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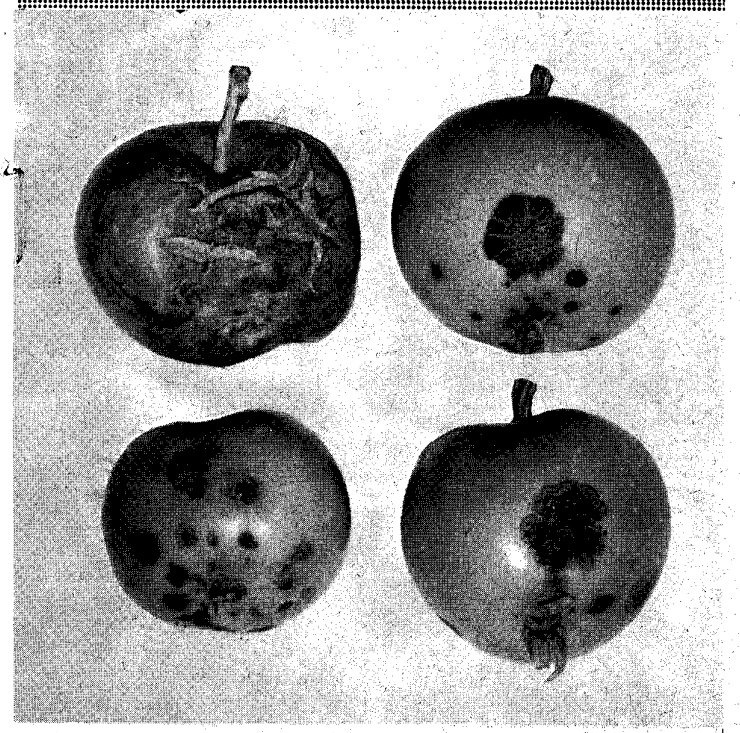

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CONTROL OF ANTHRACNOSE OF STORED TOMATOES WITH VENTURICIDIN[1]

C. L. Lockhart[2]

Abstract

A post-harvest dip of Venturicidin 12 to 24 hours after inoculation with Colletotrichum coccodes gave highly significant control of anthracnose. Venturicidin was more effective at 200 ppm than at lower concentrations, but gave inconsistent control of field infections of anthracnose and other rots of stored tomatoes.

Introduction

Anthracnose caused by Colletotrichum coccodes (Wallr.) Hughes was found by Lockhart and Harrison (2) to be a major disease of stored tomatoes in Nova Scotia. Latent infections take place in the field and the disease develops in storage as the tomato ripens. Lockhart and Eaves (1) showed that the post-harvest treatment of tomatoes with deposits of captan smoke residue gave some control of anthracnose. Recently Rhodes et al (3) produced a strong antifungal antibiotic; Venturicidin, which has no oral toxicity. Because toxic residues are a limiting factor in selecting fungicides or antibiotics for post-harvest treatment of tomatoes, Venturicidin appeared suitable to use in further investigations on the control of anthracnose.

The object of this investigation was twofold; (1) to determine the effect of various concentrations of Venturicidin dips on anthracnose, and (2) to assess the effect of different levels of inoculum and the inoculation-treatment intervals on the development of disease and control obtained with the antibiotic.

Materials and Methods

Immediately after harvesting, mature-green tomatoes, of the variety Harrow, were inoculated by dipping the tomatoes in a spore suspension of C. coccodes (500,000/ml) and incubated for 24 hours at 72° F unless stated otherwise. Ten grams of Venturicidin (Glaxo Research Ltd., Sefton Park, Stoke Poges, Buckinghamshire, England) were dissolved in 65.7 ml of ethylcellusolve, to which was added 27.6 gm of Sorbester PQ12 (mixed monolaurate and polyoxyethylene condensate from Howards of Ilford Ltd., Ilford, Sussex). Preliminary experiments showed that these levels of solvent and surfactant had no fungicidal effect. The 10 per cent formulation of Venturicidin was added to water to give desired concentrations. Each replicate consisted of 25 tomatoes. All treatments were replicated 4 times and all

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1 Contribution No. 1131 from the Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

2 Plant Pathologist.

experiments were done in duplicate at different times, hereafter indicated as I and II. The tomatoes were dipped in Venturicidin for 1 to 2 minutes. Following the dip treatments all tomatoes were stored at 52.5°F for 4 or 5 weeks in single layers on trays (18" x 56") lined with brown paper. The tomatoes were examined at weekly intervals and all fruits showing anthracnose or other rots were removed from the trays. Total rots, indicated in brackets in the tables, include anthracnose and those caused by other microorganisms.

In the level of inoculum experiments tomatoes were inoculated in 0, IX, 2X, 4X and 8X levels of C. coccodes (X=250,000/ml). At the end of the 24-hour incubation period 4 replicates at each inoculum level were dipped in Venturicidin and 4 replicates were left untreated. In another experiment tomatoes were inoculated and incubated for 0, 3, 6, 12, 21 1/2, 24, 30 and 48 hours. At the end of each incubation period 4 replicates were dipped in Venturicidin and four were left untreated.

Results and Discussion

Venturicidin was more effective in the control of anthracnose and total rots of tomatoes at 200 ppm than at lower concentrations (Table 1). Anthracnose infections increased with increasing levels of C. coccodes inoculum and, at the same time, a corresponding decrease in control of anthracnose was obtained with Venturicidin (Table 2). Rots were also caused by Alternaria tenuis Nees, Botrytis cinerea Pers. ex Fr., Sclerotinia sp., Phoma sp. Phytophthora infestans (Mont.) de Bary, spotted wilt and bacteria. Except for Alternaria and bacteria the other microorganisms were of minor importance.

The percentage of Alternaria rots on the uninoculated tomatoes for the level of inoculum in the first and second experiments were:

Venturicidin treatment	Dipped		Untreated	
	I	II	I	II
Per cent Alternaria rots	21	3	5	7

The high incidence of Alternaria rots of the dip-treated tomatoes in the first experiment accounts for 21 of the 25 per cent total rots (Table 2). The increase in Alternaria rots is attributed to minute cracks which appear to be induced during dip treatments and subsequently become centres of infection in storage. An increase in the number of rots caused by bacteria was also noted in some tests with dipped tomatoes. Bacterial rots have often been troublesome in tomatoes subjected to various fungicidal dips. Perhaps this cracking of tomatoes might be overcome by adding a non-toxic salt to the dip solution in order to increase the osmotic pressure and thus enhance the value of the fungicide or antibiotic.

The inoculation-treatment interval was found to be an important factor in the degree of control of anthracnose of tomatoes obtained with Venturididin

Table 1. Effect of Venturicidin in two experiments on the control of tomato rots at the end of 4 weeks in storage at 52.5° F.

Venturicidin in ppm	Per cent anthracnose and total rots			
	Inoculated		Uninoculated	
	I	II	I	II
0	50 (71)*	47 (55)	12 (37)	11 (29)
25	28 (40)	46 (51)	13 (30)	9 (13)
50	30 (37)	40 (47)	13 (26)	8 (14)
100	32 (42)	31 (38)	8 (19)	3 (9)
200	27 (45)	29 (33)	4 (14)	1 (7)
L. S. D. .05 Dips			7.9 (8.1)	3.8 (5.8)
L. S. D. .01			10.9 (11.4)	11.7 (8.1)
L. S. D. .05 Inoculations			2.8 (5.0)	4.6 (3.7)
L. S. D. .01			5.9 (7.0)	6.3 (5.1)

* Total rots in brackets

Table 2. Control of rots on inoculated tomatoes treated with 200 p. p. m. Venturicidin and stored for 5 weeks at 52.5° F.

Level of inoculum (*C. coccodes*)	Per cent anthracnose and total rots			
	Dipped		Untreated	
	I	II	I	II
0	3 (25)*	7 (17)	2 (14)	6 (14)
IX	27 (52)	21 (55)	32 (55)	46 (71)
2X	26 (50)	26 (49)	38 (63)	48 (59)
4X	40 (51)	39 (61)	45 (61)	59 (77)
8X	43 (50)	38 (60)	54 (66)	48 (66)
L. S. D. .05 Dips		6.8 (9.9)		6.5 (6.7)
L. S. D. .01		9.4 (12.6)		8.9 (9.3)
L. S. D. .05 Inoculum levels		5.5 (11.9)		7.9 (5.3)
L. S. D. .01		7.7 (16.7)		11.1 (6.2)

* Total rots in brackets

(Table 3). Anthracnose infections due to artificial inoculations occurred after 12 or 3 hours incubation for the first and second experiments respectively and highly significant control of anthracnose by Venturicidin was attained 12 to 24 hours after inoculation. It is of interest to note that field infections of anthracnose were not more than 7 per cent as indicated by tomatoes receiving no inoculum (Table 2). A longer storage period would result in a higher incidence of anthracnose from field infections. The control obtained with Venturicidin may depend upon when infection occurs, since in dipping experiments the Venturicidin had to be applied within 24 hours following inoculation to be effective.

Table 3. Control of rots on tomatoes treated with 200 p. p. m. Venturicidin at various intervals following inoculation and stored for 4 weeks at 52. 5° F.

Inoculation-treatment intervals in hours	Per cent anthracnose and total rots			
	Dipped		Untreated	
	I	II	I	II
0	5 (26)*	5 (9)	6 (25)	11 (28)
3	8 (22)	9 (30)	9 (24)	22 (38)
6	7 (28)	22 (25)	8 (25)	26 (40)
12	28 (43)	32 (48)	60 (74)	44 (57)
21 1/2	48 (62)	29 (42)	79 (87)	42 (52)
24	64 (75)	37 (47)	69 (80)	48 (62)
30	65 (80)	77 (86)	67 (78)	70 (79)
48	70 (86)	78 (90)	74 (93)	80 (94)
L. S. D. .05 Dips		6. 8 (5. 1)	6. 5 (7. 8)	
L. S. D. .01		9. 2 (6. 8)	8. 9 (10. 6)	
L. S. D. .05 Intervals		6. 3 (8. 9)	7. 2 (6. 1)	
L. S. D. .01		8. 8 (12. 2)	9. 9 (8. 3)	

* Total rots in brackets

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CANADA AGRICULTURE RESEARCH STATION,
KENTVILLE, NOVA SCOTIA.

THE INFLUENCE OF SEED TREATMENTS ON THE DEVELOPMENT OF SEEDLING BLIGHT OF OATS[1]

R. V. Clark[2]

Abstract

Several seed treatment materials were tested against seedling blight of oats caused by Pyrenophora avenae. Those containing organic mercury were effective in controlling visible symptoms of the disease and increasing emergence. Chemical treatment resulted in a variable increase in emergence and this increase did not appear to be associated with the control of seedling blight but rather with protection from other soil organisms. Seedling blight development appeared to be influenced considerably by environmental conditions, being particularly favored by cool soil temperatures.

Introduction

Seedling blight of oats caused by the fungus Pyrenophora avenae Ito & Kurib. (Imperfect state: Drechslera avenacea (Curtis ex Cke) Shoemaker = Helminthosporium avenae Eidam), has been common in Canada. Survey reports (6) indicate that the disease was present in some part of Canada every year for the past 30 years and was occasionally severe in both eastern and western Canada but usually not in the same season.

This disease is considered to be of minor importance in most oat growing areas. However, it has caused considerable damage particularly in the winter crop area of the southern United States (1, 2, 3, 4, 5). The primary or seedling stage of the disease has been fairly common in most parts of eastern Canada in the past few years. In 1960, the secondary, or leaf blotch, stage was quite prevalent and undoubtedly caused considerable damage to the crop, although the earlier, or primary, stage was not unduly heavy. Because of the increased prevalence of this disease, it was felt that more should be known of its development and control under conditions in eastern Canada. There are no commonly grown varieties that are resistant (3, 4). A limited number of seed treatments have been effective in other oat growing areas (3, 4) but little is known about these and other chemicals in this area. This report summarizes the results obtained when oats treated with several chemical seed treatments were grown at Ottawa at different dates of seeding throughout the growing season.

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1. Contribution No. 99 from the Genetics and Plant Breeding Research Institute, Research Branch, Canada Department of Agriculture, Ottawa, Ontario.

2. Plant Pathologist.

Materials and Methods

In 1959 an Ottawa strain of oats (5055-13) was found to have approximately a 25 per cent infection of seedling blight present in the field shortly after emergence. When the seed from this crop was plated on potato-dextrose-agar, an average infestation of P. avenae of 20 per cent was noted. This was a good source of naturally-infected seed and, in the spring of 1960, samples from it were treated with various seed dressings. Sixteen chemicals were included in the tests with each treatment consisting of a one-pound sample of seed. The chemicals were applied at concentrations recommended by the manufacturers (Table I). A small home-made treater with a tumbling action was used to apply the seed dressings uniformly. It worked equally well with dusts or liquids. Each seed sample was treated in a 1-litre stoppered Erlenmeyer flask and then kept in the same flask at room temperature until used.

In 1960 four replicates of 4-row plots with each row containing 100 seeds were planted for each treatment. A plot of untreated seed was included in each replicate. All treatments were planted in two different types of soil and the seeding was done on May 28. The same treated seed was used in additional tests in 1961 and in this case each row of 100 seeds was considered as a replicate with a total of four being used. Nine different dates of seeding were planted with all 16 seed treatments with a control included each time. The control consisted of 4 plots of 4 rows each of untreated seed placed at predetermined positions among the various treatments. The seed treatments were then randomized within each date of seeding. The first date of seeding was planted on April 24 and the next five at 10-day intervals and the last three at three-week intervals.

Emergence counts and disease notes were recorded approximately one month after planting. In most cases the disease notes were obtained by pulling the plants and examining the seedlings individually. This was necessary to insure proper identification of the disease and the extent to which it was present.

Results and Discussion

In the 1960 tests less than one per cent of the seedlings in the control plots showed disease symptoms typical of seedling blight. It was therefore impossible to evaluate the various seed treatments for disease control nor was there any evidence of a different amount of disease in the two soil types used, a sandy loam and a clay loam. The weather in 1960, when the planting was done and until the notes were taken, was quite warm. It was thought that this may have been the reason for the very low percentage of seedling blight present in the control plots.

Table 1. The effects of various seed treatments on the emergence of oats and the indicence of seedling blight.

Seed Treatment	Active Ingredient	Concentration oz/bu	Emergence[1] Percent	Seedling blight[2] Percent
Puraseed	6.35% phenyl amino cadmium lactate plus 6.35% phenyl mercury formamide	0.5	88.4*	0
Liquisan	2.25% methyl mercury 8-hydroxyquinolinate	0.75	88.1	0
Ceresan M	7.7% ethyl mercury-p-toluene sulfonanilide	0.5	87.4	0
Agrosol	1.8% methyl mercury nitrile	0.75	87.2	0
Gallotox	6.5% phenyl mercury acetate	0.75	87.2	0
Canuck organic mercury	7.06% phenyl mercury acetate and 1.06% ethyl mercury chloride	0.5	87.0	0
Panogen 15	2.2% methyl mercury dicyandiamide	0.75	86.7	0
Orthocide Dieldrin 60-15	60% N-(trichloromethylthio)-4-cyclohexene-1, 2-dicarboximide and 12.75% hexachloro-epoxy-octahydro-endo-exo-dimethanonaphthalene	1.25	86.3	6.9
Ceresan 100	3.10% ethyl mercury, 2,3 dihydroxy propyl mercaptide and 0.67% ethyl mercury acetate	0.75	86.1	0
Dexon	70% p-dimethylaminobenzenediazo sodium sulphonate	0.5	86.1	4.9
Agrox C.	7.0% phenyl mercury acetate and 1.06% ethyl mercury chloride	0.5	84.6	0
Half-ounce Leytosan	8.1% phenyl mercury urea	0.5	84.2	0
Puradrin	40% hexachloro-hexahydro-endo-exo-dimethanonaphthalene and 3% phenyl mercury formamide plus 3.45% phenyl amino cadmium lactate	1.25	83.7	0
Meragamma C.	40% gamma isomer of benzene hexachloride, 2.86% phenyl mercury acetate and 0.4% ethyl mercury chloride	1.25	82.8	0
Pandrinox	0.75% methyl mercury dicyandiamide and 34% Tech. heptachlor	1.25	82.1	0
Bunt-no-more	40% hexachlorobenzene	0.5	79.4	5.2
Control			77.8	7.4

1. Average of 9 dates of seeding
2. Average of 7 dates of seeding

* L.S.D. 3.92 - 1% level

Table 2. The influence of seeding date and seed treatments on the emergence of oats and development of seedling blight.

Date of seeding	Percent emergence		Percent seedling blight	
	Treatments[1]	Control	Treatments[2]	Control
Apr. 24	87.0	76.2	7.4	10.5
May 4	87.1	78.9	6.5	9.5
May 15	89.4	80.9	4.2	9.2
May 25	86.0	73.9	9.9	10.9
June 6	78.1	71.1	3.4	6.7
June 15	88.1	82.3	2.6	4.4
July 6	79.6	73.4	0.8	0.6
July 27	85.4	79.3	0	0
Aug. 17	88.5	84.4	0	0

1. Average of 16 treatments
2. Average of the 3 treatments showing symptoms

The same treated seed was planted in the 1961 tests. The incidence of seedling blight was higher in 1961 (Table 1) with an average of 7.4 per cent in the control plots and it was as high as 10.9 per cent in one date of seeding, May 25. (Table 2). Good control of visible symptoms was obtained with the majority of the seed treatments. Only three compounds did not control seedling blight and these did not contain mercury (Table 1). Previous work (3, 4) has shown that Ceresan M controls seedling blight of oats effectively in the southern United States. From the results of the present tests it would appear that any seed dressing containing organic mercury is quite effective against this disease, including Ceresan M and liquids as well as dusts.

Emergence counts of the oats were also recorded in 1961. All treatments were superior to the control in emergence and the majority were significantly better in this regard. Ten of the chemicals were equally effective in promoting seedling emergence. Included were Orthocide Dieldrin 60-15 and Dexon, two of the chemicals that did not control seedling blight. The results suggest that all the chemicals tested were providing protection against soil organisms; that some were superior to others; and that this improvement in emergence was not because of the control of seedling blight. Even with the best treatment approximately 10 per cent of plants failed to emerge. This may have been due to seedling blight since the seed showed a 20 per cent infestation when plated on agar and approximately 10 per cent when planted in the field. However, the mercury compounds did eliminate all above-ground lesions (Table 1) and this would help to greatly reduce the inoculum potential for the development of the secondary phase of the disease. Four combination fungicide-insecticides were included

in the tests and the three that contained mercury gave relatively poor emergence totals. The same treated seed was used in both years as these tests were carried out to study the seasonal development of seedling blight as well as to obtain information on its control. Because of the extended storage of the seed and the volatile action of the mercury compounds which may have biased the performance of these chemicals, these data cannot be used as a recommendation to oat growers. However, they do show that oat seed can be treated and then stored for 15 months without lowering the subsequent emergence.

In 1961 seedling blight developed over an extended period (Table 2). The maximum amount developed from the seed that was planted on May 25 although the three earlier plantings produced approximately the same amount. After May 25 the amount of seedling blight dropped rather quickly and very little was found on the plants that had been seeded on July 6 or those planted on July 27 and August 17. The lowest average emergence was obtained with the seed planted on June 6 but emergence was also quite low from seed planted on July 6 and May 25. It would appear that the presence of seedling blight did not influence the emergence counts to any extent since a high disease rate did not necessarily correspond with low emergence. The same pattern of seedling blight development and emergence was evident with both the treated seed and the controls.

Weather data for the spring and summers of 1960 and 1961 were obtained from the Agrometeorology Section, Plant Research Institute, Ottawa. In late May and early June of 1960 the weather was quite warm. The average daily soil temperature just below the surface of the soil reached 70° F in late May and stayed more or less high during the rest of the summer. In 1961, a soil temperature of 70° was not reached until early in July although it was fairly high during the latter part of June. It would appear from the data that fairly low soil temperatures are necessary for the development of seedling blight. The greatest amount of seedling blight developed on the plants that were seeded on May 25 (Table 2) and the weather records showed that the soil temperatures following this planting were quite low due to an unusually late snowfall.

Rainfall did not appear to be as closely associated with seedling blight development as was temperature. Conditions were dry in late April and in the early part of May 1960 but there was a heavy rainfall (1.5 inches) at the end of May, just after the seed was planted, with several others during June. Rainfall was about the same during the spring and summer of 1961 except that no very heavy rain occurred at any one time. Total rainfall in May and June of 1960 amounted to 6.50 inches with precipitation occurring on 24 days.

Ivanoff and Blount (4) have reported that in the southern United States the primary infection of seedling blight is favored by temperatures of from 75 to 82° F and a very high relative humidity with some excess moisture. The results of the present studies indicate that low soil temperatures favor seedling blight development and that temperatures exceeding 70° F greatly limit disease development. High levels of moisture would not appear to be necessary for disease development in the field.

Acknowledgements

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GENETICS AND PLANT BREEDING RESEARCH INSTITUTE,
OTTAWA, ONTARIO.

THE CONTROL OF FIELD AND STORAGE DISEASES OF TOMATOES[1]

K.A. Harrison and C.L. Lockhart[2]

Abstract

Control of a severe outbreak of late blight of tomatoes was obtained with 6 applications, at 10-day intervals, of either maneb or zineb at 2 lb per 100 gal (Imp.). Twenty-eight and 33 per cent gray-mold rot, respectively, developed on the fruit sprayed with maneb and zineb. Satisfactory control of both diseases was obtained when 1 or 2 lb of either maneb or zineb was combined with either 1 or 2 lb of thiram or 1 3/4 lb of Dyrene. The best control of storage rots followed when 2 lb of thiram was added to either maneb at 2 lb or tank mixed zineb, 1 qt nabam + 3/4 lb $ZnSO_4$.

Introduction

The commercial production of field tomatoes in Nova Scotia is largely confined to the Annapolis Valley where conditions are moderately favorable for ripening the fruit. In a previous report Harrison (1) showed that several carbamate fungicides used to control late blight, Phytophthora infestans (Mont.) de Bary, resulted in a greatly increased amount of gray-mold rot, Botrytis cinerea Pers. ex Fr., on tomatoes and that some control of this disease could be obtained by adding 2 lb of thiram, or 1 3/4 lb of Dyrene to either maneb or zineb. Lockhart and Harrison (2) found that tank-mix zineb plus thiram was more effective in controlling storage rots than factory-mix zineb plus thiram.

This paper presents the results obtained in 1962 from a comparison of a number of mixtures of fungicides used to control field and storage diseases of tomatoes.

Methods

Tomato plants of the variety Stokesdale were set in 4 blocks and the treatments randomized. Each of the 19 plots in a block contained 6 plants with the end plants serving as guards. Maneb, factory-mix zineb and tank-mix zineb were tested at the rates recommended by the manufacturers and compared with a number of mixtures of the same fungicides at full and half strengths with either thiram at 1 or 2 lb or Dyrene at 1 or 1 3/4 lb added. Blitox, a fixed copper, and ziram and Bordeaux 10-7-100 in a split program were also included because in past tests these fungicides have had very

1. Contribution No. 1135 from the Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.
2. Plant Pathologists.

little effect on the incidence of gray-mold rot and gave reasonable control of late blight. Delan* was tested for the first time. Six fungicide applications were made at approximately 10-day intervals between July 27 and September 22. The final harvest was started October 3 and, due to wet weather, was not completed until October 12. Two pickings of 25 mature-green fruit were made from each plot, the first on September 12 and the second on October 1, for storage trials. These fruits were stored at 52.5° F for 5 weeks in a single layer on trays (18" x 36") lined with brown paper. They were examined at weekly intervals and all fruits showing rot were removed from the trays. Isolation and identification of unknown rots were made on potato-dextrose-agar. A thermograph recorded the field temperatures during September and October.

Results and Discussion

Field Tests

The tomato plants grew vigorously but because of the cool wet season the fruit matured very slowly and was not ready for picking until September 27. The season was favorable for fungicide tests as both late and gray-mold rot were present in epidemic amounts.

The results of the field tests, given in Table 1, are the average of the 4 replicates of each treatment and are calculated from the total of all fruit picked during the season. Excellent control of late blight was obtained with all materials and combinations. Delan was the least effective. Stem rot, Sclerotinia sclerotiorum (Lib.) de Bary, was present in all plots. There was no evidence that any fungicide affected the amount of this disease. However, it is difficult to obtain accurate counts of other diseases of the fruit when late blight is as severe as it was in these plots. The percentages of gray-mold rot present in 1962 were the highest recorded since yearly tests were started in 1948. In 1962 the percentage of gray-mold rot was 33.0, 32.7 and 28.8 for factory-mix zineb, tank-mix zineb and maneb, respectively. The lowest percentage of gray-mold rot was obtained with a mixture of maneb and thiram.

It is interesting to note that the average per cent of the means of gray-mold rot from all plots treated with thiram at 2 lb was 5.9, at 1 lb, 10.1, and where Dyrene was used at 1 3/4 lb, 8.6 and at 1 lb, 12.0. The average per cent of the means of the 5 plots where maneb was used was 11.1, factory-mix zineb, 13.1 and tank-mix 15.5.

Storage Tests

Less rot developed in storage on tomatoes picked on September 12 than on those picked on October 1 (Tables 2 and 3). This difference was due to the increased numbers of Alternaria, gray-mold, late blight and other rots in the second picking (Table 3). The largest increase was due to Alternaria rots, indicating that some chilling injury had occurred when the minimum temperature dropped below 40° F on 3 different days prior to the second picking.

* 75% 2,3 dinitrilo-1,4-dithiaanthraquinone.

Table 1. Per cent field rots on tomatoes from spray plots.

Fungicide per 100 gallons	Per cent rots		
	B. cinerea	P. infestans	S. sclerotiorum
Maneb 2 lb.	28.8 abc[1]	1.1	1.6
Maneb 2 lb. + thiram 2 lb.	4.2 h	0.0	0.5
Maneb 2 lb. + Dyrene 2 lb.	5.9 gh	0.1	0.5
Maneb 1 lb. + thiram 1 lb.	9.4 fgh	0.2	1.5
Maneb 2 lb. + Dyrene[2] 1 3/4 lb.	7.1 fgh	0.0	0.4
Zineb factory-mix 2 lb.	33.0 abc	1.2	1.5
Zineb factory-mix 2 lb. + thiram 2 lb.	6.0 gh	0.9	0.9
Zineb factory-mix 2 lb. + Dyrene 13/4 lb.	8.1 fgh	0.1	0.5
Zineb factory-mix 1 lb. + thiram 1 lb.	6.9 fgh	1.0	1.5
Zineb factory-mix 1 lb. + Dyrene 1 lb.	11.4 fgh	0.4	1.4
Zineb tank-mix 1 qt. nabam + 3/4 lb. $ZnSO_4$	32.7 abc	0.7	1.3
Zineb full strength + thiram 2 lb.	7.6 fgh	0.0	0.8
Zineb half strength + Dyrene 1 3/4 lb.	10.5 fgh	0.1	0.6
Zineb half strength + thiram 1 lb.	14.0 defg	0.1	0.3
Zineb half strength + Dyrene 1 lb.	12.6 efgh	0.6	2.5
Blitox[2] 3 lb.	24.3 abcd	0.3	0.8
Ziram 2 lb. Bordeaux, 10-7, split program	16.3 cdef	0.1	2.6
Delan[4] 1 lb.	23.5 abcde	4.1	1.4
Control	8.5 fgh	45.4	0.1

[1] Small letters indicate Duncan's Multiple Range grouping of treatments which do not differ significantly at the 1% level.
[2] 2,4-dichloro-6-o-chloroanilino-s-triazine.
[3] 50% copper as the oxychloride.
[4] 75% 2,3 dinitrilo 1,4-dithiaanthraquinone.

Table 2. Storage in the first picking of Stokesdale tomatoes from spray plots at the end of 5 weeks in storage at 52.5° F.

Fungicide per 100 gallons	Total per cent rots	Per cent rots caused by				
		C coccodes	A tenuis	B cinerea	P infestans	Others[1]
Maneb, 2 lb.	28.3 c[2]	14.1 abc	6.0 a	6.0 ab	1.0	1.0
Maneb, 2 lb. + thiram 2 lb.	10.0 ghi	9.0 abcdef	1.0 a	0.0 b	0.0	0.0
Maneb, 2 lb. + Dyrene 2 lb.	13.0 efghi	6.0 cdef	3.0 a	3.0 b	0.0	1.0
Maneb, 1 lb. + thiram 1 lb.	13.0 efghi	6.0 cdef	5.0 a	2.0 b	0.0	0.0
Maneb 2 lb. + Dyrene 1 3/4 lb.	7.0 i	2.0 f	3.0 a	0.0 b	0.0	2.0
Zineb (factory-mix) 2 lb.	46.8 b	15.6 ab	12.2 a	12.4 a	5.1	1.0
Zineb 2 lb. + thiram 2 lb.	16.0 cdefghi	2.0 f	10.0 a	2.0 b	0.0	2.0
Zineb 2 lb. + Dyrene 1 3/4 lb.	11.1 fghi	6.0 cdef	5.0 a	0.0 b	0.0	0.0
Zineb 1 lb. + thiram 1 lb.	19.0 cdefghi	9.0 abcdef	3.0 a	5.0 b	0.0	2.0
Zineb 1 lb. + Dyrene, 1 lb.	15.0 cdefghi	7.0 bcdef	8.0 a	0.0 b	0 0	0.0
Zineb (tank-mix nabam 1 qt. + 3/4 lb. $ZnSO_4$	25.0 cdef	13.0 abcd	8.0 a	3.0 b	0.0	1.0
Zineb full strength + thiram 2 lb.	6.0 i	2.0 f	4.0 a	0.0 b	0.0	0.0
Zineb half strength + Dyrene 1 3/4 lb.	16.0 cdefghi	5.0 cdef	7.0 a	2.0 b	2.0	0.0
Zineb half strength + thiram 1 lb.	16.0 cdefghi	3.0 ef	6.0 a	5.0 b	0.0	2.0
Zineb half strength + Dyrene 1 lb.	23.0 cdefg	10.0 abcdef	7.0 a	4.0 b	1.0	1.0
Ziram 2 lb. Bordeaux 10-7, split program	28.0 cd	7.0 bcdef	8.0 a	5.0 b	2.0	6.0
Blitox 3 lb.	26.2 cde	17.0 a	5.0 a	1.0 b	0.0	3.0
Delan 1 lb.	22.0 cdefgh	7.0 bcdef	10.0 a	5.0 b	0.0	0.0
Control	66.5 a	12.0 abcd	3.5 a	0.5 b	47.5	3.0

[1] Includes P. destructiva, S. sclerotiorum and bacteria.

[2] Small letters indicate Duncan's Multiple Range grouping of treatments which do not differ significantly at the 5% level.

Table 3. Storage rots in the second picking of Stokesdale tomatoes from spray plots at the end of 5 weeks in storage at 52.5° F.

Fungicide per 100 gallons	Total per cent rots	Per cent rots caused by				
		C. coccodes	A. tenuis	B. cinerea	P. infestans	Others[1]
Maneb 2 lb.	27.0 cdef[2]	9.0 a	6.0 b	10.0 abc	1.0	1.0
Maneb 2 lb. + thiram 2 lb.	16.0 f	3.0 a	7.0 b	0.0 e	0.0	5.0
Maneb 2 lb. + Dyrene 2 lb.	16.0 f	6.0 a	6.0 b	3.0 de	1.0	0.0
Maneb 1 lb. + thiram 1 lb.	21.0 ef	4.0 a	6.0 b	7.0 abcd	0.0	4.0
Maneb 2 lb. + Dyrene 1 3/4 lb.	20.2 ef	8.0 a	8.0 b	0.0 e	0.0	4.0
Zineb (factory-mix) 2 lb.	40.4 cd	11.0 a	9.0 b	12.0 ab	0.0	6.0
Zineb 2 lb. + thiram 2 lb.	25.0 cdef	5.0 a	9.0 b	2.0 de	0.0	5.0
Zineb 2 lb. + Dyrene 1 3/4 lb.	17.3 f	8.3 a	6.0 b	2.0 de	0.0	0.0
Zineb 1 lb. + thiram 1 lb.	25.0 cdef	8.0 a	11.0 b	4.0 cde	1.0	1.0
Zineb 1 lb. + Dyrene 1 lb.	25.0 cdef	8.0 a	9.0 b	5.0 abcde	0.0	3.0
Zineb (tank-mix nabam 1 qt. +3/4 lb.$ZnSO_4$	37.0 cde	7.0 a	8.3 b	17.0 a	0.0	4.0
Zineb full strength + thiram 2 lb.	18.3 f	5.1 a	8.0 b	3.0 de	0.0	2.0
Zineb half strength + Dyrene 1 3/4 lb.	23.0 def	5.0 a	11.0 b	4.0 bcde	2.0	1.0
Zineb half strength + thiram 1 lb.	21.0 ef	4.0 a	8.0 b	7.0 bcde	1.0	1.0
Zineb half strength + Dyrene 1 lb.	28.0 cdef	6.0 a	15.0 b	2.0 de	1.0	2.0
Ziram 2 lb. Bordeaux 10-7, split program	59.0 b	12.0 a	40.0 a	2.0 de	0.0	2.0
Blitox 3 lb.	40.8 cd	20.3 a	15.3 b	4.0 bcde	0.0	0.0
Delan 1 lb.	42.0 bc	11.0 a	13.0 b	8.0 abcd	5.0	4.0
Control	76.5 a	9.6 a	5.0 b	2.0 cde	59.4	0.0

[1] Includes Penicillium sp., S. sclerotiorum, spotted wilt and bacteria.

[2] Small letters indicate Duncan's Multiple Range grouping of treatments which do not differ significantly at the 5% level.

The dominant causes of rots of stored tomatoes were Colletotrichum coccodes (Wallr.) Hughes, Alternaria tenuis Nees, B. cinerea and P. infestans. Microorganisms of lesser importance were Penicillium spp., Phoma destructiva Plowr., S. sclerotiorum, spotted wilt and bacteria.

Thiram combined with the higher rates of maneb or tank-mix zineb gave the most consistent control of rots (Tables 2 and 3). This agrees with the results obtained previously (2). The alternating program of ziram and bordeaux resulted in a high incidence of Alternaria rots in the second picking (Table 3). Blitox was unsatisfactory for the control of anthracnose. Botrytis rots increased when zineb and maneb were used alone, agreeing with the field results.

Recommendations

As a result of these tests growers in Nova Scotia are advised to use a fungicide mixture made up of either maneb or zineb at 1 lb to 100 gal (Imp.) plus 1 lb of thiram as a field spray for the control of tomato diseases. Dyrene at 1 3/4 lb can be used in place of thiram. When weather conditions favor a severe outbreak of either late blight or gray mold the amount of each fungicide should be increased to 2 lb. If fruit is to be harvested in the mature-green state for storage there would be some advantage in using tank-mix zineb instead of factory-mixes.

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CANADA AGRICULTURE RESEARCH STATION,
KENTVILLE, NOVA SCOTIA.

IONIZING RADIATION FOR THE CONTROL OF PLANT PATHOGENS

A REVIEW[1]

R.S. Willison[2]

Since conventional methods for controlling post-harvest rots in fruits often leave much to be desired, supplementary measures that do not add to the residue problem would be welcome. Accordingly, in the summer of 1962, an investigation of the control of brown rot and black mold of stone fruits by gamma radiation was proposed as a joint project involving Atomic Energy of Canada Ltd., the Ontario Horticultural Experiment Station, Vineland, and the Research Laboratory of the Canada Department of Agriculture, Vineland, Ontario. The literature was searched as a preliminary step in the project. Although studies of the reactions of micro-organisms to radiation began soon after the discovery of radioactivity (18), information on the adaptation of ionizing radiation to plant pathology is not yet voluminous. However, it was soon evident that investigations on the effects of such radiation on numerous micro-organisms, both *in vivo* and *in vitro*, were well beyond the preliminary exploratory stage. The present paper reviews various aspects of the application of ionizing radiation to the direct control of diseases in growing plants and of decays in harvested fruits and vegetables.

Types of radiation.

It may be useful to first review briefly the various types of radiation and their suitability for our purposes, as well as to define the units used in their measurements.

The ionizing effect is produced by the release of electrons and the formation of ionic pairs. Also, some of the energy lost by the radiation on impact is taken up by the surrounding atoms or molecules, induces structural changes of various degrees of magnitude, and produces heat. Ionizing radiation occurs in various forms in two categories: particulate rays, and high frequency or high energy electromagnetic waves. The main particulate radiations are:

(i) beta rays, or fast moving electrons with a negative charge.
(ii) protons, or positively charged hydrogen nuclei.
(iii) neutrons, or uncharged particles, each of the same mass as a proton.
(iv) deuterons, each composed of a neutron and a proton, therefore carrying one positive charge.
(v) alpha particles, or helium nuclei, each equivalent to a pair of deuterons and having a double positive charge.

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1 Publication No. 64, Research Laboratory, Research Branch, Canada Department of Agriculture, Vineland Station, Ontario.

2 Plant Pathologist.

The electromagnetic waves are similar to one another in type and effect, but differ in origin and often in frequency and wave-length. Gamma rays are high energy waves accompanying the emission of particles during the disintegration of certain radio-active substances. For example, Co^{60} emits both beta particles and gamma rays. X-rays are produced by a sudden change in the velocity of a stream of electrons (cathode ray) on impact with a target in a vacuum tube. They are caused by the resulting changes in the atoms of the target. X-rays are usually of longer wave-length or lower frequency than gamma rays, though modern machines are capable of producing high energy X-rays approximating the frequencies of gamma rays. X-rays and gamma rays range in wave-length from 0.01 to 1.4Å.

Ultraviolet light, with wave lengths of 2,000 to 4,000 Å, has not enough energy to be ionizing, but excites atoms by rearranging the orbits of their electrons. Ultraviolet light is useful for some types of surface sterilization, or asepsis, because many micro-organisms as well as viruses are sensitive to it. The behaviour of micro-organisms under ultraviolet light is however, different from that under ionizing radiation (21), and is outside the scope of the present review.

The alpha and beta particles are not deeply penetrating, but, as they have very high energy, cause surface burns and are particularly dangerous to humans if substances emitting them are ingested or inhaled. Gamma rays, X-rays, and neutrons, on the other hand, penetrate very deeply and even small doses, particularly of neutrons, can be very dangerous to human health. Beta radiation has been used by some plant pathologists for surface sterilization (9, 10, 17), but the more penetrating and pervasive types of radiation appear to be more suitable for most phytopathological purposes.

Units of measurement.

Several different units have been used by different workers to designate the amounts of radiation or dosages applied during irradiation. This variation in usage appears to have been due to the evolution of new concepts and a shift in emphasis, at least as far as radiation biology is concerned, from the radiations per se to the energy changes in the irradiated specimens.

The curie, "c", (10, 29) is the amount of radioactive material equivalent in activity to 1 gm of radium, in which 3.7×10^{10} atoms disintegrate per second, regardless of the products of the disintegration. For example, 1 c of cobalt 60, which yields gamma radiation, is equivalent in energy to only 2×10^{-4}c of polonium 210, which yields alpha particles.

The roentgen, "r" (10, 12, 14, 15, 26, 29, 30, 31) was originally applied to the measurement of the activity of X-rays, but more recently to gamma radiation also. The roentgen is defined as the amount of radiation that will produce 2.1×10^9 ion pairs in 1 cc of dry air at standard temperature and pressure. Exposure to a roentgen of X or gamma radiation, however, results in the uptake of almost 100 ergs per gram of irradiated water or tissue. The kilo-roentgen, 10^3 r or "kr" is used by some authors (19, 20, 31).

The roentgen - equivalent - physical, "rep" (2, 3, 4, 5, 17), derived directly from the roentgen, is the amount of energy that a roentgen of radiation delivers to a gram of wet tissue. The rep is defined as the amount of radiation that will produce an uptake of 93 ergs of energy by a gram of wet tissue.

The rad (6, 7, 8) and its multiple the Kilorad,"k rad" (1), are similar to, but more convenient than, the rep. The rad is a measure of absorbed energy induced by radiation and is equivalent to 100 ergs per gram of irradiated material, usually wet tissue. Since the rad indicates the amount of energy absorbed by a unit weight, it is independent of the type of radiation used. For practical purposes, the roentgen, rep, and rad represent approximately equivalent amounts of energy absorption.

Effects of radiation on pathogenic micro-organisms in vitro.

The sensitivity of plant-pathogenic micro-organisms to radiation in vitro appears to be highly complex, not only varying widely between organisms and between different stages of development and different functions of the same organism, but also being affected by substrate and growing conditions.

Some bacteria have been found to be more susceptible than others. According to Hellmers (14), Pectobacterium parthenii var. dianthicola (? Erwinia chrysanthemi) and P. carotovorum (E. carotovora), pathogenic to carnation, withstood gamma radiation at dosages of 1×10^5 r but not at 3×10^5 r. On the other hand, Dimond (12) and Waggoner and Dimond (29) found that cultures of Agrobacterium tumefaciens subjected to 5×10^4 r of gamma radiation at the rate of 80 r per hour were still capable of producing normal galls on non-irradiated plants, though 63 per cent of the bacteria were inactivated at 3×10^3 r and more than 90 per cent were killed at 1×10^4 r. It was therefore argued that the pathogenicity factor of A. tumefaciens was less susceptible to radiation effects than the survival factor.

Wide differences in the sensitivity of actively growing hyphae to radiation have been reported for different fungi, in some cases within the same genus (Table 1). Moreover, some species are resistant to many times the dosage lethal for others pathogenic to the same host: for example, Phytophthora infestans and Alternaria solani, isolated from potato (Table 1), and Phomopsis citri and Diplodia natalensis, from citrus fruits (Tables 1 and 2). Variations in resistance occurring within a species may be partly due to the age of the culture at the time of irradiation, since Kljajic (15) reported that the actively growing mycelium of several fungi was most sensitive in cultures 24 hours old. The nature of the medium in which the mycelium is growing is also an important factor. According to Stapleton (25), a medium containing organic complexes, such as proteins, exercised a greater protective effect on some organisms than one containing simple, chemically defined, ingredients. Sommer, Eckert, and Creasy (23) also obtained evidence of protective action when spores of Penicillium digitatum were irradiated, either in orange juice or in inoculated citrus fruits. A similar effect would also account for the different responses to irradiation

Table 1. Some plant pathogens grouped according to dosage of gamma radiation lethal to actively growing mycelium.

Dosage range*	Organism
Less than 1×10^5	Phomopsis citri, Phytophthora infestans (8).**
1×10^5 to 2.5×10^5	Botrytis allii, B. cinerea***, Monilinia fructicola, Pellicularia rolfsii, Penicillium digitatum, P. expansum, P. italicum, Phoma sp. (from blueberry,) Pythium debaryanum, Sclerotinia sclerotiorum (8); Alternaria solani,*** Ascochyta pisi,***, Aspergillus niger, Trichothecium roseum (15).
2.5×10^5 to 5×10^5	Alternaria citri, A. tenuis, Cladosporium sp. (from lemon), Gloeosporium musarum, Gloeosporium sp. (from blueberry), Rhizopus nigricans (from peach and sweet potato), Stemphylium radicinum (8).
5×10^5 to 7.5×10^5	Diplodia natalensis*** (8); Alternaria dianthi (14); Alternaria solani***, Ascochyta pisi*** (15).
7.5×10^5 to 1×10^6	Diplodia natalensis*** (8); Fusarium culmorum (14); Alternaria solani***, Ascochyta pisi***, Fusarium oxysporum f. vasinfectum (15), Botrytis cinerea*** (15) (17); F. oxysporum f. lycopersici (30).

* Dosage in rads, reference (8); in roentgens, references (14, 15, 30) in reps beta radiation, reference (17).

** Numbers in brackets indicate references.

*** Organisms in more than one category were variable in sensitivity, possibly at different stages of growth.

in Tochinai's and in Czapek's media exhibited by both conidia and hyphae of Monilinia fructicola, the peach brown rot fungus (5), and of the Penicillium species from citrus fruits (4) (Table 2). Tascher (26) found that seed-borne pathogens, such as Diplodia zeae and Gibberella saubinetti were much less sensitive to X-rays in dormant infected seed than they were in vitro on potato dextrose agar. The effect of substrate on the sensitivity of different fungi to irradiation is, however, far from uniform and varies greatly from one organism to another, as indicated by the investigations of Beraha and his colleagues (8), (see also Table 3).

In many but not all fungi, higher dosages of radiation were required to prevent germination of spores than to suppress growth of mycelium (Table 2). According to Kljajic (15), the conidia of Helminthosporium turcicum from corn, and Penicillium expansum from apple, were less susceptible than the mycelium, while the reverse was true for Botrytis cinerea from grape, and Fusarium oxysporum f. vasinfectum from cotton. In Aspergillus niger from grape, Ascochyta pisi from pea, Alternaria solani from potato, and Trichothecium roseum from plum, conidia and hyphae were equally resistant (or susceptible). Schwinghamer (21), reported that the calculated sensitivity to X-radiation of the mycelium of the flax rust, Melampsora lini, was ten times that of the uredospores irradiated at a comparable level of hydration. However, the uredospores of M. lini, Puccinia graminis f. sp. tritici, P. graminis f. sp. avenae, and P. coronata f. sp. avenae became increasingly sensitive to X-rays, gamma rays, and neutrons when their moisture content exceeded 45 per cent.

Sommer et al (23) investigated the response of spores of various fungi to gamma radiation and found that germination that occurred after irradiation commonly resulted in abnormal germ tubes capable of limited elongation only. They concluded that survival, as indicated by potential for unlimited growth, was generally more sensitive than the germination process. They also demonstrated that irradiated, abnormally germinating sporangiospores of Rhizopus stolonifer, though incapable of forming a colony, were able to hydrolyse pectin nearly as rapidly as similar, but non-irradiated spores. Castellani, Matta, and Guerzoni (10) regarded as signs of a true radiation disease the modifications in shape and structure of the germ tubes of spores of Gloesporium musarum that became more frequent with increases in dosage above 5×10^4 r (see also Table 2). On the other hand, at dosages well below the lethal level, ionizing radiation, whether gamma rays or neutrons, may stimulate germination of conidia and growth of cultures as reported by Vasudeva and colleagues (28) for Colletotrichum falcatum (Glomerella tacumanensis) and Ustilago nuda.

Effects of radiation on the host.

In peaches, abnormalities in texture and colour, such as softening of the flesh or browning of the skin, resulted from irradiation with gamma rays at doses greater than 4×10^5 rep but not at 3×10^5 rep (5). Beraha and his associates (5) considered that more subtle changes, such as an increase in ripening rate or an alteration in flavour, may occur at doses that cause no obvious injury.

Table 2. Dosage of radiation required for suppression of germination of conidia and growth of hyphae of various plant pathogens in vitro.

Organism	Host and Disease	Type of radiation	Dosages and Effect	Ref.
Botrytis cinerea	Grape and strawberry fruit rot	beta	1×10^5 rep. - growth delayed. 4×10^5 rep. - growth more or less suppressed. 8×10^5 rep. - cultures killed.	17
Diplodia natalensis Phomopsis citri	Orange - stem end rots	gamma	4.7×10^5 to 8.9×10^5 rad - hyphae killed. 4.6×10^4 to 9×10^4 rad - hyphae killed.	6
Gloeosporium musarum	Banana - anthracnose	beta	5×10^4r - affect shape and structure of germ tube 3×10^5r - germ'n. of conidia inhibited.	10
Monilinia fructicola	Peach - brown rot	gamma	2×10^5 rep - limit for hyphal growth 1×10^6 rep - 50% germ'n. of conidia*.	5
Rhizopus nigricans	Peach - black mold	gamma	4×10^5 rep - limit for hyphal growth 5×10^5 rep - 50% germ'n. of conidia.	5
Penicillium digitatum P. italicum	Citrus - fruit rots	gamma	1.03×10^5 rep - suppressed colonies** 1.57×10^5 rep - lethal to young hyphae** 4.7×10^5 rep - lethal to conidia**	4
Phytophthora infestans	Potato - late blight	gamma	2.3×10^4 rad - limit for hyphal growth 20 Kr - almost suppressed growth 100 Kr - lethal to hyphae	7 19

* In complex medium (Tochinai's, 25); in simple medium (Czapek's). 2×10^5 rep reduced germination to 30% (cf. Table 3).

** In Tochinai's medium; lower dosages required in Czapek's.

Table 3. Effect of culture medium* on resistance to gamma radiation of young actively growing mycelium of various fungi (8).

Rating order of media**	Organism
C=T	Botrytis cinerea (from blueberry), Gloeosporium sp. (from blueberry), Gloeosporium musarum (from banana).
C=H=T	Alternaria citri (from lemon), Alternaria tenuis (from tomato) Phytophthora infestans (from potato).
C=H>T	Penicillium expansum (from apple).
C>T>H	Rhizopus nigricans (from peach). (see H>C>T)
H=T>C	Monilinia fructicola (from peach).
H>C=T	Botrytis allii (from onion), B. cinerea (from grape and strawberry), Cladosporium sp. (from lemon), Phomopsis citri (from orange), Sclerotinia sclerotiorum (from bean), Stemphylium radicinum (from carrot).
H>C>T	Rhizopus nigricans (from sweet potato).
H>T>C	Penicillium digitatum (from lemon), P. italicum (from orange).
T>C	Pellicularia rolfsii (from watermelon), Phoma sp. (from blueberry).
T>C=H	Diplodia natalensis (from peach).
T>H>C	Pythium debaryanum (from potato).

* Czapek's (c), Tochinai's (T), and host (H).

** =, equally sensitive (or resistant) in both media; >, more resistant in the first than in the second medium of a pair.

The threshold for visible injury in oranges was slightly lower than that for peaches, about 2.75×10^5 rad of gamma radiation, whereas severe injury occurred at 4.5×10^5 rad and textural changes in the pulp at doses above 9×10^5 rad (6).

Nelson, Maxie, and Eukel (17) reported the grape varieties Tokay and Emperor to be somewhat less sensitive to beta radiation than the Thompson variety, for which the threshold of injury was at 2×10^5 rep. The latter variety became slightly brown and developed a cooked flavour in about 3 days at 3 to 4°C after exposure to 4×10^5 rep. At 8×10^5 rep injury was more severe, appeared earlier, and included cracking of the skin. The same authors (17) observed no injury in strawberries, variety Shasta, irradiated at a dose of 2×10^5 rep. Irradiation at 4×10^5 rep induced water-soaking, a "cooked" odour, and off flavors, and, at 8×10^5 rep, immediate exudation of juice followed by collapse in a few days.

Slight softening of potato tubers, variety Red Pontiac, occurred after irradiation at 1.37×10^5 rad. At higher doses, discoloration and softening became more pronounced (7). According to Rubin and colleagues (19) irradiation of tubers at doses of 10 kr (1×10^4 r) had no depressing effect on peroxidase activity, suberin formation, or wound biosynthesis of ascorbic acid. Periderm formation, however, was noticeably depressed, at least temporarily. Irradiation may therefore have an inhibitory effect on cell-division.

In Tascher's experiments with seed-borne diseases (26), dormant seeds of corn, wheat and barley were damaged by irradiation with X-rays at dosages between 1 and 2×10^4 r. Either the percentage of germination was reduced or the seedling plants were stunted or otherwise injured. As might be expected, germinating seed was even more sensitive than dormant seed.

Dimond (12) observed that young tomato plants irradiated with 3×10^4 r of gamma radiation at the rate of 80 r per hour had slow terminal growth, poorly developed leaf laminae, very poor root development, and the new growth lacked central parenchyma. These effects are interpreted as the result of impairment of the ability of the plant to undergo chromosomal and cell division and cell enlargement. Skoog (22) and later workers showed that exposure of plants to low doses of ionizing radiation caused temporary suppression of auxin production. At higher doses, this effect on auxins may become permanent. The interference with growth cell division observed in irradiated tomato plants (12, 29, 30, 31) is probably associated with lowered auxin levels.

Inhibition of growth in irradiated carnation cuttings was reported by Hellmers (14). Cuttings irradiated at 3×10^3 r rooted almost normally and the resulting plants grew well and produced good bloom, but after irradiation with dosages of 7×10^3 r or more, root formation was suppressed and the cuttings failed to grow. Wheat seedlings were somewhat more sensitive; Schwinghamer (20) noted that a 0.5 kr (5×10^2 r) dose of X-rays initiated inhibition of leaf development and a 1.5 kr dose caused stunting of roots and the formation of root-tip nodules.

Effects of radiation on host-pathogen relationships.

Changes effected through the host: Dimond (12) and Waggoner and Dimond (29) suppressed crown-gall formation completely on three hosts by exposing whole young plants to a dose of 3×10^4 r of continuous gamma radiation (chronic) at a rate of 1 to 2×10^3 r per day. X-rays in excess of 4×10^3 r at a rate of 1 to 5 r per second delayed the onset of gall formation in tomato for periods varying with the total dosage applied (Table 4). Irradiation also inhibited further growth of galls already present on the plants. Radiation was equally effective whether applied before or after inoculation with Agrobacterium tumefaciens, but bacteria capable of producing galls on non-irradiated plants could be isolated from galls suppressed by doses of 8 to 16×10^3 r. The authors therefore concluded that radiation suppressed the galls by affecting the host rather than the pathogen and that the formation of hyperplastic tissue was inhibited because auxins were at low levels.

Changes in the resistance of tomato to Fusarium oxysporum f. lycopersici were also observed by Waggoner and Dimond. Plants exposed to chronic gamma radiation at the time of inoculation or later were more susceptible than non-irradiated controls (30). On the other hand, irradiation with 21 kr of X-rays five to ten days before inoculation increased resistance markedly (31). When irradiation was restricted to certain parts of the plant, increased resistance was associated with treatment of the root before inoculation and, to a lesser extent, with treatment at the time of inoculation. Conversely, irradiation of the shoot at any time lowered resistance to some extent. Since F. oxysporum (Table 2) is resistant to levels of radiation much higher than the tomato plant can tolerate, it was concluded that this reduction in infection was also due to an effect on the host. It is generally accepted that root and shoot play different roles in auxin synthesis and use. Moreover, increases in the resistance of tomato to Fusarium wilt were accompanied by stunting of the plant. Furthermore, Davis and Dimond (11), working with the same host and fungus, showed that preinoculation treatments with plant growth regulators also reduced growth and increased resistance. Waggoner and Dimond (31) suggested, therefore, that, in the Fusarium - tomato interaction, radiation changes the resistance of the host by lowering its auxin level. The mechanism by which resistance is changed in this case is evidently different from that operative in the suppression of crown gall in the same host since the formation of hypertrophied tissue is not involved in the wilt disease.

In experiments with rust fungi and their specific hosts, Schwinghamer (20) used chronic doses of gamma radiation administered at the rate of 1 kr per day and acute single doses of X-rays at higher dosage rates. Chronic doses of 10 kr had no effect on the reaction of 16 varieties of flax resistant to Melampsora lini whether inoculation preceded or followed irradiation. In susceptable flax varieties, however, irradiation after inoculation caused a temporary delay in infection. In this case, the radiation was considered to have affected the rust, not the resistance of the host, since a 10 kr acute dose of X-radiation proved lethal to more than 90 per cent of day-old infections. In cereals, on the other hand, the host reacted to irradiation;

both acute and chronic irradiation before inoculation changed the reaction type of some varieties, but not of others. Any changes that occurred were invariably towards increased susceptibility. Chronic irradiation begun one day after inoculation was less effective in breaking resistance. Changes in host response were distinct at 5 kr doses of chronic radiation, but were initiated by as little as 1.5 kr of acute X-ray treatment and reached a maximum at approximately 3 kr. Changes in reaction type may be associated with specific physiological changes, since the reactions of a given variety to different races of rust changed to different degrees. For example, in wheat varieties normally resistant to races 15B and 111 of Puccinia graminis f. sp. tritici, irradiation induced a much greater shift towards susceptibility to race 15B than towards susceptibility to race 111. Irradiation of the crown or shoot apex of cereal seedlings affected both rust development and stunting of leaves much more than irradiation of either roots or leaves alone. In Schwinghamer's opinion, the association of rust reaction with a process initiated in the shoot apex and affecting growth of leaves suggests that growth-regulating substances may be involved directly or indirectly in the mechanisms governing the resistance or susceptibility of cereals to rust fungi. If so, the type of resistance encountered here differs from the examples cited in the preceding paragraphs, since the shift was towards increased susceptibility, rather than towards increased resistance.

Changes effected through the pathogen: Since Brasch and Huber (9) demonstrated the possibility of using beta rays to prevent deterioration of foodstuffs in storage, considerable work has been done on the use of radiation in the post-harvest control of pathogens causing rots, decays, or molds in fruits and vegetables. In almost every case, the dosage required either for surface sterilization or to kill the pathogen in existing infections is well above the level tolerated by the host tissue. Thus, as Hannan (13) pointed out, it is not practicable to control decays in most fruits and vegetables by using radiation as a sterilant. However, as already intimated, radiation effects start to occur in germ tubes and growing hyphae at dosages below the lethal level (10, 23). Treatment at appropriate dosages of radiation, then, could be expected to bring fungistasis into play (2, 17), so that decays or rots could be checked indefinitely or at least long enough to prevent wastage of perishable products during distribution and sale (Table 4).

Some complications may arise from the fact that low doses of radiation may stimulate fungal growth (28). For example, Beraha et al (6) reported that Diplodia natalensis induced more and faster rotting in inoculated oranges irradiated at dosages of 2×10^5 rad or less (Table 4). Also, Mathie and Marais (16) found that low doses of X-radiation increased the rate of apple decay by Penicillium expansum, and Rubin et al, (19), (also Table 4) showed that 10 kr of gamma radiation stimulated Phytophthor rot of potato tubers. Beraha and colleagues (4) described an even more involved interaction, in that irradiation at dosage levels preventing decay of oranges and lemons by radiation-sensitive fungi, such as Penicillium digitatum and P. italicum, may expose the fruit to decay by organisms that do not ordinarily attack it.

Table 4. Effects of ionizing radiation on infection.

Organism	Host and Disease	Rad'n.	Dosage and Effect	Ref.
Agrobacterium tumefaciens	Tomato crown gall	X-ray	4×10^3r delayed galls 3 or 4 days	12
		X-ray	6×10^3r delayed galls 3 weeks.	29
		gamma	3×10^4r suppressed gall form'n.	
Erwinia carotovora	Potato soft rot	gamma	4.8×10^5 rad did not suppress rot in tubers	7
Phytophthora infestans	late blight	gamma	10 kr stimulated tuber decay	19
			4.5×10^4 rad prevented decay ($\pm 1 \times 10^5$ rad)8	7
Botrytis cinerea	Strawberry rot	gamma	2×10^5 rep checked inf'n.	3
		beta	1 to 2×10^5 rep. reduced rot	17
Rhizopus nigricans	mold	gamma	2×10^5 rep checked inf'n. ($2 \times 4 \times 10^5$ rep)	3
B. cinerea	Grape rot	gamma	5×10^5 rep delayed inf'n. 10 days	3
		beta	1 to 2×10^5 rep. reduced inf'n. (2 to 4×10^5 rep)	17
Diplodia natalensis	Citrus stem-end rots	gamma	2×10^5 rad stimulated inf'n.	6
			2.75×10^5 rad checked inf'n.	
Phomopsis citri		gamma	9×10^4 rad checked inf'n.	6
			1.15×10^5 rad stopped inf'n.	
Penicillium spp.	fruit rots	gamma	1.5 to 2×10^5 rep stopped rot** (a) for 12 days at 75°F. (b) for 17 days at 55°F. checks rotted in 3 days at 75°F. (2.75×10^5 rad)	4
Penicillium expansum	Apple fruit rot	gamma	1×10^5 rep suppressed rot 6 days at 70-75°F.	3
			2×10^5 rep suppressed rot 10 days at 70-75°F.	
Monilinia fructicola	Peach brown rot	gamma	2×10^5 rep delayed rot 10 days at 80-85°F.	5
Rhizopus nigricans	mold	gamma	2.5×10^5 rep delayed inf'n. 10 days at 80-85°F. (3 to 4×10^5 rep)	5

** In brackets, threshold dose for injury to host.
Higher doses required for established than for incipient infections.

In the instance cited, Alternaria citri developed and caused rot in irradiated fruit, but not in the non-irradiated controls.

The flux of radiation, or dose-rate, is also an important factor, since it determines the effective total dose required. Dimond (12) stated that the extent of crown-gall suppression in tomato decreased as the dose-rate decreased, so that total doses that prevented gall formation when delivered at a flux of 80 r per hour had no effect when delivered at 5 r per hour. According to Beraha et al. (7), doses of 1.37×10^5 r or more of gamma radiation gave complete control of Pythium debaryanum in potato tubers when administered at 7×10^3 r per minute, but not when administered at 3×10^3 r per minute. Beraha (1) also demonstrated that blue mold (Penicillium italicum) was not completely controlled in inoculated oranges held 12 days at 75°F after irradiation with 182 k rad of gamma radiation at 3 k rad per minute whereas at 20 k rad per minute a dose of 157 to 182 k rad was effective, as was a dose of 125 to 137 k rad at 40 k rad per minute. Somewhat similar results were obtained with green mold (P. digitatum) in the same host. Similarly, 125 to 150 k rad total dose delivered at 25 k rad per minute almost completely suppressed brown rot (Monilinia fructicola) in peaches and gray mold (Botrytis cinerea) in pears for 17 days, but 210 k rad at 2.5 k rad per minute did not control decay (1).

Host response is also affected by dose rate (1). It is inferred from several reports that this response is not necessarily of the same magnitude as that of the pathogen, though there is no explicit statement to that effect. The optimum flux and total dose would have to be determined experimentally for each host-pathogen complex.

Discussion and Summary.

The information presented above leaves no doubt that micro-organisms, generally, can survive larger doses of ionizing radiation than higher plants can tolerate. The direct control of diseases in growing plants by the fungicidal action of radiation, therefore, is not likely to be feasible and, indeed, has not often been attempted. Hellmers' results are typical (14) (see also Table 1). He showed that, whereas bacterial and fungal pathogens of carnation were not seriously affected by gamma radiation at dosages of 1×10^5 r or lower, carnation cuttings were prevented from rooting by irradiation at 7×10^3 r.

Irradiation of seeds for the control of seed-borne diseases seems equally impracticable. Tascher (26) found that X-rays, in dosages great enough to inhibit the pathogens, in most cases either impaired germination or injured the young seedlings.

Controlling disease by altering the resistance of a host to its pathogen is no more promising than the more direct approach. Dimond (12) and Waggoner and Dimond (29 and 31) used gamma radiation to suppress crown gall development and to increase resistance to Fusarium wilt in tomato plants, but control of the disease was counterbalanced by deleterious effects on the treated plants. In Schwinghamer's experiments (2), irradiation not only induced abnormalities in wheat plants, but lowered their resistance to

certain strains of stem rust.

The disappointing results of irradiating plants during their growth and development are no doubt due to the cell nucleus being the principal site of damage, particularly during mitosis and meiosis. Sparrow and Woodwell (24) have correlated the sensitivity of higher plants with chromosomal and nuclear characteristics. Plants with large nuclei and low chromosome numbers are much more sensitive than polyploids and plants with small nuclei and high chromosome numbers. Plants vary so greatly in these respects that differences in sensitivity between species may be as much as 500-fold (24). As micro-organisms also vary widely in sensitivity, control of some diseases may be possible, if not practical.

Because of the sensitivity of dividing nuclei, mature tissues, in which cell division has virtually ceased, should not be subject to as wide a range of radiation damage as immature growing tissue. Irradiation for the control of wastage in harvested fruits and vegetables may therefore be feasible, but the working margin would depend on the host-pathogen combination. The effective range of treatment is determined, on one side, by the critical minimum for injury to the host and, on the other, by the critical maximum dose for stimulation of the pathogen (6, 16). In some cases, these two limits may be too close for practical purposes; for example, in the control of Diplodia stem rot of citrus fruit and Botrytis rot of grape (Table 4). In others, they overlap completely and control is not possible, as with soft rot of potato (Table 4). In still others, e. g. Phytophthora rot of potato, Phomopsis and Penicillium rots of citrus fruits, Botrytis rot and Rhizopus mold of strawberry, and Monilinia rot and Rhizopus mold of peach (Table 4), the critical points are far enough apart to offer some promise of practical application.

It should be remembered that, in almost all the experimental work with fruits and vegetables, the samples under test were artificially inoculated, usually in wounds, so that incipient infections were present before irradiation was undertaken. It is for this reason that the radiation is considered to act on the fungus, not on the host, in these cases, at least so far as control is concerned. The data presented in Table 4, therefore, represent tests made under more exacting conditions than would normally occur in properly handled commercial packs. It seems reasonable to expect more satisfactory control of wastage in sound fruit than in injured fruit in which infections have already started, since the germination of irradiated fungal spores is usually abnormal (23).

The favorable results already obtained experimentally with peaches (3, 5) warrant continuation of the proposed project to control brown rot and mold by gamma radiation. Logically, future work should proceed mainly along practical lines on a semi-commercial scale with uninjured fruit packed in the usual way in commercial containers. Experimentation should be planned to determine the effects of irradiation on the shelf life of the fruit under a variety of conditions both before and after treatment. Before the method can be put into commercial practice, the economic and mechanical aspects of the problem will also have to be considered. It is expected that safety requirements and the exacting nature of the operation will necessitate treatment at central locations under the supervision of trained personnel.

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RESEARCH LABORATORY, CANADA AGRICULTURE,
VINELAND STATION, ONT.

NATURAL ROOT GRAFTING IN CHERRY, AND SPREAD OF CHERRY TWISTED LEAF VIRUS[1]

F. W. L. Keane[2] and James May[2]

Abstract

Symptoms of the twisted leaf disease appeared in 4 uninoculated sweet cherry trees, closely interplanted with inoculated trees. This represented an exceptional rate of spread for twisted leaf virus. All trees were on mazzard seedling rootstocks. Exposure of the root systems disclosed numerous root grafts, providing union of xylem and phloem tissues.

Introduction

Natural root grafting of forest trees has received increasing attention during the past 12 years (1). Root grafting of fruit trees has been recorded (2) but has been much less intensively studied. Although movement of viruses from tree to tree through such unions is taken for granted by most investigators of tree fruit virus diseases, there have been few published reports of the correlation of virus spread with the occurrence of root grafting.

Experimental transmission of phony peach virus by root grafting has been reported by Hutchins (5). McCrum (6) has experimentally transmitted apple mosaic and stem pitting viruses by grafting together in pairs the tap roots of dormant apple seedlings.

Natural root grafting has been reported by Hunter, Chamberlain and Atkinson (4) to be responsible for transmission of apple mosaic virus, and Hobart (3) has noted passage of necrotic ring spot, yellows, and prune dwarf viruses through intraspecific root grafts of Prunus mahaleb, P. avium, and P. americana seedlings, and also through interspecific root grafts among these.

There has been evidence of the spread of cherry twisted leaf virus through natural root grafts in experimental plots at Summerland.

History of the Test Trees

Twenty Prunus avium (mazzard) seedlings were planted in a nursery row in 1953, at spacings varying from 4 inches to 15 inches. In June, 1954, 16 of the trees were inoculated, by budding, with the virus of cherry twisted leaf. In November, 1955, 4 of the inoculated trees were killed by low temperatures. The surviving trees were numbered from 1 to 16. The gaps between

[1]Contribution No. 128, Canada Department of Agriculture, Research Station, Summerland, British Columbia.
[2]Technician.

Table 1. Occurrence of natural root grafting (self grafts and intraspecific grafts) in a row of cherry trees.

Tree Nos.	Spacing between trees (inches)	Number of grafts
1-1		0
1-2	6	6
2-2		1
2-3	15	0
3-3		2
3-4	28	0
4-4		0
4-5	24	0
5-5		0
5-6	12	3
6-6		0
6-7	8	1
7-7		0
7-8	15	0
8-8		0
8-9	15	0
9-9		1
* 9-10	4	2
10-10		0
10-11	10	0
11-11		0
11-12	12	0
12-12		1
*12-13	4	3
13-13		0
13-14	12	0
14-14		0
* 14-15	12	1
15-15		0
15-16	12	1
16-16		0
10-13	26	1

*Unions between roots of inoculated and uninoculated trees.

them now ranged from 4 inches to 28 inches (Table 1). Twelve of the survivors were inoculated trees, and 4 trees, numbered 8, 10, 13 and 14, were uninoculated checks. All the 16 seedlings were budded to the variety Bing in September, 1955, and, where necessary, were re-budded to Bing in September, 1956.

Virus Spread

Twisted leaf symptoms were evident on most of the inoculated trees in 1956, and appeared on all inoculated trees in 1957. The uninoculated trees, 8, 10, 13 and 14, were symptomless in 1956. In 1957, tree 10 displayed severe twisted leaf symptoms, and tree 13 displayed mild twisted leaf symptoms. The other 2 uninoculated trees displayed no symptoms. In 1958, trees 10 and 13 displayed severe symptoms, and tree 8 had very mild twisted leaf symptoms. In 1959, trees 8, 10 and 13 displayed severe symptoms, and tree 14 displayed symptoms of moderate severity. Thus, within 5 years of the inoculation of neighbouring trees, all 4 of the uninoculated trees had become diseased as a result of natural spread.

Root Grafting

In June, 1961, a trench was dug parallel to the row of trees and water under high pressure was used to wash into this trench the soil surrounding the roots of the trees (Fig. 1). Careful examination revealed numerous root grafts, including self-grafting between roots of single trees, and intraspecific grafting among roots of neighbouring trees (Table 1).

Most of the observed grafts were among crowded roots, near their bases on the crown of the tree, but several were among relatively uncrowded roots at greater distances from the crowns. There were grafts between parallel roots (Fig. 2a) and between roots meeting at right angles (Fig. 2b). Grafting as a result of pressure was suggested by one graft of a small root within the fork of 2 larger roots (Fig. 2c). Several of the grafts were associated with galls (Fig. 2d).

Cross sectioning of root grafts demonstrated that unions of phloem and of xylem tissues had occurred (Fig. 3).

Roots of uninoculated trees 10, 13 and 14, had united with roots of inoculated trees by obvious root grafts. For tree 8 no such union was apparent.

Discussion

Natural root grafting among closely planted cherry trees has been demonstrated. The union of both xylem and phloem tissues in these root grafts indicates that a means of passage from tree to tree is provided for any virus that invades root tissues.

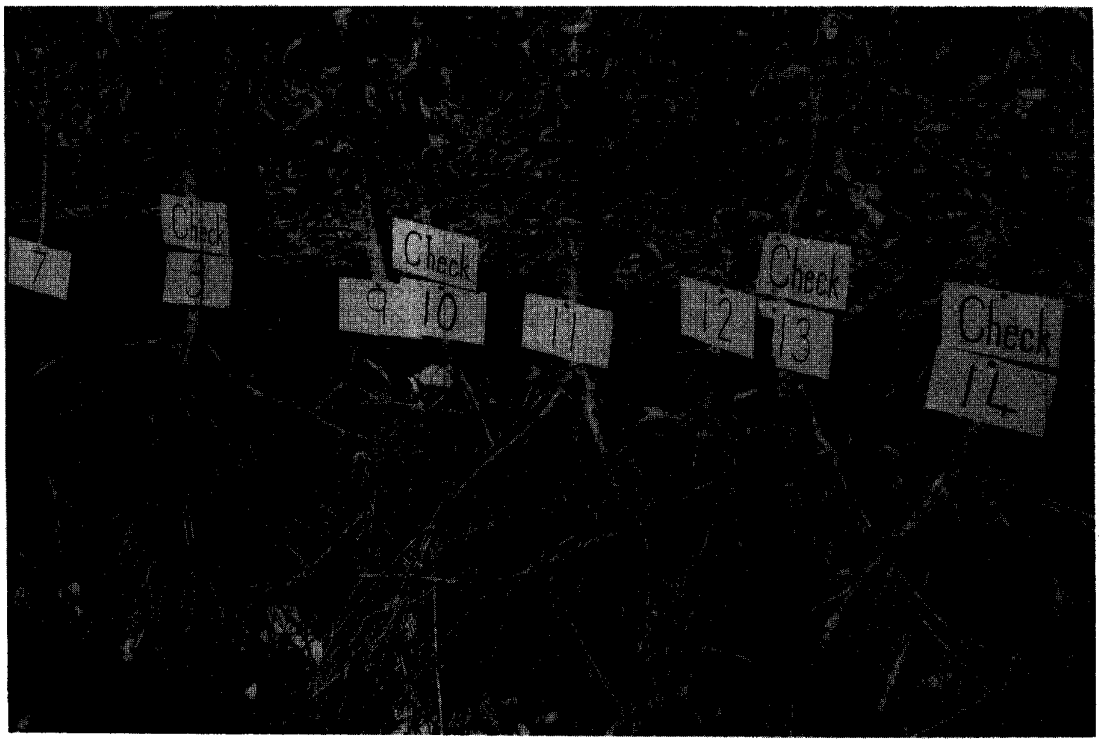

Figure 1. Cherry trees in situ following high-pressure washing of soil from roots.

The evidence supporting root transmission of the twisted leaf virus among these trees is strong. The experimental plantings among which they were growing have included many hundreds of cherry trees planted at various spacings. Among them have been trees inoculated with twisted leaf virus, inoculated with other viruses, and uninoculated trees. Natural spread of twisted leaf virus among them has been rare. Where it has occurred, it has been to trees adjacent to infected trees at spacings that would provide opportunity for natural root grafting. The recorded spread to all 4 of the uninoculated trees in the one row was an exceptional occurrence. The trees in this row were much more closely spaced than were any others in the orchard.

The method used to excavate the soil in the root zones did not allow examination of all possible sites of root grafting. Some root terminals were destroyed in digging the trench. The water pressure required to remove soil from the root systems was sufficient to damage weak graft unions and unions that had occurred among very small roots. Roots tended to break before their tips could be washed free. The absence of obvious root unions for 1 of the 4 trees that became infected could be attributed to any of these factors.

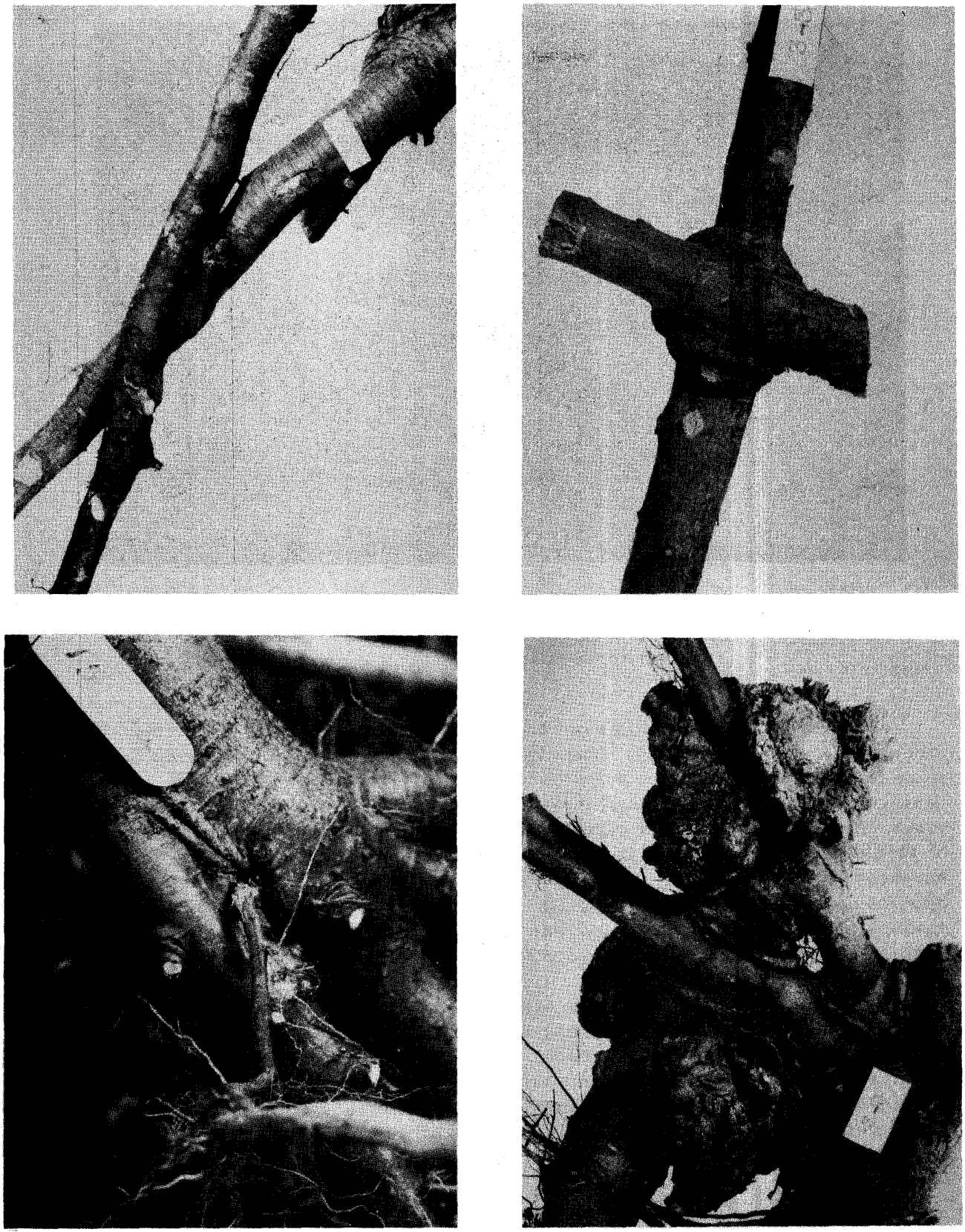

Figure 2. Natural grafting among mazzard cherry roots: (a) of parallel roots; (b) of roots meeting at right angles; (c) of a small root with larger roots between which it has been trapped; (d) associated with galls.

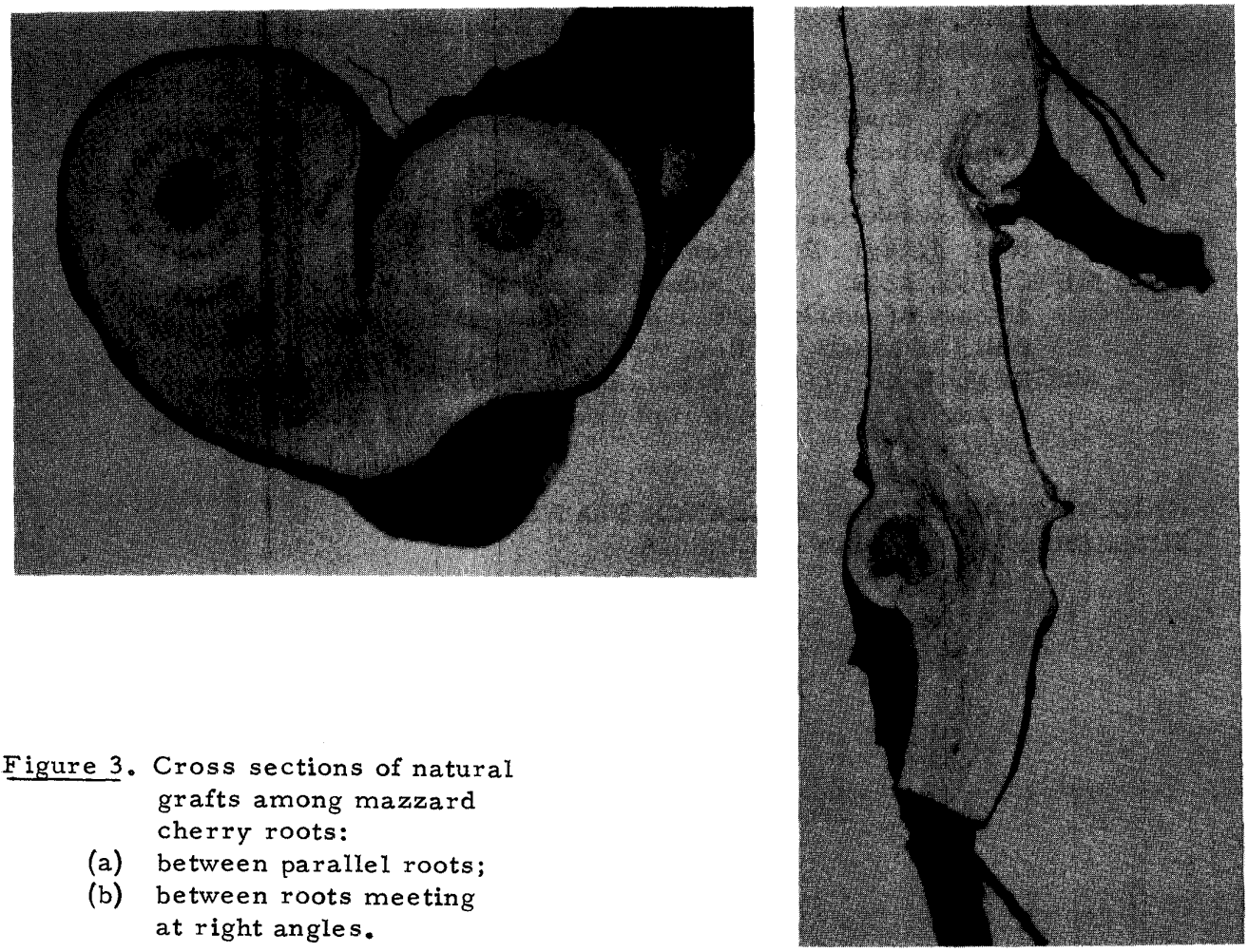

Figure 3. Cross sections of natural grafts among mazzard cherry roots:
(a) between parallel roots;
(b) between roots meeting at right angles.

These data augment the experimental and observational evidence that is accumulating to substantiate the common assumption of tree fruit virus spread among neighbouring trees through root grafts.

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CANADA AGRICULTURE RESEARCH STATION,
SUMMERLAND, BRITISH COLUMBIA.