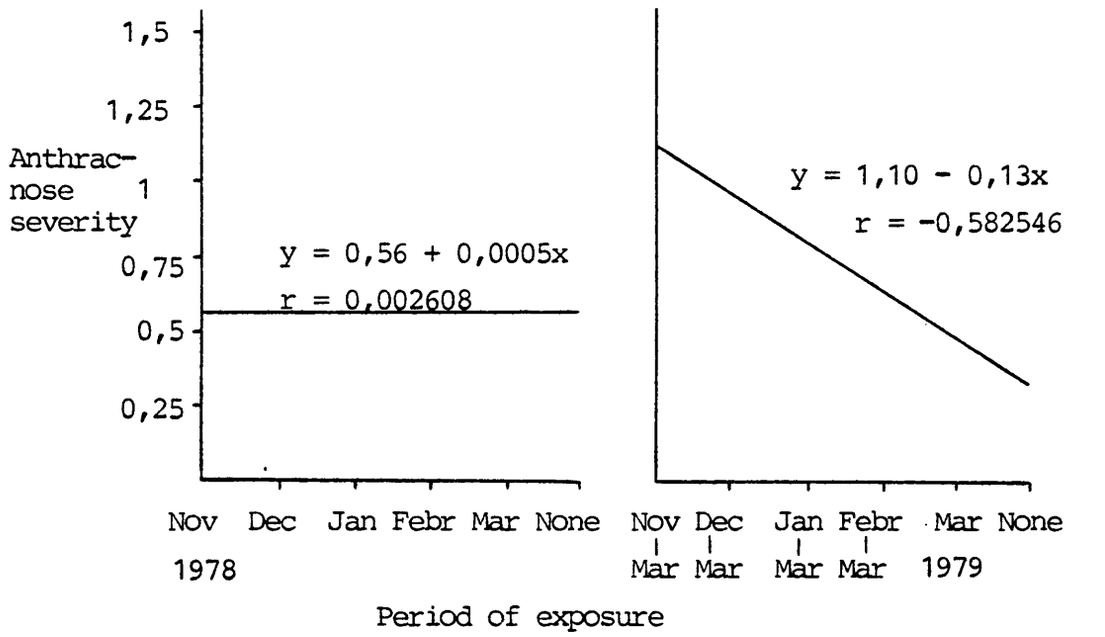

FIG. 26. - The effect of the length and timing of the exposure periods on the natural anthracnose infection in 1978/79.

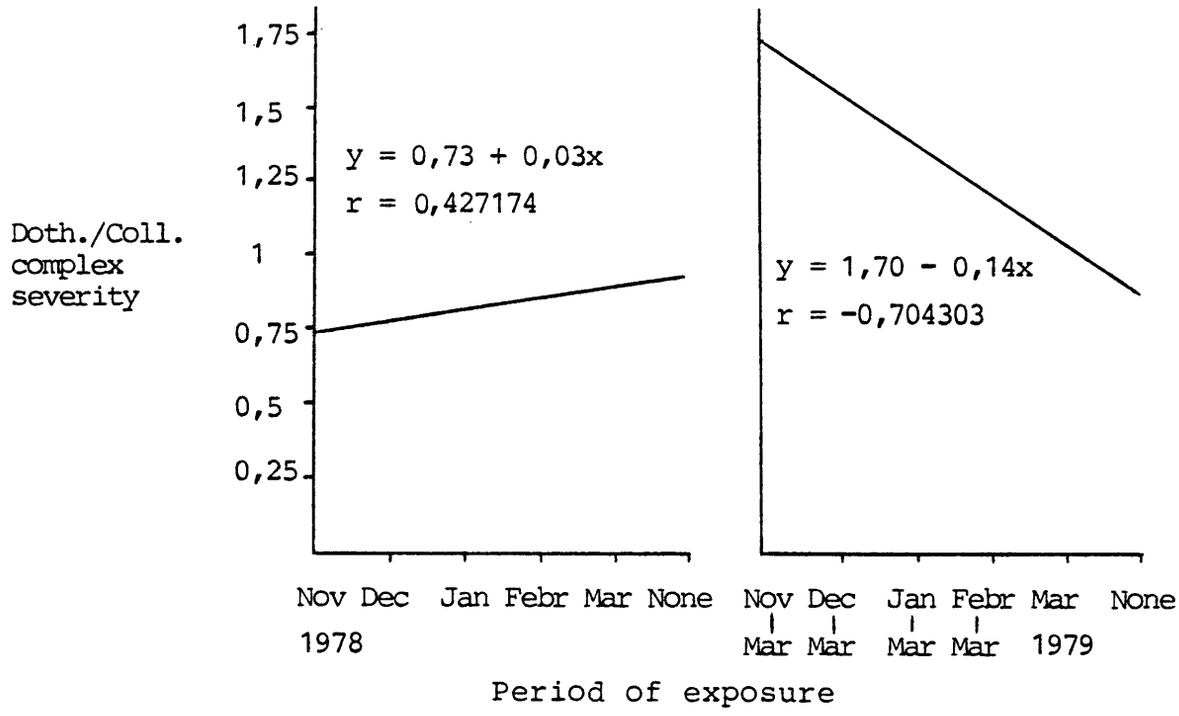

FIG. 27. - The effect of the length and timing of the exposure periods on the natural Dothiorella/ Colletotrichum complex fruit rot infection in 1978/79.

Dothiorella/Colletotrichum complex fruit rot tended to be more severe on fruit exposed late in the season in the monthly exposures with y = 0,73 + 0,03x and a correlation coefficient of r = 0,427174 which is not significant. It tended to decrease with the shortening of the exposure periods, the regression model being y = 1,70 - 0,14x, with a non-significant r = -0,704303.

Data on the post-harvest diseases in the 1978/79 season's fruit exposure experiment were also correlated to rainfall figures and the results are presented in Figs. 28, 29 and 30.

Stem-end rot incidence showed a slight negative correlation with rainfall in the monthly exposures with y = 0,64 - 0,00005x and a non-significant correlation coefficient of r = -0,005705 In monthly cumulative exposures, stem-end rot tended to increase with the increase in rainfall and the linear regression model to describe the correlation is y = 0,07 + 0,001x, with a non-significant r = 0,651586.

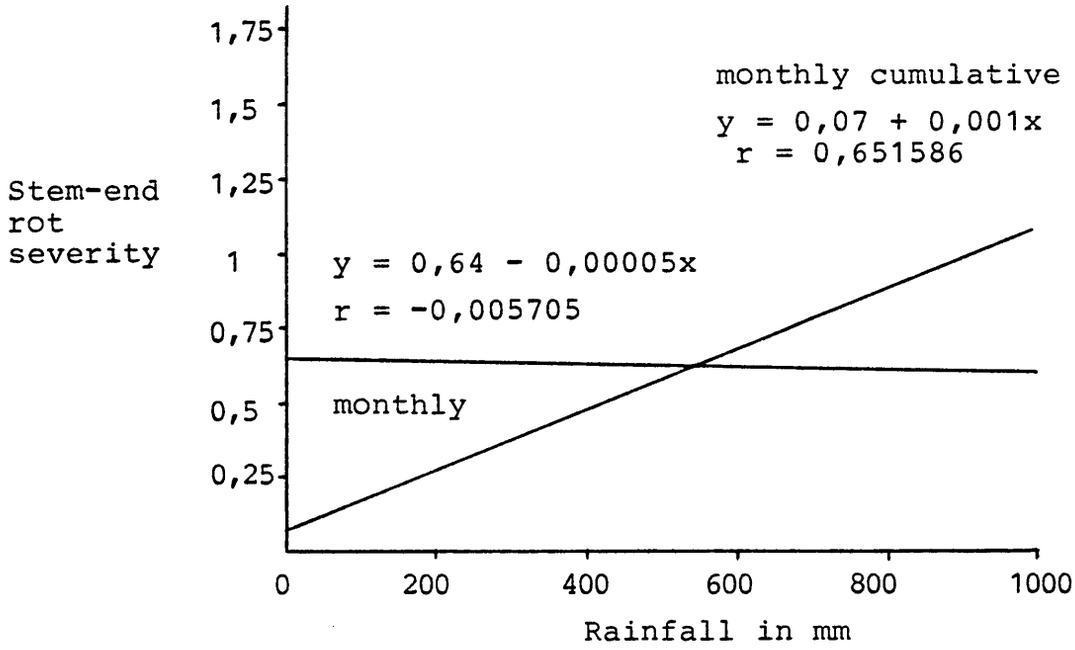

FIG. 28. - The correlation between stem-end rot and rainfall in the 1978/79 season's fruit exposure experiment.

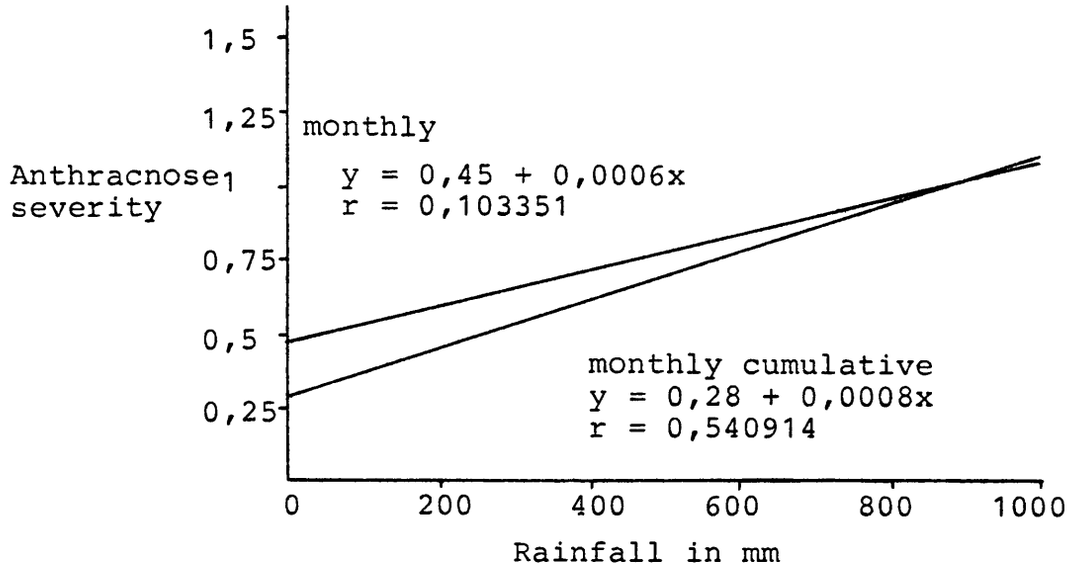

FIG. 29. - The correlation between anthracnose and rainfall in the 1978/79 season's fruit exposure experiment.

Both in the monthly and in the monthly cumulative fruit exposures, anthracnose incidence correlated positively with the rainfall. The linear regression model to describe the

correlation between anthracnose of the monthly exposures and rainfall is y = 0,45 + 0,0006x, with a non-significant correlation coefficient of r = 0,103351, while the correlation between anthracnose in the monthly cumulative exposures and rainfall is y = 0,28 + 0,0008x, with a non-significant r = 0,540914.

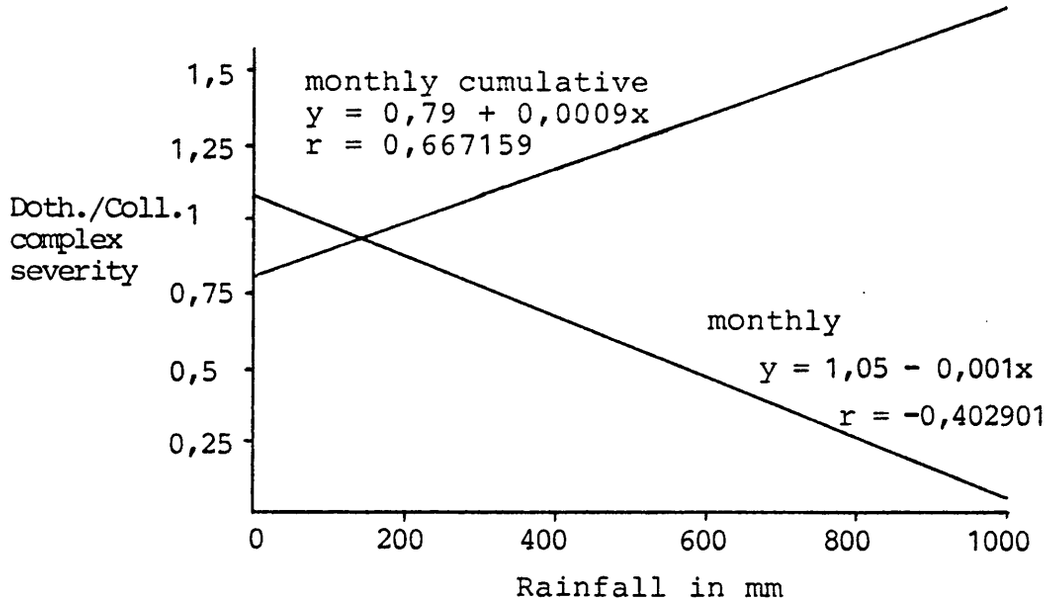

FIG. 30. - The correlation between Dothiorella/Colletotrichum complex fruit rot and rainfall in the 1978/79 season's fruit exposure experiment.

Dothiorella/Colletotrichum complex fruit rot incidence correlated negatively to the rainfall in the monthly exposures with y = 1,05 - 0,001x and an insignificant r = -0,402901. In the monthly cumulative exposure the disease increased with rainfall and the correlation is described by y = 0,79 + 0,0009x and a non-significant correlation coefficient of r = 0,667159.

4.7 LATENT INFECTIONS BY C. GLOEOSPORIOIDES AND D. AROMATICA

A considerable amount of information was obtained in the experiment in which isolations were made from Fuerte fruit skin and fruit pedicels. The extent of the actual C. gloeosporioides and D. aromatica latent infections was monitored

throughout the summer and part of the harvest season. Results of the percentage positive isolations of these fungi as well as the correlation of these data to rainfall figures in the 1981/82 season are given in Figs. 31, 32, 33, 34, 35, 36, 37 and 38.

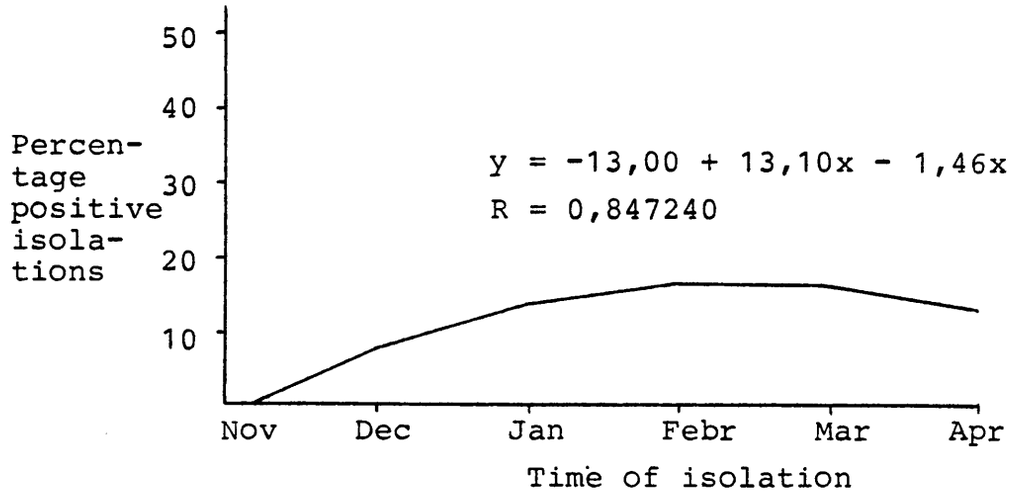

FIG. 31. - Percentage positive C. gloeosporioides isolations from Fuerte fruit skin in the 1981/82 season.

A non-linear regression model of $y = -13,00 + 13,10x - 1,46x^2$ best fitted the percentage of positive C. gloeosporioides isolations from Fuerte skin and the correlation coefficient is highly significant, R = 0,847240. The model describes a slow initial build-up of the fungus in the skin which reached a maximum in February and gradually decreased thereafter (Fig. 31).

C. gloeosporioides stem infections showed a steep increase early in the season, reached a maximum in February and decreased at a faster rate than the skin infections. The non-linear regression model to describe the correlation is $y = -24,36 + 29,60x - 4,16x^2$, with a significant R = 0,751737 (Fig. 32).

D. aromatica skin infections also fitted a non-linear regression model given by the equation of $y = -10,36 + 14,38x - 1,95x^2$, with a significant R = 0,724146. According to this model D. aromatica increased at the beginning of the rainy summer season and decreased slightly before

harvest (Fig. 33).

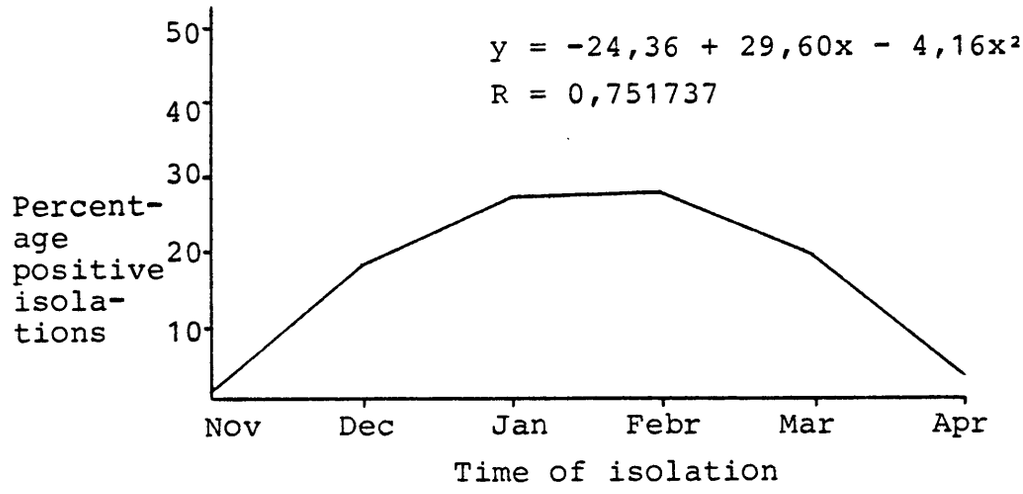

FIG. 32. - Percentage positive C. gloeosporioides isolations from Fuerte fruit pedicels in the 1981/82 season.

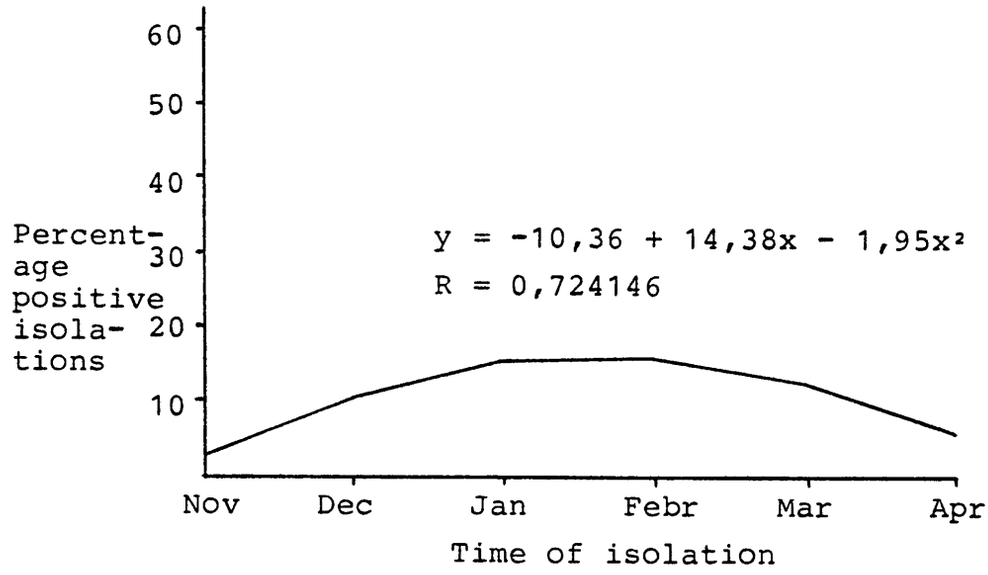

FIG. 33. - Percentage positive D. aromatica isolations from Fuerte fruit skin in the 1981/82 season.

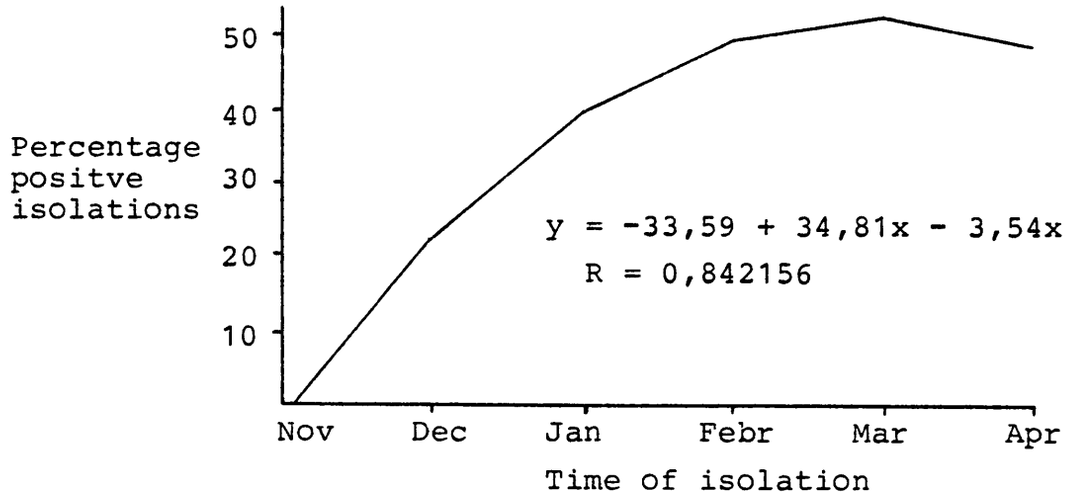

FIG. 34. - Percentage positive D. aromatica isolations from Fuerte fruit pedicels in the 1981/82 season.

Isolations from Fuerte fruit pedicels showed a rapid increase in latent D. aromatica infections which reached a maximum in March and decreased only slightly in April. The curve fitted the non-linear regression model of y = -33,59 + 34,81x - 3,54x², with a highly significant R = 0,842156 (Fig. 34).

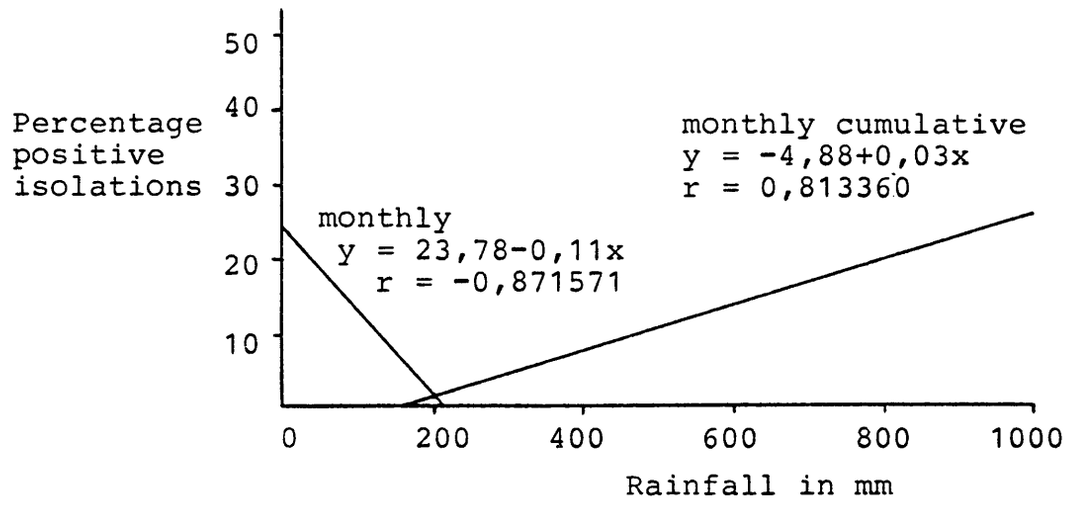

FIG. 35. - The correlation between the percentage positive C. gloeosporioides isolations from Fuerte fruit skin and rainfall in 1981/82.

The monthly rainfall figures correlated negatively with the latent infections of C. gloeosporioides, with

y = 23,78 - 0,11x and r = -0,871571 being highly significant. However, if results of the skin isolations of the fungus are correlated with the monthly cumulative rainfall, the correlation is positive and significant with y = -4,88 + 0,03x and r = 0,813360 (Fig. 35).

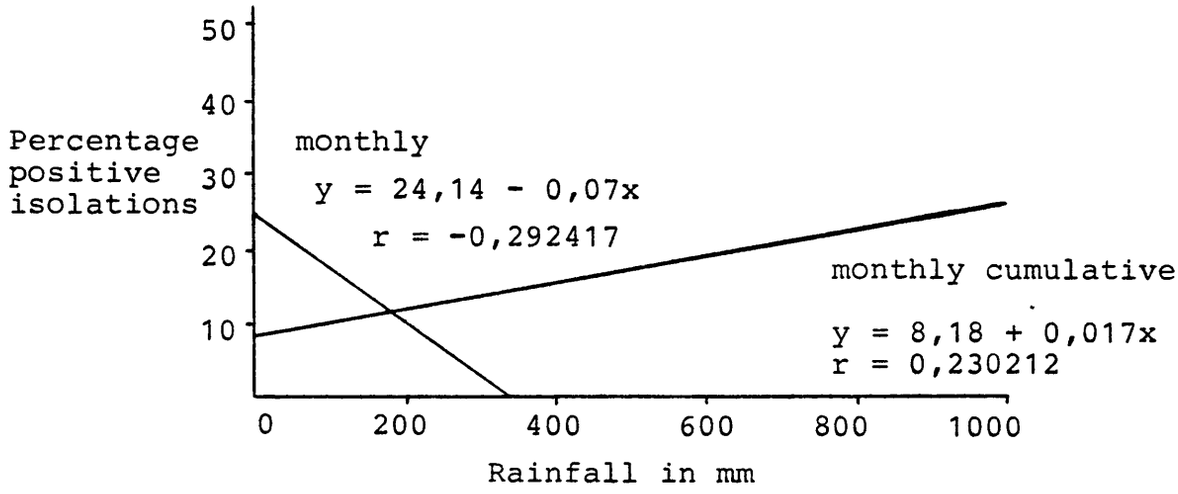

FIG. 36. - The correlation between the percentage positive C. gloeosporioides isolations from Fuerte fruit pedicels and rainfall in 1981/82.

When monthly rainfall data are correlated with the monthly positive C. gloeosporioides isolations from the fruit pedicels, again a negative correlation was found with y = 24,14 - 0,07x and a non-significant r = -0,292417. The monthly cumulative rainfall figures correlated positively with the latent infections of the fungus and may be described with the equation of y = 8,18 + 0,017x and a non-significant coefficient of r = 0,230212 (Fig. 36).

D. aromatica isolations decreased with increased rainfall if analysed on a monthly basis with y = 14,32 - 0,03x; the correlation coefficient is not significant, r = -0,302134. The monthly cumulative rainfall figures correlated positively with the amount of positive isolations of the fungus and the linear regression model for the correlation is y = 4,34 + 0,12x, with a non-significant r = 0,348220 (Fig. 37).

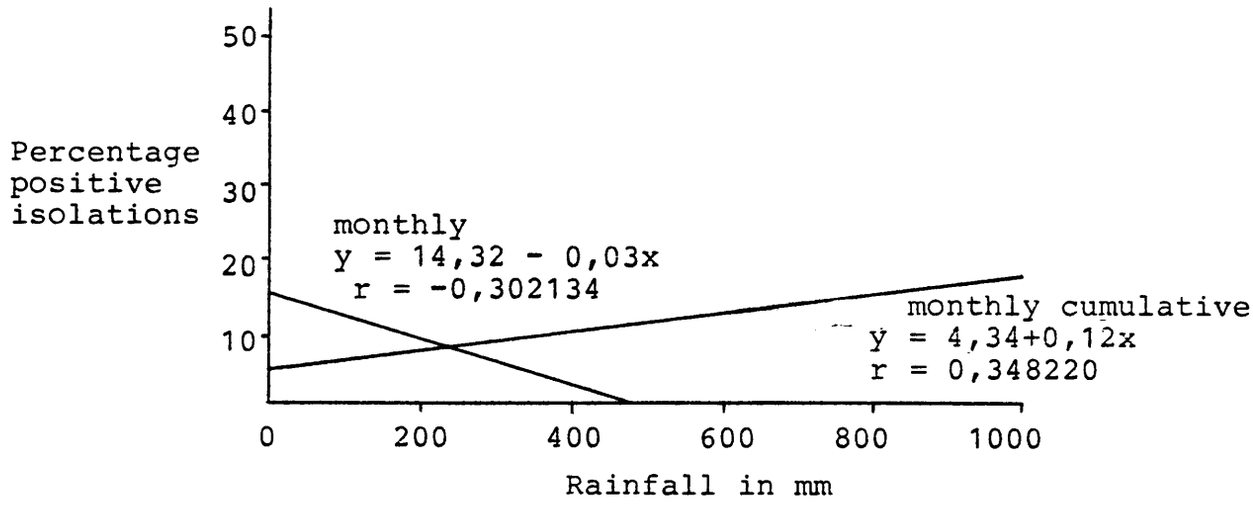

FIG. 37. - The correlation between the percentage positive D. aromatica isolations from Fuerte fruit skin and rainfall in 1981/82.

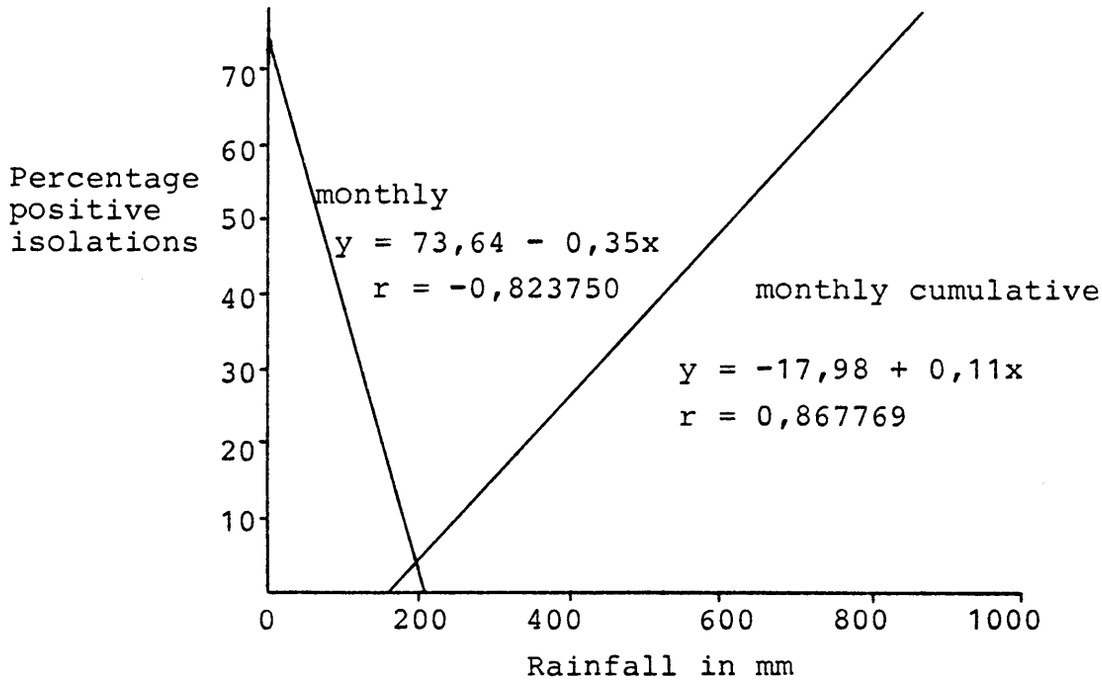

FIG. 38. - The correlation between the percentage positive D. aromatica isolations from Fuerte fruit pedicels and rainfall in 1981/82.

Latent infections of D. aromatica in the fruit pedicels correlated negatively with the monthly rainfall and can be described by the model y = 73,64 - 0,35x, with a significant correlation coefficient of r = -0,823750. The correlation between the frequency of the latent infections of the fungus and the monthly cumulative rainfall data is positive and is described by the following equation y = -17,98 + 0,11x, with a highly significant correlation coefficient of r = 0,867769 (Fig. 38).

4.8 PRE-HARVEST CHEMICAL CONTROL OF POST-HARVEST DISEASES

The first indications at Westfalia Estate that post-harvest diseases of Fuerte may be controlled with pre-harvest fungicidal sprays were obtained in the 1976/77 season's spray experiment (Table 24).

Benomyl at the lower rate significantly inhibited the development of stem-end rot and showed a non-significant reduction in anthracnose and Dothiorella/Colletotrichum complex fruit rot incidence. Fosetyl-Al had no effect against post-harvest diseases in this experiment.

Results of the 1979/80 experiment in which new additives and fungicides were tested against these diseases are presented in Table 25.

The only treatment which controlled stem-end rot statistically was Cu-oxychloride sprayed three times at 0,25% a.i. and the fungicides that also tended to reduce the disease, though at a non-significant level, were benomyl, bitertanol, Cu-hydroxide and captafol. In the case of fosetyl-Al treatment stem-end rot increased significantly. None of the treatments gave significant control of anthracnose in this experiment, on the contrary, fosetyl-Al and triple sprays of captafol resulted in a significantly increased anthracnose incidence. Dothiorella/Colletotrichum complex fruit rot was significantly controlled by benomyl, B77 + Glyodin, bitertanol and Cu-oxychloride applied twice. Although Nu-Film 17 additive tended to increase the controlling effect of benomyl, the differences between this and benomyl in

TABLE 24. - Control of post-harvest diseases on Fuerte sprayed twice during the 1976/77 season.

Treatment number	Treatments	Mean post-harvest disease severity (0 to 10 scale) (N=3200)		
		Stem-end rot	Anthracnose	Doth./Coll. complex
1	Fosetyl-Al 0,3% a.i.	0,72 a	0,28 a	0,71 a
2	Benomyl 0,02% a.i.	0,30 b	0,18 a	0,43 a
3	Benomyl 0,025% a.i.	0,54 a	0,18 a	0,38 a
4	Control	0,85 a	0,32 a	0,63 a

Means with letters a and b differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 25. - Control of post-harvest diseases on Fuerte sprayed pre-harvest with fungicides in the 1979/80 season.

Treatment number	Number of sprays	Treatments	Mean disease severity (0 to 10 scale) (N=9 600)		
			Stem-end rot	Anthracnose	Doth./Coll.
1	2	Benomyl 0,025% a.i. + Nu Film 0,02%	0,10 cd	0,66 c	0,80 d
2	2	Benomyl 0,025% a.i. + Plyac 0,02%	0,15 cd	1,08 bc	1,14 d
3	2	Benomyl 0,025% a.i. + Solvaid 0,03%	0,17 c	1,08 bc	0,73 d
4	2	B 77 150 ppm + Glyodin 0,125% + Nu Film	0,55 ab	2,03 a	1,45 cd
5	2	Bitertanol 0,01% a.i. + Nu Film 0,02%	0,11 cd	0,43 c	1,13 d
6	2	Cu-hydroxide 0,15% a.i. + Nu Film 0,02%	0,29 bc	1,06 bc	2,04 bc
7	2	Cu-oxychloride 0,25% a.i. + Nu Film 0,02%	0,12 cd	0,49 c	1,19 d
8	3	Cu-oxychloride 0,25% a.i. + Nu Film 0,02%	0,01 d	0,56 c	2,14 bc
9	2	Captafol 0,16% a.i. + Nu Film 0,02%	0,16 cd	1,30 abc	2,07 bc
10	3	Captafol 0,16% a.i. + Nu Film 0,02%	0,45 bc	1,58 ab	2,73 ab
11	2	Fosetyl-Al 0,3% a.i. + Nu Film 0,02%	0,80 a	1,76 ab	3,02 a
12	-	Control	0,39 bc	0,49 c	2,46 ab

Means with letters a, b, c, and d differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 26. - Control of post-harvest diseases on Fuerte sprayed twice in the 1980/81 season.

Treatment number	Time of application	Treatments	Mean disease severity (0 to 10 scale) (N = 1 250)		
			Stem-end rot	Anthracnose	Doth./Coll.
1	Nov. 1980 Jan. 1981	Benomyl 0,025% a.i. + Nu Film 0,02% Benomyl 0,025% a.i. + Nu Film 0,02%	0,50 a	0,42 a	2,21 a
2	Nov. 1980 Jan. 1981	Captafol 0,08% a.i. + Nu Film 0,02% Captafol 0,08% a.i. + Nu Film 0,02%	0,14 b	0,21 ab	1,39 bc
3	Nov. 1980 Jan. 1981	Captafol 0,08% a.i. + Nu Film 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 0,02%	0,14 b	0,16 b	1,01 cd
4	Nov. 1980 Jan. 1981	Captafol 0,08% a.i. + Nu Film 0,02% Benomyl 0,025% a.i. + Nu Film 0,02%	0,02 b	0,10 b	0,69 d
5	-	Control	0,21 a	0,17 b	1,91 ab

Means with letters a, b, c and d differ statistically at 0,05 level (Duncan's multiple range test)

TABLE 27. - Control of post-harvest diseases on Fuerte sprayed pre-harvest in the 1981/82 season.

Treatment number	Time of application	Treatments	Mean disease severity (0 to 10 scale) (N = 3 240)		
			Stem-end rot	Anthracnose	Doth./Coll.
1	Nov. 1981 Jan. 1982	Benomyl 0,025% a.i. + Nu Film 0,02% Benomyl 0,025% a.i. + Nu Film 0,02%	0,00 a	0,05 a	2,01 ab
2	Nov. 1981 Jan. 1982	Cu-hydroxide 0,23% a.i. + Nu Film 0,02% Cu-hydroxide 0,23% a.i. + Nu Film 0,02%	0,01 a	0,00 a	1,74 b
3	Nov. 1981 Jan. 1982	Cu-oxychloride 0,25% a.i. + Nu Film 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 0,02%	0,01 a	0,05 a	1,63 b
4	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 0,02% Cu-hydroxide 0,23% a.i. + Nu Film 0,02%	0,00 a	0,01 a	1,52 b
5	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 0,02%	0,01 a	0,01 a	1,58 b
6	Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 0,02% Cu-oxychloride 0,25% + Bitertanol 0,0125% a.i. + Agridex 0,1%	0,00 a	0,00 a	0,84 c
7	Sept.1981 Nov. 1981 Jan. 1982	Captafol 0,08% a.i. + Nu Film 0,02% Captafol 0,08% a.i. + Nu Film 0,02% Cu-oxychloride 0,25% a.i. + Nu Film 0,02%	0,01 a	0,00 a	1,41 b
8	Nov. 1981 Jan. 1982	Prochloraz 0,04% a.i. + Nu Film 0,02% Prochloraz 0,04% a.i. + Nu Film 0,02%	0,01 a	0,14 a	2,49 a
9	-	Control	0,00 a	0,06 a	1,84 b

Means with letters a, b and c differ statistically at 0,05 level (Duncan's multiple range test)

mixtures with other additives were not statistically significant.

Stem-end rot was not controlled by benomyl in the 1980/81 season's experiment, however, captafol alone and in programmes with Cu-oxychloride or benomyl reduced the disease significantly. None of the treatments controlled anthracnose effectively, in fact, benomyl sprays significantly increased the incidence of anthracnose. Dothiorella/Colletotrichum complex fruit rot was significantly less in captafol plus Cu-oxychloride and captafol plus benomyl treatments, while benomyl and captafol alone gave unsatisfactory results against the disease.

Results of the most recent experiment conducted in the 1981/82 season are presented in Table 27.

The incidence of stem-end rot and anthracnose in 1981/82 was exceptionally low and the statistical analysis showed no differences between the untreated control and fruit that was sprayed in the experiment. There was only one treatment, namely a spray programme, in which captafol was applied in November, 1981 and was followed up with a Cu-oxychloride and bitertanol combined spray, that gave a significant control of the Dothiorella/Colletotrichum complex fruit rot. Prochloraz sprays resulted in a significantly more severe complex fruit rot incidence compared to the control.

4.9 POST-HARVEST CONTROL OF POST-HARVEST DISEASES

4.9.1 Effect of the length of the ripening time

The effect of the length of ripening time (from store until eating-ripe stage) on the occurrence of post-harvest diseases was investigated in 1982 and the results are illustrated in Figs. 39, 40 and 41.

Stem-end rot incidence increased with longer ripening time and it is described with the linear regression model of $y = -1,08 + 0,19x$ and a significant correlation coefficient of $r = 0,428076$ (Fig. 39).

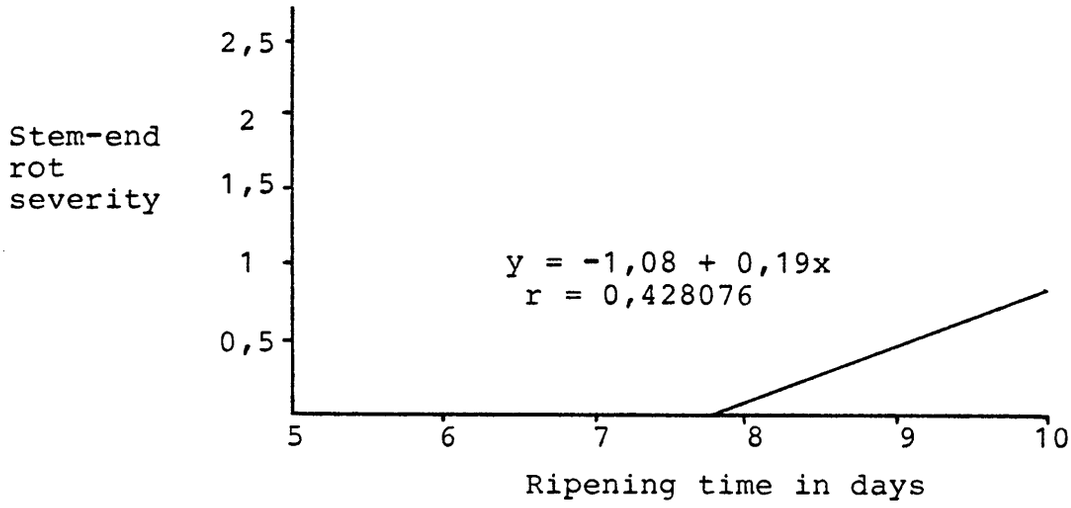

FIG. 39. - The effect of the length of ripening time on the occurrence of stem-end rot of Fuerte in 1982.

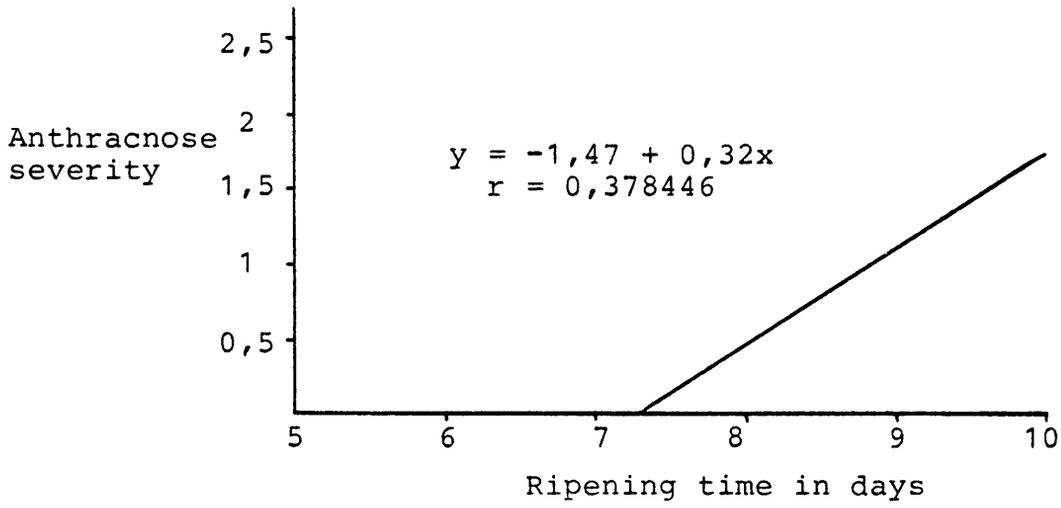

FIG. 40. - The effect of the length of the ripening time on the occurrence of anthracnose of Fuerte in 1982.

The severity of anthracnose increased significantly with the increase in the length of ripening time with $y = -1,47 + 0,32x$ and $r = 0,378446$.

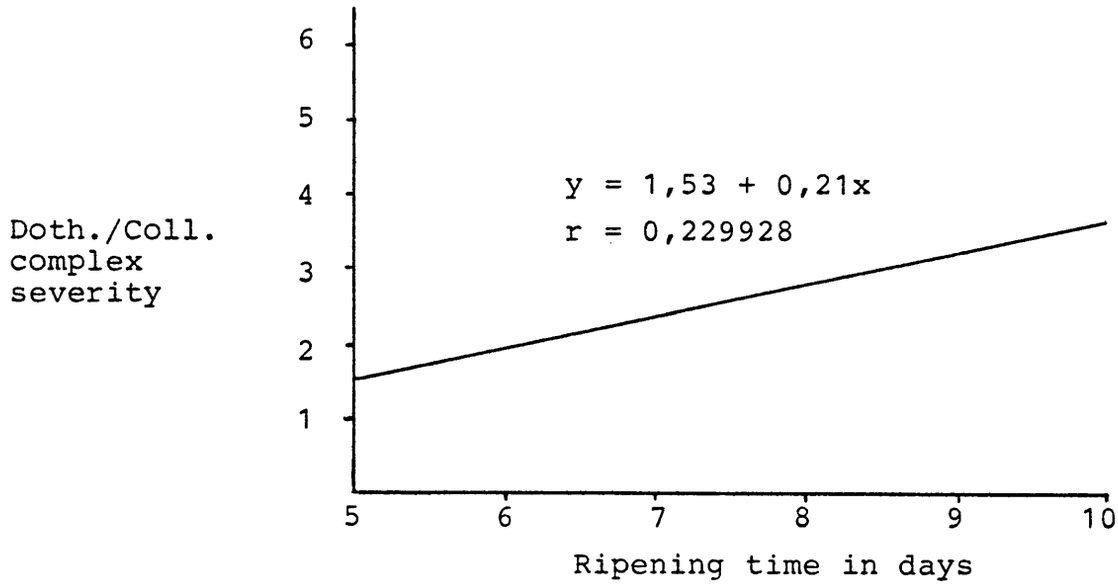

FIG. 41. - The effect of the length of the ripening time on the occurrence of Dothiorella/ Colletotrichum complex fruit rot of Fuerte in 1982.

The severity of Dothiorella/Colletotrichum complex fruit rot also showed an increase with increased ripening time and is described by y = 1,53 + 0,21x, though the correlation coefficient is not significant, r = 0,229928.

4.9.2 Effect of the removal of the fruit pedicel

The effect of removed pedicels on the incidence of post-harvest diseases of Fuerte was investigated in 1979 and the results are given in Table 28.

Stem-end rot incidence was significantly reduced by the removal of the fruit pedicel, but at the same time anthracnose increased significantly on the stemless fruit and this may be due to the statistically significant longer ripening time for the fruit without stems. There was no difference between the two treatments with regard to Dothiorella/Colletotrichum complex fruit rot.

4.9.3 Effect of rain at harvest

The influence of rain at the time of harvest was in-

TABLE 28. - Post-harvest diseases of stemless fruit and fruit with half cm long pedicel after cold storage in 1979.

Treatments	Mean disease severity (0 to 10 scale) (N=260)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Stemless fruit	0,18 b	0,47 a	0,13 a	6,0 a
Fruit with 0,5cm long pedicel	0,54 a	0,09 b	0,11 a	4,7 b

Means with letters a and b differ statistically at 0,05 level

TABLE 29. - Post-harvest disease incidence on wet and dry harvested fruit in 1978.

Treatments	Mean disease severity (0 to 10 scale) (N=280)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Wet harvest fruit	0,35 a	0,67 a	2,52 a	7,0 a
Dry harvest fruit	0,06 b	0,47 a	1,12 b	6,5 a

Means with letters a and b differ statistically at 0,05 level

vestigated by picking after rain while fruit was still wet and picking again the same day when fruit was dry (Table 29).

Stem-end rot and Dothiorella/Colletotrichum complex fruit rot were significantly more severe on fruit that was picked wet than fruit harvested dry. There were no statistical differences between the ripening times of the dry and wet harvested fruit.

4.9.4 Effect of moisture condensated on the fruit

The effect of moisture on the fruit that was induced during the pre-cooling of Fuerte in the packhouse was examined and results are given in Table 30.

Fruit moistened by condensation tended to be more severely affected by stem-end rot and anthracnose, but this was not significant. The increase in Dothiorella/Colletotrichum complex fruit rot was statistically significant on the moist fruit. Ripening time showed no difference in the two treatments.

4.9.5 Effect of washing the fruit in the packhouse

The occurrence of post-harvest diseases was recorded on fruit washed with tap water in the packhouse and compared with diseases on fruit that was handled dry (Table 31).

There was a non-significant tendency for more anthracnose to occur on the washed fruit and a statistically significant increase in stem-end rot and Dothiorella/Colletotrichum complex fruit rot was induced by washing.

4.9.6 Effect of sealing the cut end of the pedicel

Results of the experiment in which fruit was harvested and immediately sealed at the cut end of the pedicel with wax plus benomyl and TBZ is presented in Table 32.

The only post-harvest disease which showed a significant decrease after treating the stem pedicel with wax plus fungicides was stem-end rot. Anthracnose, Dothiorella/

TABLE 30. - Post-harvest disease incidence on dry fruit and fruit moistened by condensation due to pre-cooling in 1980.

Treatments	Mean disease severity (0 to 10 scale) (N=350)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Moist fruit	0,03 a	0,20 a	0,46 a	6,8 a
Dry fruit	0,01 a	0,04 a	0,12 b	6,9 a

Means with letters a and b differ statistically at 0,05 level

TABLE 31. - Post-harvest disease incidence on dry fruit and fruit washed with tap water in 1978.

Treatments	Mean disease severity (0 to 10 scale) (N=280)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Washed fruit	0,43 a	0,68 a	2,52 a	7,0 a
Dry fruit	0,06 b	0,44 a	1,12 b	6,5 a

Means with letters a and b differ statistically at 0,05 level

Colletotrichum complex fruit rot and ripening time remained statistically unchanged with the pedicel treatment.

The results of the 1979 experiment with pedicel treatment on the incidence of the various pathogens in Fuerte stem-end rot are presented in Table 33.

Waxing of fruit in the packhouse aggravated stem-end rot if compared to untreated control. The sealing of the cut end of the pedicel with TAG wax plus 0,1% a.i. benomyl plus 0,2% a.i. TBZ in the orchard immediately after picking greatly reduced the number of fruits with stem-end rot.

No T. pseudotrichia was isolated from pedicel sealed fruit indicating that the fungus is a wound pathogen.

4.9.7 Effect of cellophane wrapping

The wrapping of Fuerte fruit with cellophane was compared to unwrapped fruit in respect of post-harvest disease incidence (Table 34).

Although the wrapping of fruit resulted in a higher stem-end rot, anthracnose and Dothiorella/Colletotrichum complex fruit rot incidence, the increase was not significant due to large variation within the treatments.

4.9.8 Effect of waxing

The effect of wax (TAG) applied to Fuerte fruit on post-harvest diseases was investigated in 1982 and the results are given in Table 35.

There was a non-significant increase in stem-end rot incidence on the waxed fruit, while a significant increase in anthracnose and Dothiorella/Colletotrichum complex fruit rot severity was induced by the use of wax. It also took significantly longer for the waxed fruit to ripen.

4.9.9 Effect of fungicides added to the wax

The changes in post-harvest diseases of Fuerte by the

TABLE 32. - Post-harvest disease incidence on fruit sealed at the cut end of the pedicel and on untreated fruit in 1979.

Treatments	Mean disease severity (0 to 10 scale) (N=280)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Fruit with sealed pedicel	0,08 b	0,18 a	0,09 a	8,0 a
Control	0,84 a	0,18 a	0,06 a	7,7 a

Means with letters a and b differ statistically at 0,05 level

TABLE 33. - The incidence of stem-end rot pathogens on Fuerte fruit waxed in the packhouse or sealed at the cut end of the pedicel and on untreated fruit in 1979.

Treatments	Percent fruit with stem-end rot	Percentage occurrence (N = 360)			
		T. pseudotrichia	C. gloeosporioides	D. aromatica	P. perseae
TAG waxed in packhouse	33	78	18	2	2
Pedicel sealed with TAG wax + 0,1% a.i. benomyl + 0,2% a.i. TBZ	3	0	100	0	0
Untreated control	16	90	5	5	0

TABLE 34. - Post-harvest disease incidence on cellophane wrapped and unwrapped fruit in 1980.

Treatments	Mean disease severity (0 to 10 scale)(N=280)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Cellophane wrapped fruit	0,20 a	0,37 a	1,09 a	6,8 a
Control	0,01 a	0,10 a	0,63 a	6,0 b

Means with letters a and b differ statistically at 0,05 level

TABLE 35. - Post-harvest disease incidence on waxed and unwaxed fruit in 1982.

Treatments	Mean disease severity (0 to 10 scale) (N=280)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
Waxed fruit	0,15 a	0,19 a	1,94 a	6,5 a
Control	0,01 a	0,05 b	1,47 b	5,2 b

Means with letters a and b differ statistically at 0,05 level

addition of benomyl and TBZ to TAG wax were evaluated in 1982 (Table 36).

Stem-end rot incidence was not statistically different between the various treatments. Anthracnose was aggravated greatly by waxing and this significant increase was reduced by the addition of fungicides. Dothiorella/Colletotrichum complex fruit rot was also reduced by fungicides in the wax, but the reduction was not significant. Ripening time was longer with application of wax regardless whether fungicides were added to it or not.

TABLE 36. - Post-harvest diseases on Fuerte fruit waxed with and without the addition of fungicides in 1982.

Treatments	Mean disease severity (0 to 10 scale) (N=360)			Ripening time in days
	Stem-end rot	Anthracnose	Doth./Coll. complex	
TAG wax	0,15 a	0,19 a	1,94 a	6,5 a
TAG wax + 0,05% a.i. benomyl + 0,2% a.i. TBZ	0,08 a	0,09 b	1,60 ab	6,7 a
Control	0,01 a	0,05 b	1,47 b	5,2 b

Means with letters a and b differ statistically at 0,05 level

5 - DISCUSSION

Post-harvest losses caused by stem-end rot, anthracnose and Dothiorella/Colletotrichum complex fruit rot are closely correlated with the oil content of avocado fruit. Fuerte fruit with a low oil content early in the picking season is more prone to post-harvest diseases than fruit harvested later in the season when the oil content is higher (Figs. 16, 17 and 18). This aspect of post-harvest disease incidence on avocados has not previously been clarified.

Stem-end rot is the most devastating post-harvest disease of Fuerte avocados particularly during early harvesting at Westfalia Estate. This is not in accordance with the statement made by Jacobs (1974) regarding the generally low occurrence of the disease in the avocado industry as a whole.

Observations on losses caused by anthracnose at Westfalia Estate confirm earlier observations on the importance of this disease in South Africa (Jacobs, 1974; Brodrick et al., 1974).

Data on the losses caused by Dothiorella fruit rot in combination with the anthracnose fungus, constitute the first record of this complex and its relative importance in South Africa.

The symptom description of stem-end rot in the literature was found to be incomplete (Horne, 1931). Observations on stem-end rot in this study revealed that symptoms are frequently pathogen specific and an experienced person can therefore often identify the causal organism successfully on the basis of symptoms. Additional information on the symptoms in respect to colour of rot, discolouration of vascular bundles and external sporulation by the pathogen associated with stem-end rot are given for the first time in tabulated form (Table 13).

Although there is little to add to the existing descriptions of typical anthracnose symptoms (Stevens, 1922; Ocfemia and Agati, 1925; Horne, 1934; Zentmyer, 1953), it was found essential to classify the superficial anthracnose caused by C. gloeosporioides together with Dothiorella fruit rot caused

by D. aromatica as a "Dothiorella/Colletotrichum complex fruit rot". The fruit rot symptoms caused by Dothiorella and the superficial form of anthracnose are usually not symptomatically distinguishable at Westfalia Estate. The problem of separating Dothiorella fruit rot from anthracnose on the basis of symptoms only has also been encountered in Australia (Muirhead, 1977).

Isolations from stem-end rot diseased avocados revealed that the following organisms are involved in the disease at Westfalia Estate: T. pseudotrichia, C. gloeosporioides, D. aromatica, P. perseae, L. theobromae, P. versicolor, F. decemcellulare, F. sambucinum, F. solani, R. stolonifer and D. setariae. Of these, T. pseudotrichia, R. stolonifer, F. decemcellulare, F. sambucinum, D. setariae and P. versicolor have not previously been reported from avocado stem-end rot. D. aromatica, P. perseae and F. decemcellulare are reported for the first time in South Africa from any host. Several species of Alternaria and Cladosporium were also isolated from stem-end rot, confirming the findings of Horne (1934). In addition species of the genera Epicoccum and Nigrospora were also encountered, but none of these isolates showed pathogenicity towards avocados in subsequent inoculations.

Anthracnose is caused by C. gloeosporioides at Westfalia Estate and it is a relatively easy organism to isolate and identify.

Some controversy exists regarding the identity of Dothiorella species on avocados. It was reported from California that D. gregaria (Botryosphaeria ribis) is the cause of a fruit rot disease and also stem-end rot (Horne, 1934). Zentmyer (1946; 1953) quoted the same fungus as the cause of Dothiorella fruit rot. According to Ivory (1967), however, the asexual stage of B. ribis is Fusicoccum tingens Doig., which is very similar to, but not identical with Dothiorella gregaria Sacc. Muirhead (1977) found in Australia that D. aromatica (no sexual stage is known for the fungus) is the cause of avocado fruit rot and stem-end rot. During the present study the latter species was found in Dothiorella fruit and stem-end rot of avocados at Westfalia Estate.

Several of the post-harvest pathogens rapidly lose their capability to sporulate in culture on the commonly used media such as PDA, in particular D. aromatica, P. perseae, L. theobromae, F. sambucinum and the Stilbella form of T. pseudotrichia. The use of sterile avocado fruit segments in the culturing of post-harvest disease pathogens proved to be a highly successful method for inducing the production of conidia by these fungi and maintaining their ability to sporulate. The technique has been used previously for avocado pathogens by Binyamini and Schiffmann-Nadel (1972).

It was found also for the first time that the incidence of certain post-harvest diseases shows variation relating to the position of the fruit on the trees. There is a non-significant tendency for Dothiorella/Colletotrichum complex fruit rot to be most serious on fruit from the northern side of the trees. Anthracnose incidence was generally low on all fruit in the survey with no appreciable differences between fruit harvested from the different aspects. Stem-end rot was significantly more prevalent on fruit from the eastern aspect of the trees while fruit from the southern and western sides were the least affected (Table 14).

It was also interesting to find that post-harvest diseases were more severe on trees least affected by root rot and that the incidence of these diseases decreased with the retrogression of the trees (Figs. 19, 20 and 21). A possible explanation for this phenomenon is the humid microclimate which is conducive to infections by post-harvest diseases on the healthy trees due to the full leaf coverage.

During an investigation on the frequency of various pathogens it became evident that T. pseudotrichia is the most common fungus causing stem-end rot of Fuerte, followed by C. gloeosporioides, P. perseae, D. aromatica and F. decemcellulare (Table 15). Although L. theobromae is seldom seen at Westfalia Estate and was not isolated in this experiment, it was recovered from Fuerte stem-end rot in other isolations. The 28 days cold storage of fruit at 6°C to simulate export conditions greatly decreased the incidence of T. pseudotrichia, but at the same time increased the relative frequency of other

pathogens (Table 15).

In contrast to the stem-end rot organisms of Fuerte, the major pathogen causing stem-end rot of Edranol fruit was D. aromatica, while C. gloeosporioides was the second most common (Table 16).

Findings on the source of inoculum of post-harvest disease fungi are in agreement with earlier reports (Ocfemia and Agati, 1925; Horne and Palmer, 1935). Most of these organisms were found in great numbers on dead leaves, branches and fruit of avocado trees. A survey showed that T. pseudotrichia was the most common pathogen occurring on dead Fuerte branches and twigs (Table 17).

Pre-harvest inoculation studies with spore suspensions of the various pathogens showed that only C. gloeosporioides and D. aromatica can cause both stem-end rot and fruit rot through unwounded epidermis, while L. theobromae can penetrate and rot fruit from the pedicel end and F. decemcellulare induces small fruit spots. All the other organisms isolated from stem-end rot lesions are believed to be secondary invaders through the cut pedicel or pedicel scar (Table 18). The active pathogenic invasion of fruit by C. gloeosporioides found in this study, is in contrast to the statements by Stevens (1922) and Zentmyer (1953) who believed that the fungus was a secondary invader and unable to enter unwounded fruit. However, direct penetration of avocado fruit by C. gloeosporioides has been reported previously by Horne (1926a), Wardlaw et al. (1939), Ruehle (1943b) and Binyamini and Schiffmann-Nadel (1972).

Post-harvest inoculations through the pedicel scar of debuttoned fruit showed that the most virulent stem-end rot organisms were L. theobromae, C. gloeosporioides, D. aromatica and R. stolonifer (Tables 19 and 21). The same fungi were also the most potentially pathogenic organisms in inoculations through 0,5cm long fruit pedicels, with the exception of R. stolonifer, which was unable to penetrate fruit via the pedicel (Tables 20 and 22).

Post-harvest inoculations through wounds inflicted on the side of the fruit revealed that the most pathogenic

fruit rotting organisms were R. stolonifer, L. theobromae and C. gloeosporioides (Table 23). Inoculated fruit softened more rapidly than control fruit. A similar phenomenon was observed in Israel by Zauberman and Schiffmann-Nadel (1974) on avocados inoculated with F. solani. R. stolonifer has caused some concern in California and was first described as an avocado fruit rot pathogen by Horne (1926a; 1926b). He stated that it is the most active pathogen involved in avocado spoilage and that the fungus is primarily a wound pathogen. This was confirmed in the present study. The fungus was recovered from cold stored avocados at Westfalia Estate and this points to the necessity of cleaning and disinfecting cold stores before the start of the harvest season.

A little-known avocado fruit rot caused by Trichothecium roseum Link was described by Yale and Johnstone in 1951. This fungus has been seen occasionally on overripe avocados at Westfalia Estate, but since it has no commercial significance, it was not included in the experiments.

Studies made on the critical infection period for post-harvest diseases under natural conditions in the 1977/78 season showed that all post-harvest diseases increased with increase in exposure time (Figs. 22, 23 and 24). Correlations between exposure time and post-harvest disease incidence in the 1978/79 season were non-significant, but still showed a tendency similar to the previous season's results (Figs. 25, 26 and 27). Results suggest that stem-end rot and Dothiorella/Colletotrichum complex fruit rot are more serious when arising from infection which occurs late in the growing season, whereas anthracnose remains at a relatively constant level throughout the summer.

There are indications that monthly rainfall is not a suitable indicator for forecasting post-harvest disease incidence and that the non-significant but rational correlations between rainfall on a cumulative basis and monthly cumulative exposure data, are due to the cumulative time factor (Figs. 28, 29 and 30). These results confirm the findings of Stevens (1922) who reported that young avocado fruit show no indication of anthracnose infection and that the disease is

rarely observed until after the fruit is half mature. According to this information, fungicide sprays applied later in the growing season should be more effective against post-harvest diseases than early sprays. The importance of late season sprays against Dothiorella fruit rot was also emphasized by Horne and Palmer (1935).

Isolations from fruit skin and pedicels gave useful information on the natural latent infections of Fuerte fruit by C. gloeosporioides and D. aromatica. The incidence of C. gloeosporioides increased slowly in the skin, reaching a maximum in February - March and decreased only slightly during the peak harvest time of Fuerte in April. In pedicels the infections increased more rapidly, reaching a maximum in January - February and then decreased considerably until April (Figs. 31 and 32). Latent infections of D. aromatica in fruit skin increased slowly at the beginning of the summer, reaching a maximum in February and then decreased again until April (Fig. 33). D. aromatica in the fruit pedicel showed a rapid increase early in the summer with a maximum in March and only a slight decrease in April (Fig. 34).

In this study the correlation between latent infection of these fungi and the rainfall on a monthly basis proved to be an unreliable criterion for forecasting disease severity and the significant correlations between infection and the monthly cumulative rainfall is again evidently a function of time (Figs. 35, 36, 37 and 38). This is in agreement with the findings of Peterson (1978) who showed that the natural infection of avocado fruit by these fungi was related to rainfall, but that the duration of rainy periods was a more significant factor than the total rainfall.

The decrease in the latent infections by C. gloeosporioides and D. aromatica later in the picking season as indicated by direct isolations (Figs. 31, 32, 33 and 34) and decrease in the incidence of the post-harvest diseases caused by them later in the harvest season (Figs. 16, 17 and 18), are in complete agreement.

The latent phase in the pathogenesis of the anthracnose fungus is a well documented fact previously described by

Wardlaw et al. (1939), Ruehle (1943b) and Binyamini and Schiffmann-Nadel (1972). It is apparently due to the presence of antifungal substances in hard avocados which break down during the ripening of the fruit (Prusky, 1981 - unpublished). It seems likely that the longer the harvest time is prolonged after the last infections took place, the longer these fungi remain latent and the more of these latent infections will become non-infective.

Numerous fungicides and additives were tested in the pre-harvest chemical spray experiments for the control of post-harvest diseases.

Due to the high rainfall experienced at Westfalia Estate it was decided that Nu Film 17 (Pinolene) should be used as a standard with all fungicide sprays since this additive effectively reduced the washing-off of fungicides from the fruit surface (Table 25). It was first shown in 1976/77 that benomyl gave satisfactory control of stem-end rot and a non-significant reduction of anthracnose and Dothiorella/Colletotrichum complex fruit rot (Table 24).

Cu-oxychloride controlled stem-end rot and Dothiorella/Colletotrichum complex fruit rot, while benomyl, B 77 plus glyodin and bitertanol were effective against Dothiorella/Colletotrichum complex fruit rot in the 1979/80 season (Table 25).

Captafol, captafol plus Cu-oxychloride and captafol plus benomyl controlled stem-end rot and Dothiorella/Colletotrichum complex fruit rot in the 1980/81 season (Table 26). Captafol in combination with Cu-oxychloride and bitertanol was the best treatment against the Dothiorella/Colletotrichum complex fruit rot in 1981/82 (Table 27). The above results are in accordance with the findings of Stevens (1922), Palmer (1932), Horne and Palmer (1935), Ruehle (1942; 1943b), Zauberman et al. (1974), McMillan (1976), Allen (1977), Peterson and Inch (1980) and Kotzé et al. (1981), who all found that post-harvest diseases of avocados can be controlled by pre-harvest fungicide sprays.

An increase in post-harvest disease incidence was observed in some experiments following sprays with benomyl (Table 26),

captafol, fosetyl-Al (Table 25) and prochloraz (Table 27). Since benomyl has been used for several years in avocado orchards at Westfalia Estate, it is possible that the pathogens have developed an increased tolerance towards the chemical. The situation could have been aggravated by a selective inhibition of the microflora antagonistic towards post-harvest disease pathogens by benomyl as well as by the other fungicides ("boomerang effect").

A large number of experiments were conducted during the investigation of post-harvest control of post-harvest avocado diseases. Tremendous variations were evident in the development of post-harvest diseases on fruit and these variations were influenced by a number of factors including the physiological composition of the fruit, maturity, position of the fruit on trees, root rot severity, differences between trees, orchards, climatic areas etc. These variations often resulted in statistically non-significant readings of experiments, committing the author to use tendencies as guidelines. In this study only selected experiments are included which are believed to be representative of a general trend obtained with some of the post-harvest treatments.

A positive correlation was found between the length of the ripening process of avocados and the severity of post-harvest diseases (Figs. 39, 40 and 41). This effect appears to be a basic principle in the plant-disease relationship and it has been seen in many of the post-harvest treatments that influenced the ripening process of the fruit. It also confirms reports by Hatton and Reeder (1972) and Oudit and Scott (1973) who found a greatly increased incidence of anthracnose when ripening time was extended. It was also recognised by Jacobs (1974) that artificial extension of shelf-life resulted in considerable stem-end rot damage.

The removal of the pedicel of Fuerte fruit reduced the incidence of stem-end rot, probably by removing some latent infections present in the pedicel, but at the same time it increased anthracnose, possibly due to the resultant longer ripening time (Table 28). This extended shelf-life brought about by the removal of the fruit pedicel is in contrast to

results obtained by Tingwa and Young (1975) who concluded that removal of the pedicel shortens the ripening time of avocado fruit in California.

Moisture of any source on the fruit leads to an increase in post-harvest disease incidence (Tables 29, 30 and 31). It is emphasized that the rapid drying of wet fruit in the packhouse is not likely to reduce the problem since moisture is retained in the lenticels where many of the latent infections are located (Horne and Palmer, 1935).

The sealing of the cut end of the fruit pedicel with wax plus fungicides is an effective way of controlling stem-end rot (Table 32). This finding agrees with the theory of Horne (1931) who recommended post-harvest pedicel treatments for stem-end rot control. It has been proved experimentally that the sealing of pedicels with wax plus fungicides controls T. pseudotrichia very effectively (Table 33).

The wrapping and waxing of fruit is widely practiced in the South African avocado export industry and it is done to ensure a longer shelf-life on the overseas market. While these practices indeed slow down the ripening process of the avocado fruit to some extent, they have a detrimental effect on the quality of the fruit by inducing an increase in the severity of post-harvest diseases (Tables 34 and 35). It has been shown that some of these treatments considerably reduce the rate of loss in fruit mass (Kotzé and Kuschke, 1978) by creating a closed environment for the fruit. This increases humidity around the fruit which may provide a favourable microclimate for the development of post-harvest diseases. The effect of waxing and/or wrapping of fruit on post-harvest diseases is thus twofold, it extends shelf-life and permits a longer period for the pathogens to cause damage and also creates a microclimate conducive to the development of these diseases. The increased pathological losses could only partially be reduced by the addition of fungicides to the wax (Table 36).

As far as the overall chemical control of post-harvest diseases is concerned, it is emphasized that successful control measures will have to include pre-harvest fungicidal sprays

which reduce the amount of latent infections in the fruit. Furthermore, some form of post-harvest treatment will have to be developed to control the remaining latent infections. The latter implies post-harvest treatments in the packhouse at one specific point where all the fruit pass through. No effective or commerically applicable method of control has been developed to date for the post-harvest treatment of avocados and this certainly remains one of the most challenging fields for future investigation.

SUMMARY

Cercospora spot disease of avocados caused by Pseudocercospora purpurea (Cke) Deighton is described for the first time in South Africa, where it was found to be the most important pre-harvest fruit disease at Westfalia Estate, in the North Eastern Transvaal (longitude 30°10' and latitude 23°45'). Losses caused by the disease were investigated in relation to rainfall, cultivar, time of harvest, root rot severity of trees and the position of the fruit on the tree.

A detailed description of Cercospora spot symptoms is given. The analysis of spore trap results and weather data produced statistical models suitable for forecasting conidia production by the pathogen. These models may be used to determine high risk infection periods, thereby facilitating accurate timing of fungicidal sprays.

Infections taking place early in the growing season were found to give the highest disease incidence at harvest. There is a latent period of about three months in duration in the disease cycle.

Cercospora spot disease can be controlled by benomyl and also by some of the non-benzimidazole fungicides such as captafol, Cu-hydroxide and Cu-oxychloride. It was found that several years' continued use of benomyl results in a significant decrease in the efficacy of the fungicide against the disease.

On the basis of symptoms and the pathogens involved, the following post-harvest diseases were recognised at Westfalia Estate:

Stem-end rot caused by Thyronectria pseudotrichia (Schw.) Seeler, Colletotrichum gloeosporioides (Penz.) Sacc., Dothiorella aromatica (Sacc.) Petr. and Syd., Phomopsis perseae Zerova, Fusarium decemcellulare Brick and to a lesser extent Pestalotiopsis versicolor (Speg.) Steyart, Lasiodiplodia theobromae (Pat.) Griff. and Maubl., Rhizopus stolonifer (Ehr. ex Fr.) Lind., Fusarium sambucinum Fuckel, Fusarium solani (Mart.) Sacc. and Drechslera setariae

(Sawada) Subram. and Jain.

Anthracnose caused by Colletotrichum gloeosporioides (Penz.) Sacc.

Dothiorella/Colletotrichum complex fruit rot caused by Dothiorella aromatica (Sacc.) Petr. and Syd. and Colletotrichum gloeosporioides (Penz.) Sacc.

Losses caused by these post-harvest diseases were studied with respect to oil content of fruit, root rot severity of trees and the position of the fruit on the tree. A comprehensive description of the post-harvest disease symptoms is given.

The pathogenicity of the fungi involved in post-harvest diseases was thoroughly studied. Infections taking place later in the growing season are more critical and result in a higher post-harvest disease incidence at harvest than infections occurring early in the growing season. The natural latent infections by C. gloeosporioides and D. aromatica build up in the fruit during the growing season and decrease again during the dry harvest season.

Pre-harvest sprays with benomyl, captafol, bitertanol, Cu-hydroxide and Cu-oxychloride were found to control post-harvest diseases to some extent.

In post-harvest handling of avocados, the length of the ripening time has a marked influence on the incidence of post-harvest diseases and any post-harvest treatment that extends shelf-life increases the disease incidence. This increase could not be fully counteracted by the addition of fungicides to waxes.

Moisture on the fruit at harvest was found to aggravate post-harvest diseases whereas the sealing of the fruit pedicel with wax plus fungicides as well as the removal of the pedicel reduced losses due to stem-end rot.

By accurate identification of the organisms involved in pre- and post-harvest diseases of avocados, studying their epidemiology and selecting effective fungicides, it has been possible to give growers in this area a better understanding of what measures should be taken for the control of these diseases.

OPSOMMING

Cercospora-vlek veroorsaak deur Pseudocerospora purpurea (Cke) Deighton is vir die eerste keer in Suid-Afrika beskryf en daar is gevind dat dit die belangrikste voor-oes siekte van avokadovrugte te Westfalia Landgoed in die Noord-Oos Transvaal (lengtegraad 30°10' en breedtegraad 23°45') is. Verliese veroorsaak deur dié siekte is ondersoek ten opsigte van reën, kultivar, tyd van pluk, graad van wortelvrot simptome van die bome en die posisie van vrugte aan die bome.

'n Volledige beskrywing van die siekte-simptome word gegee. Statistiese modelle is verkry deur die analise van die resultate van die spoorvanger en die weersyfers. Die modelle kan gebruik word om hoë risiko tydperke vir infeksie aan te dui, en dit kan dus gebruik word vir die presiese bepaling van spuitdatums met swamdoders.

Besmettings wat vroeg in die groeiseisoen plaasvind gee die hoogste siektevoorkoms by pluktyd. 'n Drie-maande latente fase in die siekte se ontwikkeling is gevind.

Cercospora-vlek kan effektief beheer word deur benomil, kaptafol, Cu-oksichloried en Cu-hidroksied. 'n Statisties betekenisvolle verlaging in die effektiwiteit van benomil teen die siekte is gevind na 'n aantal jare se aanhoudende gebruik van dié swamdoder.

Op grond van simptome en betrokke patogene, word die volgende na-oes siektes van avokados te Westfalia Landgoed beskryf:

Stingelendbederf veroorsaak deur Thyronectria pseudotrichia (Schw.) Seeler, Colletotrichum gloeosporioides (Penz.) Sacc., Dothiorella aromatica (Sacc.) Petr. en Syd., Phomopsis perseae Zerova, Fusarium decemcellulare Brick en tot 'n mindere mate Pestalotiopsis versicolor (Speg.) Steyart, Lasiodiplodia theobromae (Pat.) Griff. en Maubl., Rhizopus stolonifer (Ehr. ex Fr.) Lind., Fusarium sambucinum Fuckel, Fusarium solani (Mart.) Sacc. en Drechslera setariae (Sawada) Subram. en Jain.

Antraknose veroorsaak deur Colletotrichum gloeosporioides (Penz.) Sacc.

Dothiorella/Colletotrichum-kompleks vrugtevrot veroorsaak deur Dothiorella aromatica (Sacc.) Petr. en Syd. en Colletotrichum gloeosporioides (Penz.) Sacc.

Verliese veroorsaak deur na-oes siektes is bestudeer met betrekking tot olie-inhoud van vrugte, graad van wortelvrot simptome van die bome en die posisie van vrugte aan die boom. 'n Deeglike beskrywing van die na-oes siektesimptome word gegee.

Die patogenisiteit van die betrokke swamme in na-oes siektes, is ondersoek. Besmettings wat laat in die groeiseisoen plaasvind is die belangrikste en dit gee aanleiding tot 'n hoër voorkoms van na-oes siektes by pluktyd as besmettings wat vroeg in die groeiseisoen plaasvind. Die natuurlike latente besmettings deur D. aromatica en C. gloeosporioides bou op in die vrug gedurende die groeiseisoen en toon 'n afname in die droë plukseisoen.

Voor-oes spuite met benomil, bitertanol, Cu-hidroksied en Cu-oksichloried gee 'n mate van beheer teen na-oes siektes.

Dit is gevind , gedurende die na-oes hantering van avokados, dat die lengte van die rypwordingsperiode 'n besliste effek op die voorkoms van na-oes siektes het. Enige na-oes behandeling wat raklewe verleng, verhoog ook na-oes siektes. Hierdie verhoging kon nie met die toediening van swamdoders volledig teengewerk word nie.

Vog op die vrug ten tye van pluk verhoog na-oes siektes terwyl die verseëling van die vrugtesteel met waks plus swamdoders, sowel as die verwydering van die vrugtesteel, verliese wat veroorsaak word deur stingelendbederf, verminder.

Deur noukeurige identifikasie van die organismes betrokke by voor- en na-oes siektes van avokados, bestudering van epidemiologie en selektering van doeltreffende swamdoders, was dit dus moontlik om aan kwekers in hierdie area 'n beter begrip te gee van die maatreëls wat getref moet word vir die bestryding van hierdie siektes.

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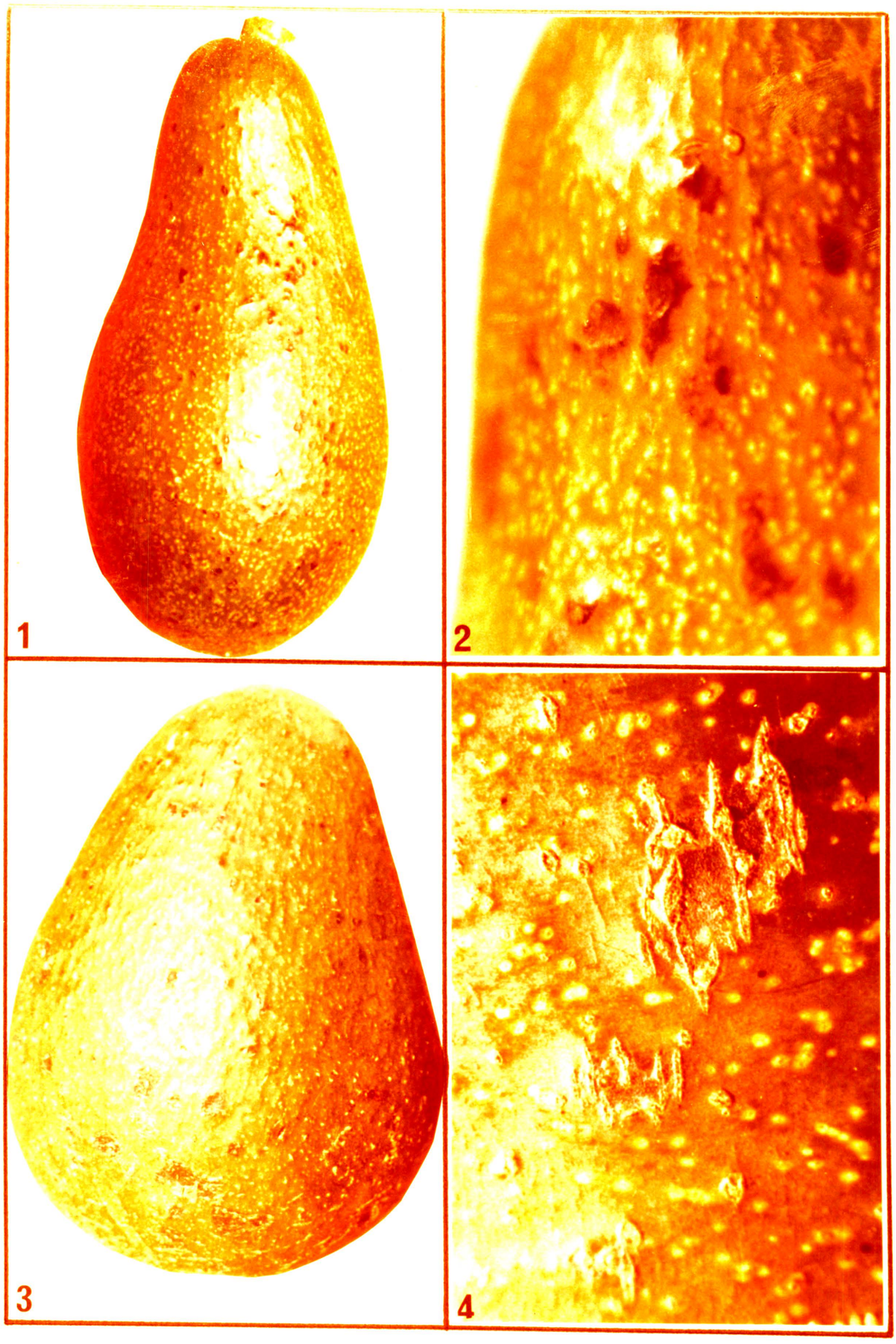
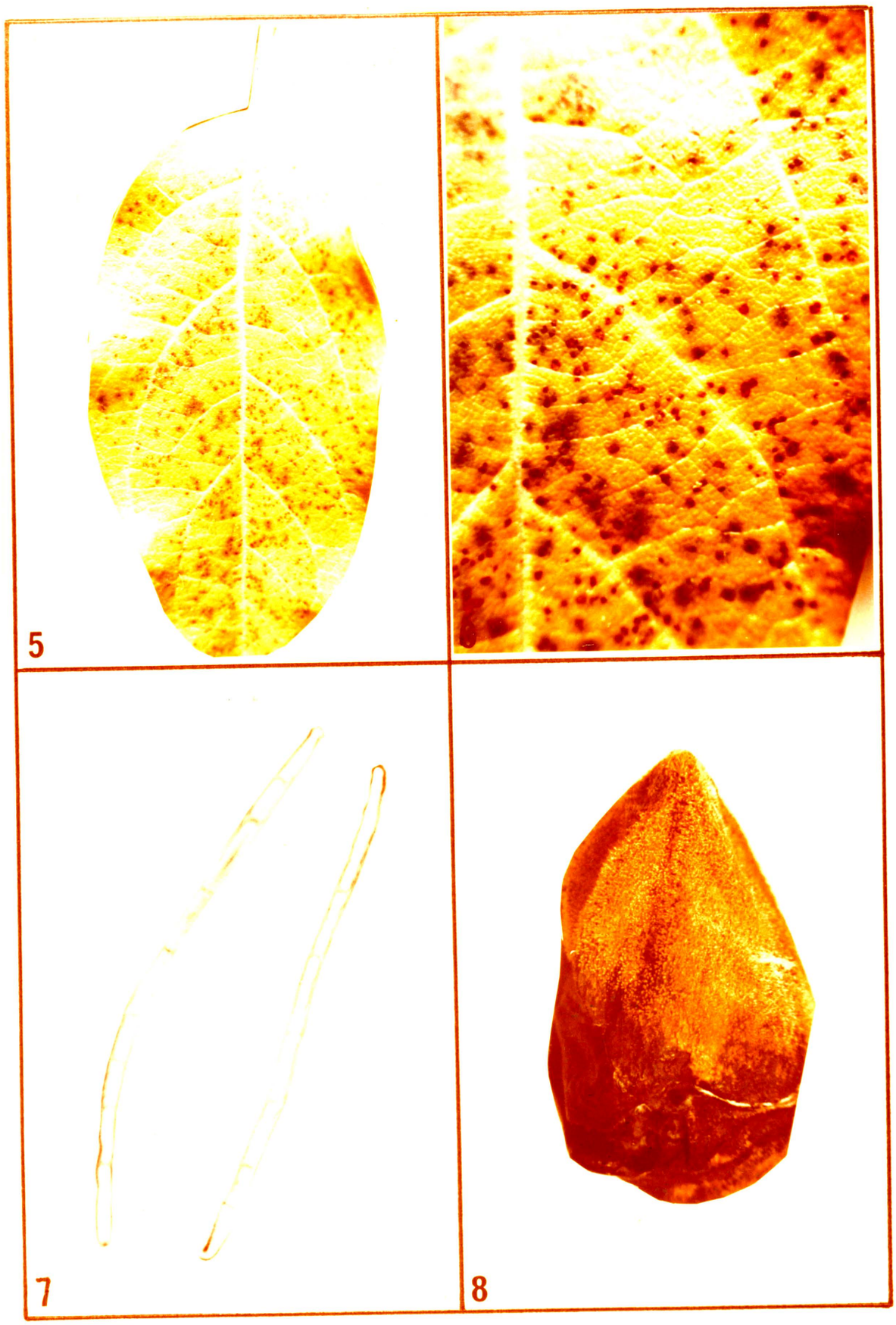


Photos : 1 : Cercospora spot on Fuerte - raised type

2 : Cercospora spot on Fuerte - raised type (close-up)

3 : Cercospora spot on Fuerte - sunken type

4 : Cercospora spot on Fuerte - sunken type (close-up)

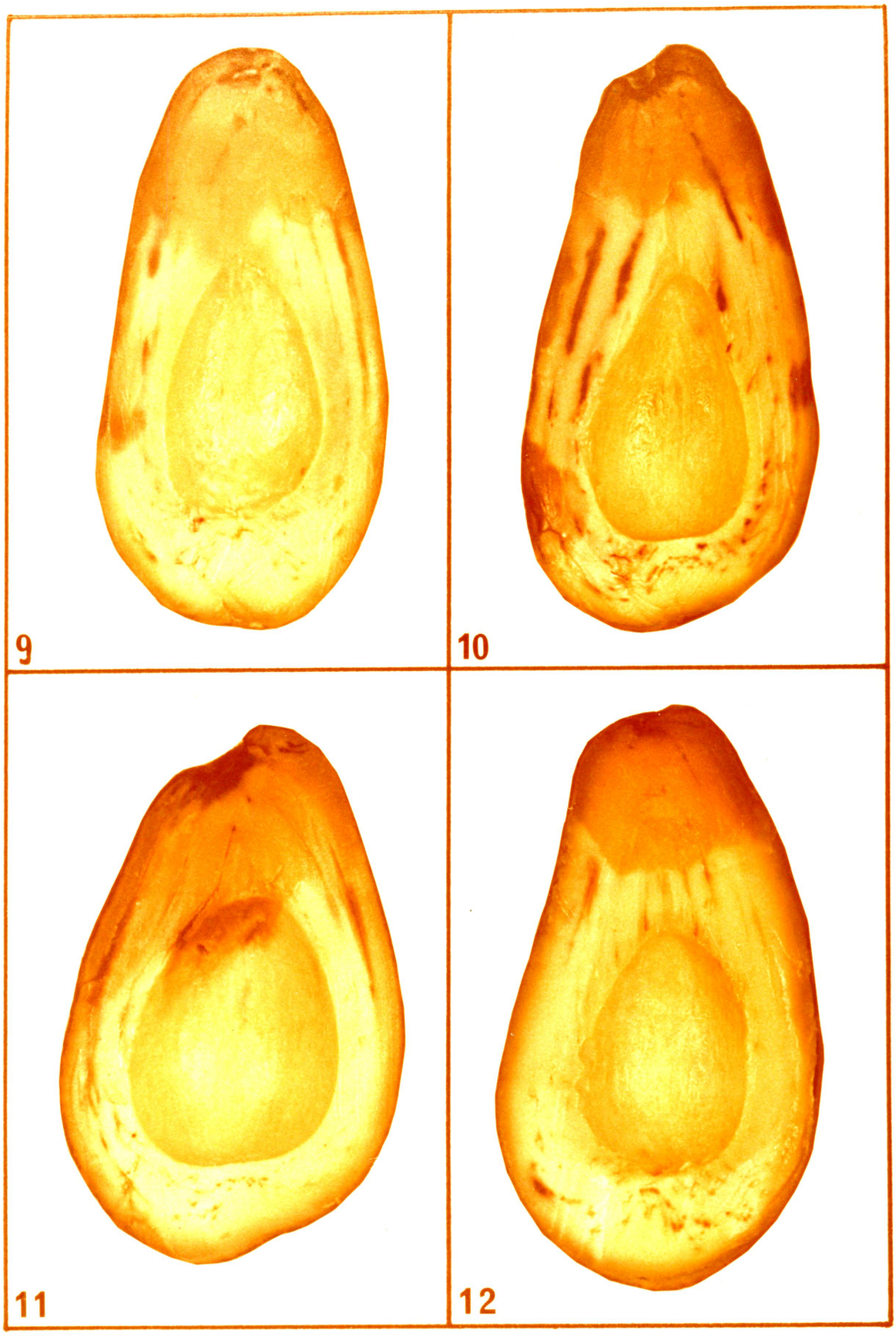


Photos : 5 : Cercospora spot on Fuerte leaf

6 : Cercospora spot on Fuerte leaf (close-up)

7 : Conidia of Pseudocercospora purpurea (1600 X)

8 : Fuerte fruit mummified by Thyronectria pseudotrichia

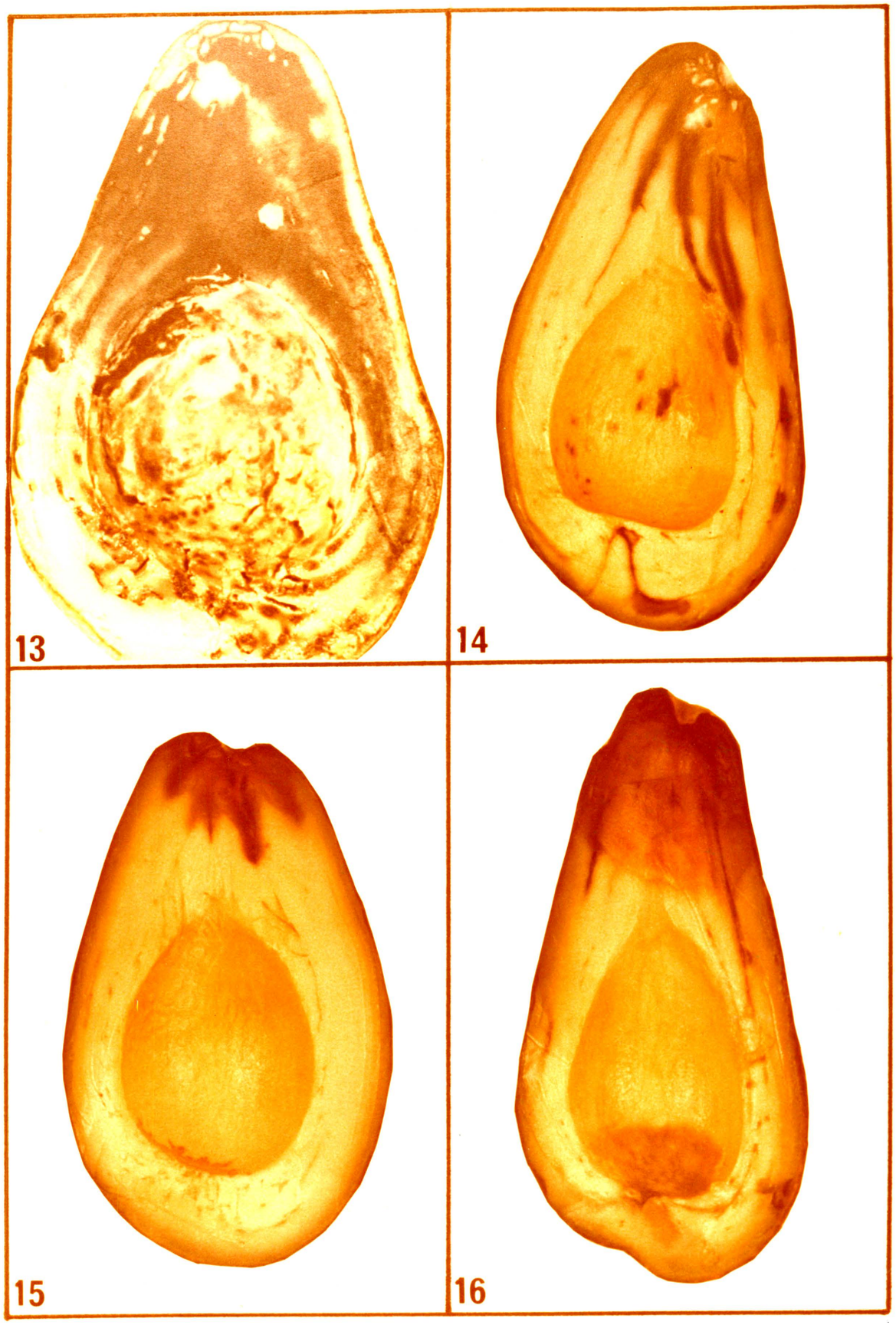


Photos : 9 : Stem-end rot by Thyronectria pseudotrichia

10 : Stem-end rot by Colletotrichum gloeosporioides

11 : Stem-end rot by Dothiorella aromatica

12 : Stem-end rot by Phomopsis perseae

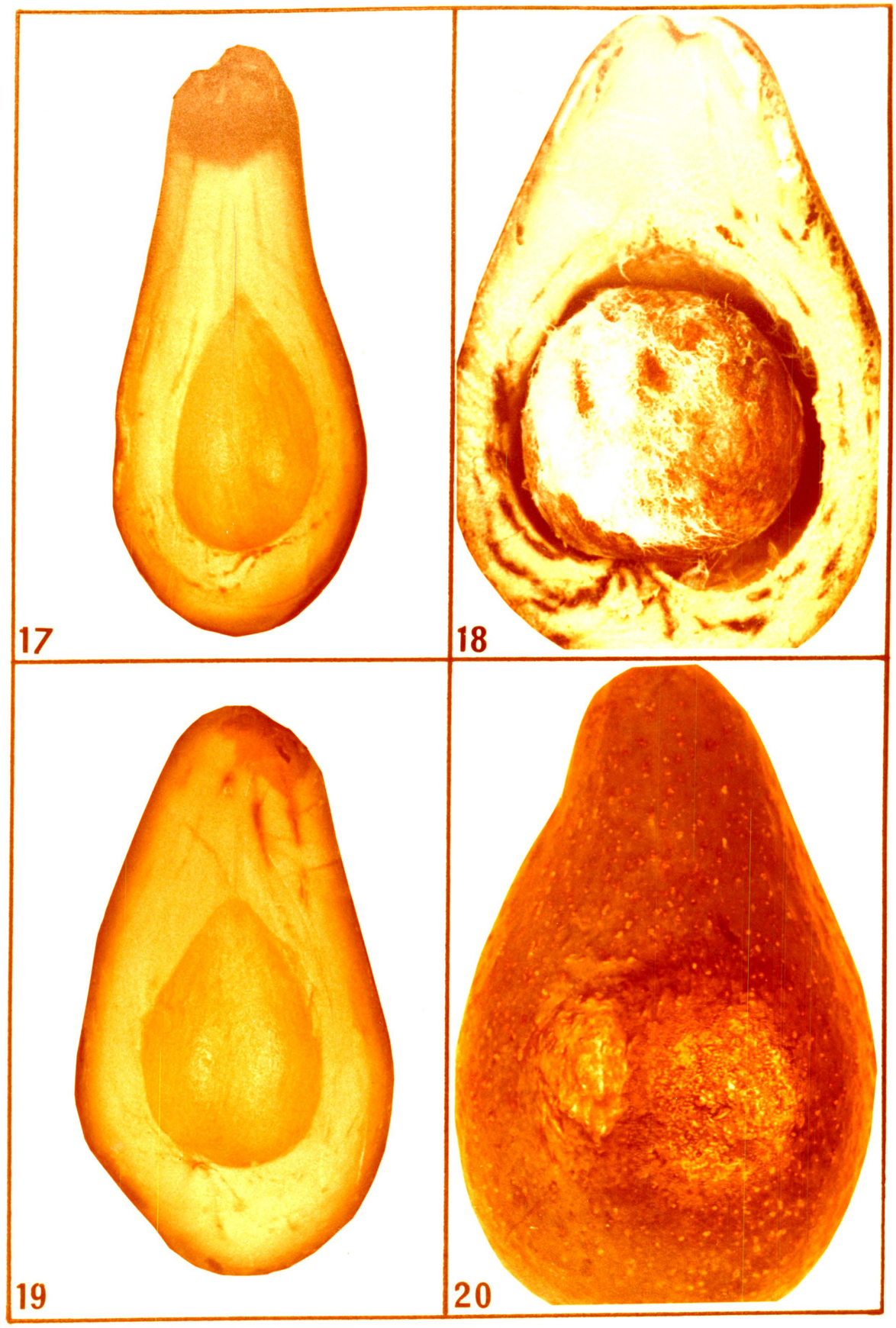


Photos : 13 : Stem-end rot by Lasiodiplodia theobromae

14 : Stem-end rot by Pestalotiopsis versicolor

15 : Stem-end rot by Fusarium decemcellulare

16 : Stem-end rot by Fusarium sambucinum

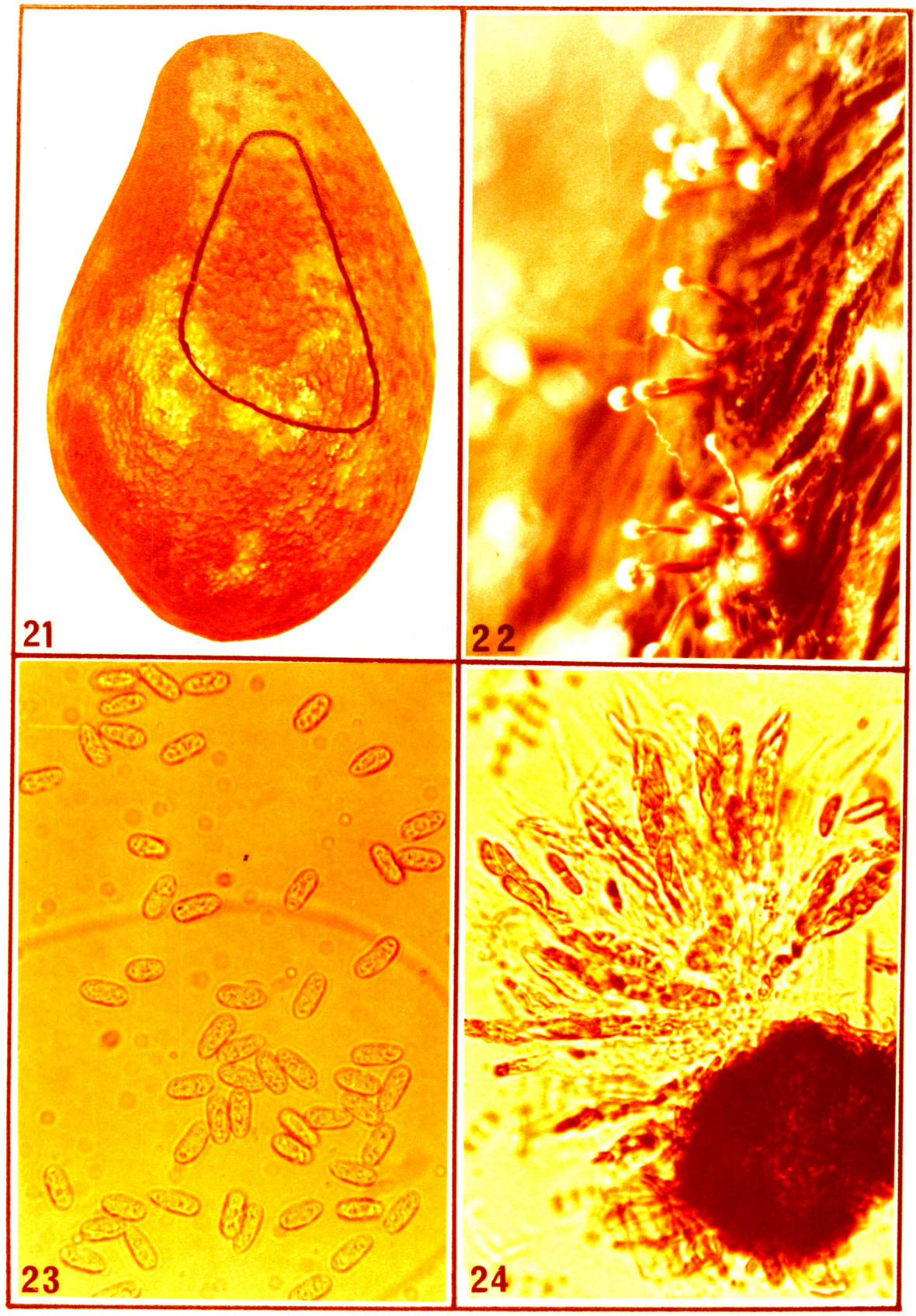


Photos : 17 : Stem-end rot by Fusarium solani

18 : Stem-end rot by Rhizopus stolonifer

19 : Stem-end rot by Drechslera setariae

20 : Anthracnose by Colletotrichum gloeosporioides

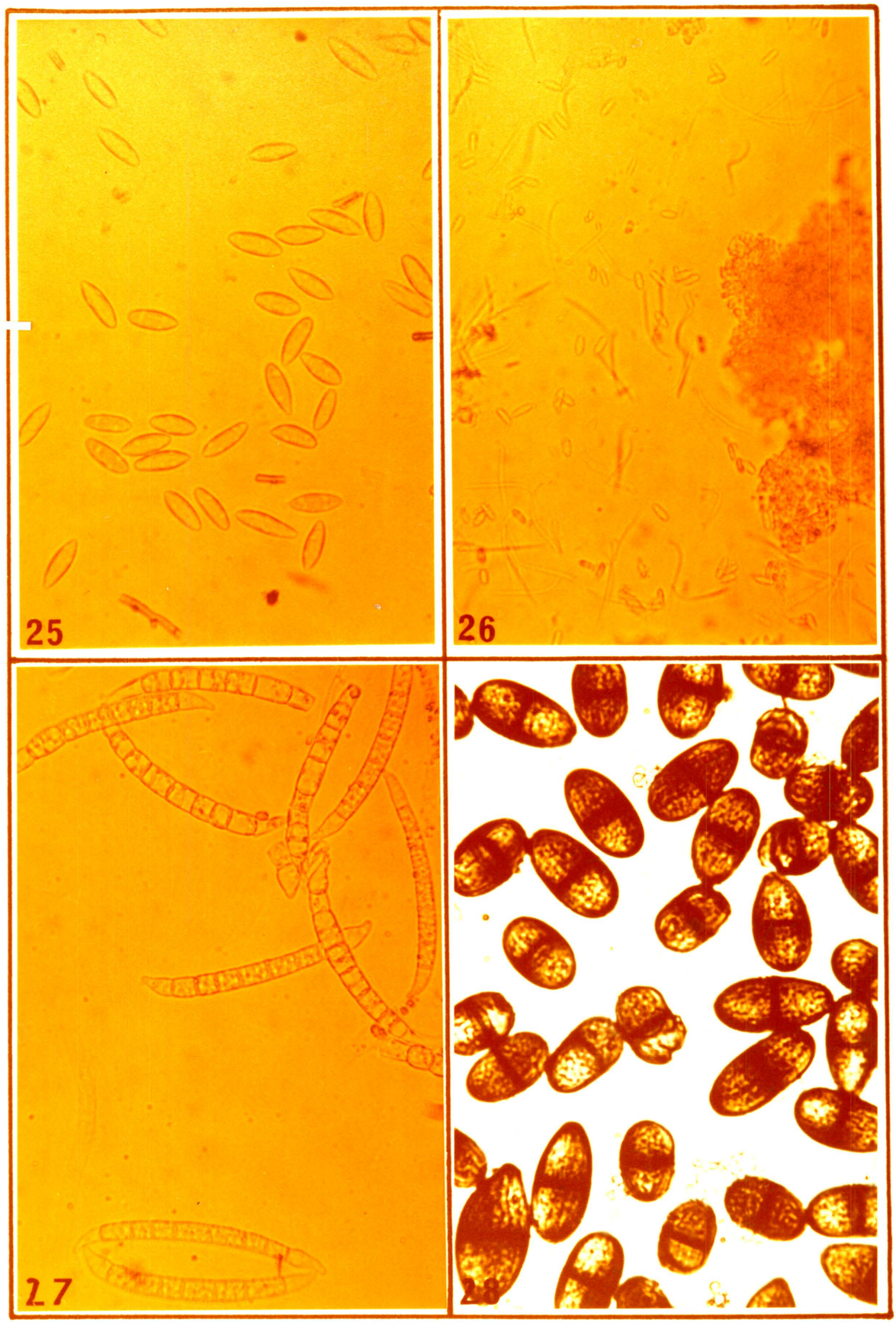


Photos : 21 : Dothiorella/Colletotrichum complex fruit rot

22 : Stilbella asexual stage of Thyronectria pseudotrichia (40 X)

23 : Conidia of Colletotrichum gloeosporioides (400 X)

24 : Perithecium and asci of Glomerella cingulata (400 X)



Photos : 25 : Conidia of Dothiorella aromatica (400 X)

26 : Conidia of Phomopsis perseae (100 X)

27 : Macroconidia of Fusarium decemcellulare (100 X)

28 : Conidia of Lasiodiplodia theobromae (400 X)