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GUEST EDITORIAL

With this issue of the Canadian Plant Disease Survey, information on the disease situation in Canada will appear quarterly instead of in a single Annual Report. The most obvious advantage in the more frequent appearance of the Survey is the privilege of submitting valuable bits of information for inclusion while they are still news. The present compiler and editor, Mr. D. W. Creelman, is to be congratulated in achieving this change-over.

It is well to recall that the Canadian Plant Disease Survey arose from a keen desire of Canadian plant pathologists for a medium where "data on the annual prevalence of the commoner plant diseases" might be recorded. Formal action was taken at Guelph in 1919 at the first meeting of the Canadian Branch of the American Phytopathological Society. The first report was compiled by Dr. W.H. Rankin, St. Catharines, and Mr. W.P. Fraser, Saskatoon, from observations collected in the 1920 season and the Department undertook to mimeograph the report. In 1922 the Department assumed the further responsibility in its publication by assigning the compilation of the report to an officer of the Division of Botany at Ottawa. Dr. F.L. Drayton prepared the next two annual reports and an excellent 5-year summary for 1920-1924 (Canada Dept. Agr. Bull. 71, N.S., 1926). Mr. J.B. McCurry was responsible for the reports for 1925 to 1928, when in 1929 I came to Ottawa to head the Survey. Alone or assisted by other officers, notably Dr. D.B.O. Savile, I compiled the reports from 1929 to 1956. From the outset it was recognized that the success of the project depended upon the voluntary efforts of plant pathologists and others interested in plant diseases across Canada. A debt of gratitude is owed to those who were willing to take time from their busy research and teaching programs to make available their observations on current outbreaks of plant diseases for our mutual benefit.

It cannot be stressed too strongly that the continued success of the Survey depends in a large measure on the continued willingness of plant pathologists to contribute to the publication. The new quarterly will provide far greater freedom and flexibility in the publication of special reports by individual authors than was possible in an annual report on account of space limitations. In addition, the quarterly should be a suitable vehicle for results of fungicide trials, where prompt publication is all important.

However, if the quarterly is to prosper, it behooves us to submit our contributions promptly when requested and encourage Mr. Creelman wherever possible to make the new publication a success.

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VARIETAL REACTION OF OATS TO THE SEPTORIA DISEASE UNDER FIELD AND GREENHOUSE CONDITIONS[1]

R. V. Clark and F. J. Zillinsky[2]

Abstract

Varieties of common oats (Avena sativa) show very little resistance to the Septoria disease in field and greenhouse tests. Several selected varieties and strains showed moderate tolerance under field conditions but gave a susceptible reaction when heavily inoculated in the greenhouse. A number of varieties from wild species, especially those in the diploid group, showed a much higher level of resistance. The differences in reaction between these and varieties of common oats was particularly noticeable when the varieties were compared in the greenhouse several weeks after inoculation. Macrospore inoculations in the greenhouse resulted in too severe an infection to permit the detection of small differences in reaction. The use of ascospores as inoculum indicated that this might be a better means of screening varieties in the greenhouse for resistance.

Introduction

The disease of oats caused by the fungus Leptosphaeria avenaria Weber f. sp. avenaria (imperfect stage = Septoria avenae Frank f. sp. avenae) has become, in recent years, a major problem of this crop in Canada. This disease is especially prevalent in Ontario, Quebec and the Atlantic Provinces. Infection by the fungus usually results in severe leaf lesioning and necrosis, stem blackening and kernel blight. In some years extensive lodging occurs which results in an almost complete loss of crop. Invasion of the oat glumes and kernels is also of considerable importance in certain years. In 1954, Derick (2) reported that under natural conditions some varieties showed more tolerance than others to the causal fungus in Eastern Canada but the tolerance was not present in the commonly grown varieties. Several workers in the United States have also reported differences in resistance among varieties (3, 4, 5, 6, 7, 8, 9, 10, 11).

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[1]Contribution No. 37 from the Genetics and Plant Breeding Research Institute, Research Branch, Canada Agriculture, Ottawa.

[2]Plant Pathologist and Cerealist respectively.

During the past few years numerous varieties and hybrids have been tested at Ottawa for their varietal reaction to L. avenaria f. sp. avenaria both in the field and greenhouse. This paper summarizes the results obtained since 1956.

Materials and Methods

In 1956, a selected group of varieties and hybrids was grown in field tests at 8 locations throughout Eastern Canada, including Ottawa. In 1957 and 1958 the material under test was grown only at Ottawa and was inoculated artificially with a highly virulent isolate of L. avenaria f. sp. avenaria using a water suspension of macrospores and mycelium. The fungus was grown on oat-leaf agar in plates for 7 days; then the cultures were macerated in a Waring blender in 100 c.c. of water per plate. A further dilution of 1:5 with water was made and finally a few drops of the spreader Tween 20 was added. The inoculum was applied with a knapsack type sprayer and inoculating was done only in the evenings.

Leaf blotch readings were taken twice during the growing season for each entry tested by estimating in percent, the area of plant covered by lesions. Substantial differences in maturity date of the entries made it necessary to take more than one reading. Stem infection readings were taken at maturity by estimating in percent, the amount of stem blackening. In the 1956 test, samples of straw of each entry grown at the 7 outside locations were sent to Ottawa and examined but the leaf blotch notes were recorded by the co-operators at the testing stations.

Testing in the greenhouse was carried out in 2 ways. The material was grown in a greenhouse bed for 3 to 4 weeks with at least 2 replicates included. Several hours before the plants were inoculated, the bed was completely covered with a large polyethylene sheet supported by a wire frame. The air inside was saturated with moisture by using a pneumatic atomizing nozzle placed at one end of the bed. During the winter months, the soil temperature was maintained at 70°F by means of a thermostatically controlled heating cable. The plants were inoculated with a spore-mycelium suspension of a highly virulent isolate as previously described, with the exception that the second dilution of 1:5 with water was not made. A pressure-vacuum sprayer supplying 5-10 lb. pressure was used to inoculate the plants and high humidity was maintained for 48 hours following inoculation. The second method of testing was to grow the test material in greenhouse flats and, in a few cases, greenhouse pots for 3 to 4 weeks and then to place the flats or pots in glass-enclosed chambers in which the humidity and temperature were controlled. The plants were inoculated in the same manner as those grown in the greenhouse bed and the high humidity was maintained for 48 hours.

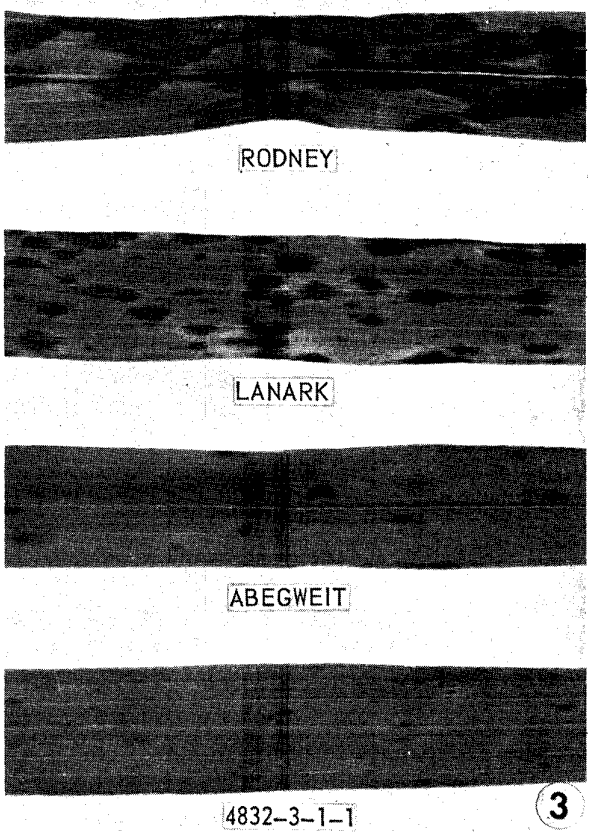

FIG. 1. LESION DEVELOPMENT ON THREE VARIETIES OF OATS APPROXIMATELY ONE MONTH AFTER INOCULATION IN THE GREENHOUSE WITH MACROSPORES OF *SEPTORIA AVENAE* F. SP. *AVENAE*. FROM LEFT TO RIGHT: RODNEY, C.D. 3820 AND ABEGWEIT. FIG. 2. BLACK STEM DEVELOPMENT ON THREE VARIETIES OF OATS AT MATURITY AFTER INOCULATION IN THE GREENHOUSE WITH MACROSPORES OF *SEPTORIA AVENAE* F.SP. *AVENAE*. FROM LEFT TO RIGHT: C.D. 3820, ABEGWEIT AND RODNEY. FIG. 3. LESION DEVELOPMENT ON LEAVES OF FOUR VARIETIES OF OATS INOCULATED WITH ASCOPORES OF *LEPTOSPHAERIA AVENARIA* F. SP. *AVENARIA*. FROM TOP TO BOTTOM: RODNEY, LANARK, ABEGWEIT AND 4832-3 -1-1.

In the greenhouse, disease readings were determined 10 to 14 days following inoculation by estimating the percent infection on the leaves. The fungus does not develop in the greenhouse to any extent on tissues produced after inoculation and therefore, only the inoculated portions of the leaves were considered in the readings. In a few instances, the plants were kept for approximately a month after inoculation and readings taken then were compared with those taken earlier.

A study of infection resulting from inoculation with ascospores was also made, since this spore form has been found to be the principal source of initial infection in the field (1). The plants were grown in greenhouse flats and pots and before inoculation placed in polyethylene-enclosed chambers in which the air was saturated with moisture. The ascospore inoculum was obtained by placing a number of pots containing overwintered oat stubble, showing typical black stem symptoms, in the chambers. Also, overwintered oat straw was suspended over the flats or pots in the chambers. The test plants and the straw were completely saturated with moisture several times to insure the release of the ascospores from the fruiting bodies and to favor infection and subsequent disease development. Disease reaction ratings were made just prior to heading.

Results

Forty-four selected varieties and hybrids were tested in the field in 1956, 43 in 1957 and 88 in 1958. Those tested included many varieties which had shown some resistance in previous tests in Eastern Canada and in other areas, principally the United States. Some of the entries were tested all three years while others were tested for only one or two years. Highly susceptible varieties were discarded after one year's testing. All varieties showing any indication of tolerance in the field were tested in the greenhouse to determine if the reaction obtained in the field was comparable to that following greenhouse infection by macrospores. A random group of approximately 100 varieties was exposed to ascosporic infection in the greenhouse.

The average leaf-infection reading of all entries in the field in 1956 was 27 percent, in 1957 42 percent and in 1958 46 percent. The 1956 average reading was low because the varieties were tested at 8 locations and the disease incidence was low at some of the locations outside Ottawa. The varieties that showed the best average tolerance to the Septoria disease in the field are shown in Table 1. In all tests the varieties Rodney and Abegweit were used as checks. Rodney was consistently more susceptible than Abegweit. A few varieties of Avena sativa including three Ottawa strains, 4832-3-1-1, 5055-13 and 5055-46 (Russell) showed as good as or better tolerance than Abegweit. These three strains have Abegweit in their

parentage, which may account for their high tolerance. Scots Berlie, an introduction from Scotland, also showed good tolerance but this is a semi-winter type and not suitable for commercial use in Canada.

In 1959 several hundred panicle selections from each of the most tolerant entries were grown in single rows in a Septoria disease nursery in the field. These were carefully examined and compared with the check varieties, Abegweit and Rodney, in an effort to obtain selections with improved tolerance. The selections of all strains except Scots Berlie were very uniform in reaction and provided little or no improvement over the original strain from which they were obtained.

Several varieties derived from the species A. brevis, A. nudibrevis, A. strigosa, and A. wiestii also showed good tolerance to this fungus and some were considerably better than those from A. sativa (Table 1). However, all the non-cultivated species did not show this tolerance, as evidenced by A. byzantina and all the varieties within a species were not uniformly tolerant, e.g., C.D. 1009, A. strigosa.

Some varieties varied in response from year to year in the field. Apparently these varieties either happened to be in a location unfavorable for disease development or escaped because of maturity differences. There was good agreement between leaf and stem reaction with most varieties. If the leaf reaction was susceptible then black stem would usually be plentiful as well. Some late-maturing varieties showed less stem lesioning than early-maturing varieties. The variety Alexander (Table 1) was an exception in that it showed very little leaf lesioning in 1956 but a high rating for black stem. However, the following years the leaf lesioning was quite high and the black stem symptoms considerably reduced.

Moderate to high infection ratings were obtained on all varieties of A. sativa in the greenhouse using macrospores as inoculum (Table 1). Some of the varieties from the wild species showed more tolerance than the common varieties but the greenhouse readings were much higher than the field ratings. Apparently, with an increase of the spore concentration in the inoculum the disease incidence was also increased. There was a relatively good agreement between field and greenhouse results. The most tolerant varieties in the field were also the most tolerant in the greenhouse.

Greenhouse tests, in which plants were kept for a month or more following inoculation, showed that the more tolerant varieties, especially those from the wild species, developed numerous, small leaf lesions (initially) but these did not enlarge to any extent and there was very little evidence of wilting and dying. In the common varieties, however, the lesions continued to enlarge and many of the infected leaves died. Figure 1 shows the disease development on plants of Rodney, C.D. 3820 and Abegweit inoculated approximately one month previously with macrospores of S.

Table 1. Disease ratings in percent for a selected group of oat varieties and hybrid strains which were grown under field and greenhouse conditions and subjected to natural and artificial inoculation with the fungus Leptosphaeria avenaria f. sp. avenaria. This group includes the varieties that showed the most tolerance to this fungus.

Species and Variety	Identification*	Field infection						Greenhouse leaf infection	
		Leaf lesioning			Stem blackening				
		1956	1957	1958	1956	1957	1958	Macro-spores	Asco-spores
A. sativa									
Abegweit	C.A.N. 693	27	35	30	28	30	40	65	42
Rodney	C.A.N. 761	38	50	50	72	70	60	80	57
Scots Berlie	C.A.N. 208	24	25	25	36	20	40	70	30
Wolverine	C.A.N. 106	-	45	35	-	40	40	60	-
Alexander	C.I. 1592	16	45	60	60	30	40	60	50
4832-3-1-1	C.A.N. 871	-	25	25	-	30	30	60	20
5055-13	C.A.N. 845	-	25	25	-	30	30	60	30
5055-46	C.A.N. 844	-	30	30	-	35	40	65	40
A. byzantina	C.D. 6872	-	30	45	-	40	60	75	60
A. brevis	C.D. 813	22	-	20	22	-	10	50	10
	C.D. 999	20	-	25	18	-	30	45	-
	C.D. 1001	21	-	25	22	-	30	45	20
	C.D. 1002	24	-	30	24	-	30	40	30
A. nudibrevis	C.D. 1017	20	-	15	18	-	30	40	20
A. strigosa	C.D. 1009	28	45	40	36	30	40	70	60
	C.D. 1014	20	20	25	22	20	30	35	30
	C.D. 3820	22	-	25	26	-	30	40	20
Saia	C.D. 4002	-	-	30	-	-	20	40	20
A. wiestii	C.D. 814	28	-	30	26	-	30	50	20

* C.A.N., C.I. and C.D. refer to Canadian Accession Number, Cereal Investigation Number, U.S.D.A. and Cereal Crops Division Number respectively.

avena f. sp. avenae. The lesions on Rodney were large and plentiful with many of the infected leaves dead. Abegweit was approximately the same but fewer dead leaves were evident. The lesions on C. D. 3820, however, were small and there was no evidence of the infected leaves dying. When these inoculated plants were allowed to reach maturity in the greenhouse there was considerable difference in the development of black stem. The tolerant varieties from the wild species showed much less black stem than did the common varieties. The same three varieties mentioned above are shown at maturity in Figure 2. The susceptible variety Rodney showed severe black stem and portions of the stems were badly rotted and lodging was complete. There was also considerable black stem on Abegweit and many of the stems had lodged. However, black stem on C. D. 3820 was not severe and all culms were standing erect.

The infection reaction obtained on a group of varieties inoculated with ascospores in the greenhouse indicated that the same pattern of susceptibility and tolerance existed as with field and greenhouse infection with macrospores but the range in reaction was much broader, especially when compared with macrospore infection in the greenhouse. The response of a few of the varieties to ascospore infection is presented in Table 1. Again, there was good correlation between the different tests with the tolerant varieties showing up well. The type of leaf infection obtained from inoculation with ascospores is illustrated in Figure 3. The varieties Rodney and Lanark were quite susceptible but they reacted differently. Lanark showed numerous, small lesions while Rodney had fewer but much larger lesions. Abegweit was intermediate in reaction and 4832-3-1-1 was the most tolerant. These results suggest that the inoculation of test plants with ascospores in the greenhouse might provide a good means of determining resistance among varieties of common oats and it appears to work equally well with wild species (Table 1).

Discussion

Field and greenhouse testing of oat varieties for resistance to Leptosphaeria avenaria f. sp. avenaria showed that varieties derived from the species A. sativa were quite susceptible. A few varieties and strains had some tolerance in the field but when these were tested in the greenhouse with a heavy macrospore inoculum there was little difference in tolerance between them and the more susceptible varieties.

Some varieties derived from some of the wild species of oats such as A. brevis, A. nudibrevis, A. strigosa, and A. wiestii appear to have considerably more resistance than common oats. This was evident not only in the field but in the greenhouse. However, not all wild species nor all varieties within a species contained the necessary gene or genes for

resistance. The resistance expressed by these varieties was not the absence of disease development but rather a much slower disease development. Individual lesions remained small and did not coalesce to the extent that they did on susceptible varieties. As a result the black stem phase was considerably reduced. Only a limited number of varieties from wild species have been tested and it is possible that with more extensive testing, even better resistance will be found. Among the wild species tested, those showing resistance were diploids. Zillinsky and Derick (12) have shown that resistance to crown rust is also present in these species and that there are several means of transferring the genes responsible for crown rust resistance from diploid to hexaploid oats. This suggests that it should be possible to transfer resistance to L. avenaria f. sp. avenaria in the same way.

Field evaluation would appear to be the best method of locating resistance in the common oat varieties since field tolerance is all that can be expected from this group. However, all entries would have to be tested several years as, invariably, some escape infection each year and if tested only once might appear quite tolerant. The availability of field tolerance in commercial varieties would be of considerable importance, especially in reducing lodging and increasing yields. In Eastern Canada strains 4832-3-1-1, 5055-13 and 5055-46 (Russell) appear to have considerable field tolerance and this is further indicated by an increase in yield over the more susceptible commercial varieties. Disease reaction among the wild species can be detected either in the field or greenhouse. However, in the greenhouse a true appraisal of this material can only be obtained by taking disease notes approximately a month after inoculation. Disease development on this material shortly after inoculation was similar to, but not as severe as that of the susceptible varieties. Disease development was, for some reason, slowed down on the resistant material and a month following inoculation the differences in disease reaction were most apparent.

The use of ascospores as inoculum in the greenhouse was studied in a preliminary way. It appeared that the range in reaction was much wider with this inoculum than with macrospores and there was a good correlation in the response to the two types of inoculum. Clark and Zillinsky (1) have shown that ascospores are the major source of primary inoculum and if resistance against this type of infection could be found, disease development in the field would be greatly reduced. A limiting factor in the use of ascospores as inoculum is the inability to produce this spore form on artificial media in the laboratory. Ascospores must be obtained from infected oat straw which has overwintered in the field.

Acknowledgements

The authors with to thank the co-operators at the Research Stations and Universities in Eastern Canada for their assistance and A.A. Scott for preparing the photographs.

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INFLUENCE OF ORCHARD FUNGICIDES ON GLOEOSPORIUM ALBUM ROT AND STORAGE SCAB OF APPLES[1]

R. G. Ross and C. L. Lockhart[2]

Abstract

Excellent control of a storage rot of apples, caused by Gloeosporium album, was obtained with an orchard spraying program of captan in the pre-cover followed by 3 regular and 2 late cover sprays of captan or a mixture of captan and zineb. Dyrene gave good control without late cover sprays. Some control was obtained when captan or the mercurial fungicides, Phelam and Phix, were used in the pre-cover and followed by 3 regular captan cover sprays.

Control of storage scab of apples, caused by Venturia inaequalis, was obtained with captan in the pre-cover followed by 3 regular and 2 late cover sprays of zineb or a mixture of captan and zineb. A regular schedule of dodine without late cover sprays gave excellent control of storage scab.

Introduction

A rot of apples, often referred to as bitter rot or ripe spot, caused by Gloeosporium album Osterw., is an important cause of fruit loss of stored apples in Nova Scotia. In many apple growing areas the anthracnose fungus, Gloeosporium perennans Zeller & Childs, is usually the more common species of Gloeosporium causing storage rots but this organism has not been found in Nova Scotia. Recently in Nova Scotia Eaves et al. (2) found that pre-cover sprays of phenyl mercury acetate or seasonal applications of captan resulted in less rot by G. album than did ferbam, glyodin or sulphur paste. A lime sulphur-colloidal sulphur schedule has been reported (8) to give no control of G. album but thiram or captan continued for 3 cover sprays gave good control. Brooks (1) reported satisfactory control of G. album and G. perennans with phenyl mercury chloride followed by captan cover sprays. Marsh et al. (6) obtained better control with late cover sprays of captan or ziram than with glyodin. Several other workers (3, 4, 5, 7) have reported some control of G. album and G. perennans with late cover sprays of captan.

This paper shows the effect of orchard fungicides on the development in storage of apple rots caused by G. album and some results are given of their effectiveness against storage scab caused by Venturia inaequalis (Cke.) Wint.

[1]Contribution No. 1047 from the Research Station, Canada Agriculture, Kentville, Nova Scotia.

[2]Plant Pathologists.

Materials and Methods

The apples used in this work were from a young orchard located near Kentville, N.S., in which fungicide trials were carried out in 1957 and 1959. In 1957 the orchard was divided into 2 parts, one being used to test apple scab fungicides in a protectant schedule and the other for tests on eradicant or post-infection sprays. Each part was laid out in 2 blocks and the treatments were randomized in each block. The protectant plots received 5 pre-cover (April 29; May 9, 16, 27; June 6-7) and 3 cover (June 17-19, 26-28; July 9-10) sprays. A captan plot in one block received 2 extra cover (July 31, August 29) sprays. The eradicant plots received 3 pre-cover (May 14, 28; June 10) and, except for dodine, the cover sprays were applied as in the protectant schedule. Dodine was applied as an eradicant in the cover sprays on June 19, 28 and July 11.

In 1959 all fungicides were applied in a protectant schedule. The orchard was divided into 2 blocks with the treatments randomized in each block. Except for one dodine treatment, where the sprays were applied about 12 days apart giving a total of 7 applications with the final one on July 20, the plots received 5 pre-cover (May 7-8, 14-15, 21-22; June 1-2, 11-12) and 3 cover (June 22-23; July 2-3, 13-15) sprays. In one block 3 captan plots received extra cover sprays on July 27 and August 17.

The sprays were applied dilute with a hand gun and the trees were sprayed to run-off. The fungicides used were:

Phelam, phenyl mercury dimethyl dithiocarbamate, 3% (F. W. Berk & Co., Ltd., London, England)

Phix, phenyl mercury acetate, 22% (Chemley Products Co., Chicago, Ill.)

Dodine (Cyprex) n-dodecyl guanidine acetate, 65% (Cyanamid of Canada, Ltd., Toronto, Ont.)

Captan (Captan 50-W), N-(trichloromethylthio)-4-cyclohexene-1, 2-dicarboximide, 50% (Stauffer Chemical Co., New York, N.Y.)

Dichlone (Phygon XL), 2, 3-dichloro-1, 4-naphthoquinone, 50% (Naugatuck Chemicals, Elmira, Ont.)

Glyodin (Crag Fruit Fungicide 341), 2-heptadecyl-2-imidazoline acetate, 34% (Green Cross Insecticides, Montreal, Que.)

Dyrene, 2, 4-dichloro-6-(o-chloroanalino) triazine, 50% (Chemagro Corporation, Kansas City, Mo.)

Zineb (Parzate), zinc ethylene bisdithiocarbamate, 50% (Du Pont Co. of Canada, Ltd., Montreal, P.Q.)

In 1957 one bushel of unblemished McIntosh apples from each plot were stored at 32°F and removed for examination after 210 days. In 1959 one

bushel of McIntosh and one of Cortland apples from each plot were stored at 38°F and examined after 215 days. Records were made of the number of fruit showing G. album rot and storage scab. In calculation of the percentage of fruit with rot or scab every affected fruit was included whether it had one infection or many. When spores were not present on the rotted areas for identification of the causal organism, isolations were made on an agar medium.

Results and Discussion

The 1957 results are presented in Table 1 and those from 1959 are in Table 2. Except for the captan plots, where the effect of extra cover sprays was obtained, the data are the average of 2 replicates.

Table 1

Influence of orchard fungicides on G. album rot of McIntosh apples - 1957

Fungicide and rate per 100 gallons		Percent rot
Pre-cover	Cover	
Eradicant Schedule		
Phelam, 1 lb.	Captan, 1 1/2 lb.	3.2
Phix, 1/4 lb.	Captan, 1 1/2 lb.	4.9
Dodine, 1 lb.	Dodine, 1 lb.	7.3
Protectant Schedule		
Captan, 2 lb.	Captan, 1 1/2 lb.*	0.0
Captan, 2 lb.	Captan, 1 1/2 lb.	4.9
Dichlone, 1/2 lb.	Zineb, 2 lb.	7.0
Zineb, 2 lb.	Zineb, 2 lb.	7.5
Dodine, 1 lb.	Dodine, 1 lb.	9.0
Glyodin, 3/4 qt. + Phix, 1/8 lb.	Glyodin, 1 qt.	13.4
Dyrene, 2 lb.	Dyrene, 2 lb.	2.4

* 2 extra cover sprays

Table 2

Influence of orchard fungicides on G. album rot and storage scab of apples - 1959

Fungicide and rate per 100 gallons		Percent rot and scab			
		McIntosh		Cortland	
Pre-cover	Cover	Rot	Scab	Rot	Scab
Captan, 2 lb.	Captan, 1 1/2 lb.*	0.0	9.1	0.0	10.9
Captan, 2 lb.	Captan, 1 1/2 lb.	1.6	71.8	4.7	29.1
Captan, 2 lb.	Zineb, 2 lb.*	3.8	0.0	0.7	0.0
Captan, 2 lb.	Zineb, 2 lb.	4.3	26.7	3.8	11.3
Captan, 2 lb.	Captan, 1 lb.+Zineb, 1 lb.*	0.5	0.0	0.0	0.0
Captan, 2 lb.	Captan, 1 lb.+Zineb, 1 lb.	3.6	93.2	1.5	11.1
Dodine, 3/4 lb.[a]	Dodine, 3/4 lb.[a]	3.1	1.8	0.4	0.0
Dodine, 3/4 lb.	Dodine, 3/4 lb.	5.0	0.0	3.5	2.1
Dodine, 1/2 lb.	Dodine, 1/2 lb.	11.3	24.2	3.8	0.3
Dichlone, 1/2 lb.	Dichlone, 1/4 lb.	6.8	59.5	4.2	24.4
Glyodin, 1 1/2 qt.	Glyodin, 1 qt.	5.1	48.5	6.7	8.4
Glyodin, 1 qt.	Glyodin, 3/4 qt.	10.6	67.7	7.1	14.5

*2 extra cover sprays

[a] Sprays 12 days apart

The results for 1957 show the value of the extra captan cover sprays for controlling G. album fruit rot. This is in agreement with the results of other workers (3, 4, 5, 7). The results with Dyrene are interesting in that it held the rot to a low level without extra applications. Captan without extra cover sprays, or the mercurial fungicides, Phelam and Phix, followed by captan also gave some measure of control. The results with glyodin are similar to those of other workers (2, 6) who found that it was not as effective as captan.

In 1959 (Table 2) the extra captan cover sprays again gave complete control of G. album rot in both apple varieties. Zineb as a cover spray was less effective but the mixture of captan and zineb gave almost complete control. The results with dodine suggest that extra cover sprays at the 3/4 lb. rate might be very effective. There was less rot in the 12-day treatment which received its final cover spray on July 20 than in the regular dodine treatment where the final spray was applied 5-7 days earlier. The results

with glyodin, except at the higher rate on McIntosh, agree with those of 1957.

Storage scab did not show up on the 1957 crop but in 1959 it was possible to compare the effectiveness of the various treatments for the control of this disease. The results in Table 2 show dodine at the 3/4 lb. rate to be very effective. Without extra cover sprays all other treatments were relatively ineffective. Where extra cover sprays were applied zineb and the mixture of captan and zineb gave complete control of storage scab. Without extra cover sprays zineb was much more effective than the mixture of captan and zineb or captan alone.

The mixture of captan and zineb with extra cover sprays in July and August appears to be an excellent spray for the control of both G. album rot and storage scab of apples.

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CONTROL OF GREEN ALGAE ON ENGLISH HOLLY

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Abstract

Green algae seriously mar the appearance of the foliage of English holly in British Columbia and the Pacific Northwest. Six fungicides were tested for algaecidal properties. Nabam plus zinc sulphate spray gave the most effective control.

Introduction

Green algae (Protococcus sp.), when present, cause severe disfigurement to the leaves of English holly (Ilex aquifolium L.) in British Columbia and the Pacific Northwest States. The heavy fall rains and cool, wet winters common in this region are ideal for the development of algae on the surface of leaves, twigs, branches, and main trunks of holly trees. The algae neither penetrate nor distort holly tissues and the condition arising from their presence on holly can in no way be considered a disease. However, the presence of algae renders the holly commercially unacceptable.

Bordeaux mixture (4:4:40 Imperial measure) and tri-basic copper sulphate (3 pounds per 100 gallons Imperial) have in the past been used for the control of algae and of leaf-spotting fungi on holly. However, local observations have indicated that these materials are relatively ineffective for the control of algae and recently (2) tri-basic copper sulphate has been shown to cause red spotting of holly leaves. In addition, Bordeaux mixture cannot be used as a pre-harvest spray because of its objectionable residue. Tri-basic copper sulphate is readily weathered by the fall rains and because of this characteristic it does not give protection for the required length of time. The experiments reported here were initiated to find an algaecide which would give a greater degree of control, reduce the harvest residue problem, and eliminate injury.

Materials and Methods

The orchard used in these experiments was planted in 1925 with 224 trees in 13 rows running east to west, 16 trees per row, with 14 x 14 ft. spacing. The trees are now 15 to 18 feet high. Air circulation within the planting is poor, a condition which has contributed to a relatively uniform growth of algae. Eight trees were used for each treatment. Sprays were applied at 300 pounds pressure with a machine with mechanical agitation. The following materials were applied in the first week of February 1960:

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1. Liquid Parzate (19% active nabam), 2 quarts plus 1 lb. zinc sulphate monohydrate per 100 Imperial gallons water.
2. Tri-basic copper sulphate (52% copper), 3 lb. per 100 Imperial gallons.
3. Parzate (65% active zineb), 2 lb. per 100 Imperial gallons.
4. Maneb (70% active), 2 lb. per 100 Imperial gallons.
5. Lime sulphur, 1 Imperial gallon per 100 Imperial gallons.
6. Parzate (65% active zineb), 2 lb. plus 1 quart Diazinon (25% active) per 100 Imperial gallons.
7. Bordeaux mixture (4:4:40 Imperial measure).
8. Water.

All treatments except 5 and 7 were applied at the rate of 4 Imperial gallons per tree and contained two ounces Dupont spreader-sticker.

Eight leaves were taken at random from each of five trees per treatment to estimate the kill of algae. These estimates, based upon the percentage of plasmolysed or necrotic algal cells, were made independently by two persons to avoid bias. Evaluations were made four weeks, six weeks, and eight weeks after spraying.

A second experiment was set up to determine the tolerance of holly to the nabam-zinc sulphate treatment. Male holly trees in the same orchard were sprayed at 1X, 2X, and 4X the 2 quarts nabam plus 1 pound zinc sulphate per 100 Imperial gallons treatment and examined two days, one month, and three months after spraying for signs of injury and residue.

Results and Discussion

The nabam-zinc sulphate treatment gave the best control of green algae, the kill being 84% eight weeks after application (Table 1). Nabam and zinc sulphate react to form a compound identical to zineb so it might be assumed that zineb should give equivalent control. However, zineb was consistently less effective at all three periods when the leaf samples were examined. The addition of Diazinon to control mites lowered the algaecidal property of zineb. Bordeaux mixture and tri-basic copper sulphate, which in the past have been used extensively for the destruction of algae (1), gave essentially no control and, since red spot of holly leaves has been attributed to copper injury (2), the use of these materials should be discontinued for this purpose. Lime sulphur was an effective algaecide but left an objectionable residue. An objectionable residue was also left by the Bordeaux mixture spray.

Table 1

Effect of fungicides on algae

Fungicide	Percent algal kill		
	4 weeks	6 weeks	8 weeks
Nabam + zinc sulphate	65.3	79.1	84.1
Lime sulphur	65.4	78.8	74.2
Zineb	60.8	71.1	74.2
Zineb + Diazinon	50.9	57.3	54.5
Tri-basic copper sulphate	40.8	46.8	53.9
Maneb	44.5	50.7	50.0
Bordeaux mixture	42.4	48.1	44.2
Check	40.1	38.9	35.2

In the second experiment, doubling the concentration of the nabam-zinc sulphate spray did not cause any discernible injury nor did it leave an objectionable residue.

Tripling the concentration caused leaves to assume a more upright position on the stem and an upward trough-like curling of the leaf margin. A heavy residue was visible immediately following spray application, but in 4 weeks this residue had almost disappeared. Recovery from leaf curling was complete within six weeks.

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THE VALUE OF SEED PROTECTION FOR VEGETABLE CROPS IN EASTERN CANADA[1]

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Abstract

The results of vegetable seed treatment trials with fungicides and fungicide-insecticide combinations for the improvement of emergence and the prevention of damping-off in pea, bean, corn, cucumber and squash seed are summarized. The trials were held at St. Catharines and Ottawa, Ontario and St. Jean, Quebec during the summers of 1957, 1958 and 1959. The significant increases in emergence resulting from treatments containing thiram, captan, and dichlone are proof that vegetable seed treatment is essential in these areas.

For the past three summers, 1957, 1958 and 1959, vegetable seed treatment trials comprising fungicide and fungicide-insecticide combinations have been held at Ottawa and St. Catharines in Ontario and St. Jean, Quebec. Although conditions of soil temperature and moisture differ somewhat in these areas, the conditions at Ottawa and St. Jean are comparable. Year to year observations have shown that the mean soil temperature at Ottawa and St. Jean is approximately two degrees lower in May than at St. Catharines. Although the precipitation at the three locations is approximately the same, snow cover remains for a longer period in the spring at Ottawa and St. Jean.

Over the past years it has been found that stands produced from untreated vegetable seeds were often low in emergence. Because of high soil moisture and cool soil temperatures at planting time, in the areas where the trials were carried on, vegetable seeds germinate slowly and there is a long period of susceptibility to attack by soil-borne organisms.

The tests were made on seed of peas, beans, cucumber, squash and corn. All seed was sound and had a germination rate of over 80 per cent with the exception of corn. Corn seed used in 1957 and 1958 had a germination rate of between 50 and 60 per cent.

Seed was treated in two ounce lots for the small seeded vegetables and up to one pound lots for the larger seeded kinds at the rates indicated in

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[1]Joint contribution from the Plant Research Institute, Ottawa, Ontario (Contribution No. 70), the Plant Pathology Laboratory, St. Catharines, Ontario and the Research Laboratory, St. Jean, Quebec, Research Branch, Canada Department of Agriculture.

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Table 1. All treated seed was stored at 70°F. for two to four weeks before being sown in the field. The seed of each treated lot was replicated four times at each location and sown in rod row plots in randomized blocks. The plot arrangement was identical at each location. Emergence counts were taken when the first true leaves had unfolded or as soon as possible after that time. The counts expressed as percentages are shown in Tables 2, 3 and 4. Results at St. Jean are only available for 1958 and 1959.

For peas each year some of the treatments caused significant increases in emergence at all locations and all treatments were beneficial at Ottawa and St. Jean. Significant increases were recorded in beans only at St. Catharines in 1957, at Ottawa and St. Catharines in 1958, and at Ottawa in 1959. Corn seed treatments produced significant increases all three years at Ottawa, in two years at St. Catharines and one year at St. Jean. Significant increases were recorded each year at all locations for squash and cucumber seed treatments; however, the only treatments producing significant increases in emergence for squash seed at St. Catharines in 1957 and 1958 were compounds containing dichlone or captan.

The addition of an insecticide had been shown to be beneficial in controlling the seed corn maggot in a former trial (1). Although insecticides were incorporated in the trials reported here no evidence of injury by this insect was noted. However, the use of an insecticide is recommended where injury from seed corn maggot is a problem.

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Table 1. Dosages of seed treatment materials per 100 lb. of seed.

Treatments	Dosage oz. per 100 lb. of seed				
	Peas	Beans	Corn	Cucumber	Squash
Arasan SF-M --(75% thiram + 2% methoxychlor)	2	1 1/3	3	3	3
Arasan 75 --(75% thiram)	3	2	5 1/3	3	3
Arasan 75 + lindane (1:1)(37.5% thiram + 12.5% lindane)	4	4	4	6	4
Arasan 75 + aldrin (2:1)(50% thiram + 6.66% aldrin)	3	3	3	4 1/2	3
Arasan 75 + dieldrin (2:1)(50% thiram + 16.66% dieldrin)	3	3	3	4 1/2	3
Captan 75 --(75% captan)	2 1/2	2 1/2	1 1/2	3	2
Captan 75 + aldrin (2:1)(50% captan + 6.66% aldrin)	2 1/2	2 1/2	1 1/2	3	2
Captan 75 + dieldrin (2:1)(50% captan + 16.66% dieldrin)	2 1/2	2 1/2	2	3	2
Delsan AD -- (60% thiram + 12.75% technical dieldrin)	3	3	4 1/2	3	3
Orotho Seed Guard --(50% captan + 16.5% lindane)	3	3	3	3	3
Ortho 75 --(75% captan)	2 1/2	2 1/2	1 1/2	3	3
Phygon XL --(50% dichlone)	2	2	1 1/2	4	4
Phygon XL + lindane (1:1)(25% dichlone + 12.5% lindane)	4	4	3	8	8
Phygon XL + aldrin (2:1)(33.33% dichlone + 6.66% aldrin)	3	3	2 1/4	6	6
Phygon XL + dieldrin (2:1)(33.33% dichlone + 16.66% dieldrin)	3	3	2 1/4	6	6
Thioneb 50 W --(50% polyethylene thiram sulphides)	4	4	3	4	8
Thioneb 50 W + lindane (2:1)(33.33% p.t.s. + 8.33% lindane)	6	6	4 1/2	12	12
Thioneb 50 W + aldrin (4:1)(40.0% p.t.s. + 4.0% aldrin)	5	5	3 2/3	10	10
Thioneb 50 W + dieldrin (4:1)(40.0% p.t.s. + 10% dieldrin)	5	5	3 2/3	10	10

Table 2. Mean percentage field emergence from vegetable seed treated with various seed treatments, based on four replicates of 100 seeds each. (St. Jean)

Treatment	1958					1959				
	Peas	Beans	Corn	Cucumber	Squash	Peas	Beans	Corn	Cucumber	Squash
Control	49.2	85.8	7.2	11.0	41.2	59.8	83.2	85.0	20.2	72.8
Arasan SF-M	83.8[c]	87.2	31.2	36.5	30.0	86.8	84.8	76.8	34.0	81.8
Arasan 75	89.8	86.0	40.8	46.5	46.5	89.5	83.8	65.8	34.5	75.8
Arasan 75 + lindane	86.2	75.2	24.5	37.2	36.8	86.8	84.2	75.8	36.0	80.8
Arasan 75 + aldrin	87.2	80.8	29.5	35.2	37.0	85.8	81.8	72.5	41.0	67.2
Arasan 75 + dieldrin	77.5	86.2	29.8	45.2	34.5	86.2	84.2	69.2	35.0	80.2
Captan 75	91.8	86.2	40.2	45.0	59.5	85.5	80.2	75.2	35.2	70.5
Captan 75 + aldrin	85.0	87.0	34.0	48.5	41.2	81.0	83.5	79.8	38.5	71.0
Captan 75 + dieldrin	74.5	87.2	28.2	47.0	39.8	84.8	78.0	79.2	34.5	68.0
Delsan A.D.	85.5	83.0	46.8	43.8	54.5	80.2	80.5	82.5	43.0	74.8
Ortho Seed Guard	79.0	80.0	25.2	43.2	44.0	74.0	85.2	73.0	40.5	66.5
Ortho 75	94.0	86.2	37.5	44.0	61.0	91.0	88.0	83.5	44.8	79.5
Phygon XL	85.0	81.2	32.0	45.0	62.0	89.5	84.8	78.2	33.0	72.5
Phygon XL + lindane	85.5	84.0	29.8	40.8	59.8	90.8	76.8	69.2	38.2	81.5
Phygon XL + aldrin	83.0	80.8	29.5	39.0	56.5	85.5	88.0	78.8	27.8	79.0
Phygon XL + dieldrin	87.8	81.2	33.0	40.5	54.5	85.5	84.2	86.8	41.2	69.8
Thioneb 50W	93.2	79.8	18.0	44.2	47.0	70.5	84.5	78.8	44.5	79.8
Thioneb 50W + lindane	81.5	81.5	19.8	36.2	37.0	89.5	84.2	75.2	31.0	72.5
Thioneb 50 W + aldrin	76.7	82.0	17.0	38.2	38.0	88.8	83.0	86.0	30.5	68.8
Thioneb 50W + dieldrin	82.2	85.8	21.0	40.5	30.5	86.8	86.0	78.0	35.8	82.0
L.S.D.[a]	12.0	---b	4.5	11.6	11.4	7.4	---b	---b	12.8	8.6

[a] L.S.D. at the 5% level

---b No significant increase between treatments.

c Underlined numbers indicate significant increase in emergence at 5% level.

Table 3. Mean percentage field emergence from vegetable seed treated with various seed treatments, based on four replicates of 100 seeds each. (Ottawa)

	1957					1958					1959				
	Peas	Beans	Corn	Cucumber	Squash	Peas	Beans	Corn	Cucumber	Squash	Peas	Beans	Corn	Cu-cumber	Squash
Control	10.0	82.0	23.0	23.0	77.5	56.0	78.2	4.2	14.8	48.5	29.8	65.0	78.5	23.8	71.5
Arasan SF-M	66.2[c]	79.0	53.0	53.0	92.5	91.2	89.2	27.0	64.2	53.5	81.2	84.5	90.2	72.0	88.2
Arasan 75	72.2	84.8	56.0	56.0	92.5	87.5	88.8	35.0	59.8	62.0	87.8	83.8	93.8	84.5	89.2
Arasan 75 + lindane	60.8	83.8	52.0	52.0	90.2	86.0	80.2	20.5	66.0	62.8	79.2	82.5	90.2	80.0	88.2
Arasan 75 + aldrin	62.8	83.8	51.8	79.2	89.5	82.8	86.8	28.2	63.5	57.2	83.0	82.5	87.2	78.2	87.2
Arasan 75 + dieldrin	60.8	88.0	52.0	75.2	90.2	90.5	78.8	27.2	69.2	50.0	82.2	78.0	92.2	84.0	85.0
Captan 75	69.0	86.2	50.2	80.5	92.0	93.2	87.0	18.0	73.8	57.8	90.5	85.2	90.5	80.5	92.0
Captan 75 + aldrin	50.5	87.5	45.2	77.5	85.0	86.0	85.2	16.5	56.0	56.5	84.2	85.0	88.5	77.2	88.5
Captan 75 + dieldrin	59.0	87.2	44.5	81.0	87.0	84.5	83.2	16.0	59.5	62.5	77.5	85.2	88.5	73.2	92.2
Delsan A.D.	66.2	86.8	55.0	54.0	92.5	94.0	80.8	41.5	70.0	72.5	87.0	86.8	92.5	85.0	95.8
Ortho Seed Guard	58.2	81.0	42.5	42.5	85.8	91.8	83.5	14.8	51.8	55.0	71.5	80.2	90.8	79.8	88.5
Ortho 75	75.5	82.2	50.0	78.8	86.8	88.5	86.0	17.5	63.0	66.5	90.5	87.0	90.2	85.2	84.5
Phygon XL	70.5	85.5	44.8	74.5	95.2	89.2	84.2	15.5	65.5	82.8	83.2	78.2	89.2	76.8	94.2
Phygon XL + lindane	63.5	85.0	44.0	77.8	93.0	88.5	80.5	15.2	64.2	75.8	77.0	79.5	88.0	69.8	93.8
Phygon XL + aldrin	68.8	85.8	40.5	78.8	92.5	88.2	85.8	17.0	60.8	73.8	82.2	82.0	86.8	75.0	95.5
Phygon XL + dieldrin	61.5	86.8	44.2	78.2	94.0	86.2	81.2	18.0	66.2	68.0	80.0	82.0	87.0	80.0	93.2
Thioneb 50 W	62.0	83.8	37.2	37.2	86.8	85.2	88.5	16.0	64.8	50.8	73.5	80.5	92.2	80.0	95.8
Thioneb 50W + lindane	45.5	82.0	41.0	79.5	79.5	79.5	85.5	10.2	57.5	52.2	75.8	80.5	89.2	78.2	93.8
Thioneb 50W + aldrin	51.0	80.2	40.7	78.2	70.2	84.5	79.8	13.5	66.2	55.0	69.8	86.2	86.5	77.2	93.0
Thioneb 50W + dieldrin	50.8	86.8	35.0	81.0	75.0	82.2	84.5	11.2	56.2	58.8	73.2	81.2	89.0	75.8	94.0
L.S.D.[a]	8.4	---b	7.0	8.5	9.5	10.6	6.8	6.7	9.9	8.8	13.7	7.1	6.0	7.8	8.9

a L.S.D. at the 5% level.
---b No significant increase between treatments.
c Underlinded numbers indicate significant increase in emergence at 5% level.

Table 4. Mean Percentage field emergence from vegetable seed treated with various seed treatments, based on four replicates of 100 seeds each. (St. Catharines)

	1957					1958					1959				
	Peas	Beans	Corn	Cucumber	Squash	Peas	Beans	Corn	Cucumber	Squash	Peas	Beans	Corn	Cucumber	Squash
Control	26.2	59.2	16.8	15.2	54.8	53.2	71.5	8.2	34.8	56.0	56.8	78.5	66.5	35.0	56.0
Arasan SF-M	46.8[c]	56.2	38.2	20.8	61.0	75.5	82.2	19.0	46.2	52.2	84.5	79.2	68.0	67.2	78.2
Arasan 75	46.8	48.0	21.2	17.0	67.8	76.5	81.2	28.5	50.5	54.4	89.0	81.5	79.8	67.2	76.0
Arasan 75 + lindane	42.2	44.5	48.2	10.0	62.8	68.0	82.2	22.0	48.8	39.8	81.8	84.0	72.5	64.8	71.8
Arasan 75 + aldrin	54.5	79.8	52.0	27.2	65.0	75.0	81.0	32.0	49.8	34.5	86.5	84.8	73.8	74.5	83.2
Arasan 75 + dieldrin	54.8	82.5	36.5	51.0	62.2	69.2	84.0	26.0	46.0	46.5	79.0	85.8	84.2	69.5	74.2
Captan 75	43.2	50.2	51.0	17.0	77.2	79.0	82.2	29.8	50.2	71.8	86.5	79.5	62.2	77.2	84.5
Captan 75 + aldrin	64.5	76.0	58.8	40.0	80.8	70.0	82.2	26.5	58.8	52.2	86.8	84.2	79.5	68.8	83.8
Captan 75 + dieldrin	61.2	91.5	36.8	59.2	77.0	80.5	81.5	25.0	58.5	60.5	82.0	79.0	79.8	66.2	77.0
Delsan A. D.	47.0	84.2	41.0	46.0	61.8	82.2	78.2	26.0	71.7	66.5	87.5	84.2	79.8	68.5	78.0
Ortho Seed Guard	55.0	59.5	35.8	20.8	67.0	75.5	83.5	17.0	63.0	59.2	92.2	83.2	84.2	70.5	79.0
Ortho 75	39.2	39.5	30.0	20.5	82.8	83.8	86.8	32.0	60.5	77.8	85.8	84.2	79.5	70.5	80.2
Phygon XL	31.2	40.5	43.8	17.5	80.0	75.0	81.0	17.0	50.5	76.0	86.8	80.5	57.8	72.5	81.5
Phygon XL + lindane	52.2	59.5	34.2	13.5	85.8	73.0	76.0	23.8	45.0	63.2	84.0	79.8	86.8	68.8	87.2
Phygon XL + aldrin	47.0	80.0	42.0	29.2	85.5	79.2	78.8	20.2	55.5	69.8	81.2	77.5	72.8	66.2	78.0
Phygon XL dieldrin	62.2	82.5	60.2	54.2	89.8	71.5	82.0	25.5	51.8	71.0	86.5	84.0	73.0	64.5	81.0
Thioneb 50W	30.5	45.0	29.5	18.0	31.0	72.2	70.0	10.8	48.2	41.0	75.2	79.0	77.0	64.0	89.0
Thioneb 50W lindane	34.8	65.8	28.0	6.8	28.8	74.0	75.5	14.2	40.0	49.0	86.2	85.0	79.2	64.0	84.0
Thioneb 50W aldrin	44.5	70.5	43.8	20.5	48.8	65.5	86.5	14.5	51.5	48.5	86.2	80.8	88.0	61.8	73.1
Thioneb 50W dieldrin	41.5	81.8	40.8	32.5	46.8	68.8	76.8	18.5	41.0	38.5	83.5	81.5	68.0	63.0	78.8
L. S. D.[a]	18.0	17.0	21.0	16.2	17.0	15.9	10.4	9.9	16.8	11.6	12.2	---b	---b	10.8	14.7

a L. S. D. at the 5% level.
---b No significant increase between treatments.
c Underlined numbers indicate significant increase in emergence at 5% level.

CURRENT STATUS OF BRAMBLE VIRUSES[1]

Richard Stace-Smith[2]

Introduction

Virus diseases of raspberries and blackberries are a problem wherever these crops are grown. Over the years our knowledge of the bramble virus diseases has slowly accumulated to the point where some thirty diseases have been described and, for approximately half of these, their mode of transmission has been determined. Since some of the diseases have not been studied critically and comparative studies have not been made between diseases described from the various geographical areas, synonymy can not be assigned with any degree of certainty. Also, as latent viruses are now known to be carried by a number of commercial varieties, investigators often unwittingly described the symptom expression of a complex infection rather than that caused by a single virus. In this paper, I shall endeavour to summarize our current knowledge of the bramble viruses and virus diseases and discuss some of the problems that are in need of additional research.

I MODE OF SPREAD OF BRAMBLE VIRUSES

The viruses in the Rosaceae in general are exceedingly difficult to transmit mechanically. In recent years, some of them have been transmitted to herbaceous test hosts but, in these instances, it is difficult to transmit the viruses back to the original host. Such is the case with the viruses that occur in plants belonging to the genus Rubus. Attempts to transmit these viruses mechanically have traditionally failed and it is only within the past few years that a small group of the viruses have been transmitted to herbaceous hosts but attempts to transmit these back to Rubus have not succeeded. Thus some vector is probably involved in the transmission of all of the viruses that are known to attack brambles. Vectors for some of these viruses are known but for others the vector has yet to be determined.

Our knowledge of the mode of spread of the bramble viruses was extended rather slowly. Mosaic and leaf curl were shown to be spread by

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[2]Plant Pathologist.

the raspberry aphids in 1922. As other viruses were distinguished, aphids were usually found to be vectors. In fact, where the mode of spread was unknown, an aphid vector was usually assumed or implied. It is only within the past decade that our concept of the mode of spread of the bramble viruses has been broadened. As a result of recent European work, we now recognize two other methods of spread, namely by leafhoppers and through the soil.

A. Aphid Transmission

In North America, two species of aphids predominate in all regions where raspberries are grown. These are readily separated in the field; one is large with long legs, commonly called the large raspberry aphid, Amphorophora rubi Kalt. The other is small and inconspicuous, commonly called the small raspberry aphid, Aphis rubicola Oestlund. When research on the spread of bramble viruses was started in the early 1920's, raspberry aphids were logical suspects as vectors of the two diseases that were known at the time, mosaic and leaf curl. Early workers succeeded in demonstrating that both diseases were spread by the raspberry aphids.

In Europe, the Rubus aphids are similar to those in North America. Again two species predominate, Amphorophora rubi and Aphis idaei v.d.G. Early attempts to transmit bramble viruses in Europe with these two aphids failed repeatedly and it is only within the past decade that they have been shown to be important agents of spread.

Other aphid species occur on wild and cultivated brambles in both Europe and North America but the evidence suggests that these aphids are of relatively minor importance as vectors. The viruses involved are vector specific, those spread by Amphorophora spp. are not spread by Aphis spp. and vice versa. These vectors are restricted to plants in the genus Rubus. For this reason, the natural host range of the aphid-transmitted bramble viruses is very restricted; none has been found in crops outside of the genus Rubus.

Aphids that are omnivorous feeders do not generally colonize brambles with the result that the many aphid-transmitted viruses that attack a wide variety of crops are not a problem on brambles. The only one of this type known to occur in brambles is cucumber mosaic virus, which was recently isolated from Lloyd George red raspberry in Scotland (25). The evidence suggests, however, that even this virus is not transmitted from raspberry to raspberry within the field but, having been transmitted to raspberry from some other plant, it may be perpetuated in raspberry by propagation of infected plants.

B. Leafhopper Transmission

Leafhoppers are known to be important vectors of many of the stone fruit viruses hence it is reasonable to consider them potential vectors for viruses of Rubus spp. Leafhoppers are particularly suspect as vectors of those viruses that induce a proliferation of the shoots of affected plants. One such virus disease, called Rubus stunt, is characterized by such symptoms and a leafhopper vector has been demonstrated. The disease was known for many years in Europe but it was not until 1953 that workers in the Netherlands proved that the virus was transmitted by one of the bramble leafhoppers, Macropsis fuscula (Zett.) (21). At the time this work was done, rubus stunt was the only leafhopper-borne virus known from Europe. Bramble diseases similar to Rubus stunt have been described from North America but none have been shown to be transmitted by leafhoppers. However, the vector of Rubus stunt has been introduced into North America from Europe. At the present time it is restricted to southwestern British Columbia, where it was first found in 1952 (2).

C. Transmission through the Soil

Until 1956, all bramble viruses whose vector had been determined were shown to be spread by insects. For those viruses whose vector had not been determined, it was usually assumed or implied that future investigation would incriminate an aerial vector. Recently, workers in Scotland demonstrated that an important group of viruses that could not be transmitted by the usual vectors were readily transmitted to healthy plants grown in soil taken from a site of a disease outbreak. The main, and perhaps the only infection court for these viruses was the root system. These soil-borne viruses appear to be widely distributed in England and Scotland but, up to the present, they have not been recorded from brambles in North America or from other regions where brambles are grown.

In the raspberry growing areas of eastern Scotland, the most serious virus disease is raspberry leaf-curl. In 1956, Cadman (14) mechanically transmitted a virus from leaf-curl diseased plants to a series of herbaceous test plants. He also transmitted viruses from sugar beet seedlings and weeds growing in the soil where diseased raspberry plants were found. The possible relationships between the viruses isolated from raspberries, sugar beets, and weeds was not known at the time. By means of serological and plant protection tests, these isolates were found to include three distinct viruses, all of which were shown to be transmitted through the soil and to affect brambles. All were thought at first to be undescribed and were given the names raspberry ringspot, beet ringspot, and raspberry yellow-dwarf. Beet ringspot has subsequently been shown to

be a strain of tomato black ring and raspberry yellow-dwarf has been identified with Arabis mosaic.

The soil-borne nature of the viruses was demonstrated by growing raspberries and other test plants in soil collected from fields where leaf curl disease occurred naturally. Attempts to render autoclaved soil infective by adding purified virus preparations did not succeed, suggesting that some other factor, presumably a soil-inhabiting organism, is necessary for transmission. Recently, independent experiments in England and Scotland showed that a nematode of the genus Xiphinema behaves as a vector of Arabis mosaic virus (28, 31). The method of spread of the other two viruses has yet to be determined.

II BRIEF SYNOPSIS OF BRAMBLE VIRUSES

A. Aphid-borne

(1) Viruses transmitted by the genus Aphis

Leaf curl (Bennett, 1930)

Leaf curl is restricted to the raspberry growing areas of North America and is more of a problem in the east than in the west. The disease is prevalent in red, purple, and black raspberries, and occurs to a lesser extent on blackberry, dewberry, and wineberry. The characteristic symptoms are curled leaves that are darker green than normal and stunted plants. The virus is transmitted by Aphis rubicola. Two strains are recognized; one affects red raspberry but not black raspberry, the other affects both red and black raspberry.

Raspberry vein chlorosis (Cadman, 1952)

This virus has been described from red raspberry in Scotland by Cadman (2). Characteristic symptom is clearing of the tissue bordering the small leaf veins, the clearing being localized in small, coalesced patches. Cadman demonstrated that the vector in Europe was Aphis idaei. In British Columbia most stocks of Lloyd George are infected but there is no evidence of spread to other varieties. Presumably infected stocks were imported from Europe and the virus is merely perpetuated by the propagation of this original stock.

(2) Viruses transmitted by the genus Amphorophora

(a) Mottle viruses; heat labile

Red raspberry mosaic (Bennett, 1932)

The term "red raspberry mosaic" was used by Bennett to apply to a group of symptoms rather than a specific virus, since several viruses or virus strains were considered to be involved. The viruses were symptomless or mild on red raspberry and induced a graduated range of symptoms varying from mild to severe mottling and necrosis in black raspberry. A similar group of viruses has been isolated from red raspberry in British Columbia, all of which are transmitted by A. rubi .

Leaf mottle (Cadman, 1951)

Leaf mottle was the name applied to a virus isolated from European raspberry varieties by Cadman (8). The virus was symptomless or mild in red raspberry but caused necrosis in Rubus henryi and mosaic in R. saxitalis and R. occidentalis. The vector is A. rubi.

Black raspberry necrosis (Stace-Smith, 1955)

This name was applied to a virus in British Columbia that was symptomless or mild in red raspberry but caused severe necrosis in black raspberry. The virus was identified with a portion of the red raspberry mosaic complex described by Bennett (5). The vector is A. rubi.

Raspberry leaf spot (Cadman, 1952)

Cadman applied the name leaf spot to a virus in red raspberry in Scotland. Most varieties carry it without symptoms but a few develop chlorotic angular spots which frequently coalesce, distorting the lamina. The virus is transmitted by A. rubi.

Thimbleberry ringspot (Stace-Smith, 1958)

This virus occurs in British Columbia on the native thimbleberry, Rubus parviflorus. The evidence suggests that it is restricted to this host. The virus is heat labile. It is transmitted by three aphids that colonize thimbleberry, Amphorophora parviflori, Masonaphis dividsoni, and M. maxima.

(b) Yellows viruses; heat stable.

Yellow mosaic (Bennett, 1927)

Yellow mosaic was described as a field disease of black raspberry by Bennett (3) in 1927, and later (5) the same name was applied to a similar disease in red raspberry. Yellow leaves and stunted plants are the symptoms in both hosts. Yellow mosaic is not well characterized in the literature, presumably because most varieties that were grown at the time the disease was studied were infected with viruses of the red raspberry mosaic group, and these viruses influenced the expression of other viruses.

Raspberry yellows (Cadman, 1952)

Raspberry yellows is known to affect only red raspberry varieties (Cadman, 1952). The symptoms are most conspicuous early in the spring, when leaves of affected plants show a vivid yellowing and bronzing of the interveinal areas. Later in the season the chlorosis is less intense and frequently forms a watermark or oak-leaf pattern. The causal virus is heat stable and is transmitted by the aphid A. rubi (Cadman, personal communication).

Rubus yellow-net (Stace-Smith, 1955)

This name was applied to a virus in British Columbia that causes a net-like chlorosis of the foliage of Himalaya blackberry, red raspberry, and black raspberry. The vector is A. rubi. This virus, when combined with black raspberry necrosis virus, induces the mosaic disease in red raspberry (42). A virus corresponding to rubus yellow-net has not been reported from Europe. However, a disease in Europe known as veinbanding is considered similar to mosaic in North America. Preliminary testing in 1958 indicated that a virus comparable to rubus yellow-net is present in veinbanding diseased plants in Scotland (Cadman and Stace-Smith, unpublished).

B. Leafhopper-Transmitted

Rubus stunt (Prentice, 1950)

Rubus stunt was described by Prentice (37) as a virus disease of loganberry, blackberry, and raspberry in southern England. The virus mostly affects blackberries in England, but in the Netherlands a virus that

is thought to be the same is a serious problem in red raspberries. The leafhopper, Macropsis fuscula, is the vector in the Netherlands; in England a vector has not yet been determined. Infection in all hosts resulted in the production of numerous, weak canes, giving the plant a bushy appearance. Some infected plants developed abnormal flowers whose floral parts were modified into leaves. Thung (45) reports inactivation of the virus in shoots held at an air temperature of 46°C for two hours.

C. Soil-borne

Raspberry ringspot (Cadman, 1956)

Raspberry ringspot is one of the viruses responsible for the leaf curl disease of raspberries in Scotland. A strain of the virus has also been isolated from raspberries in southern England. The symptoms vary considerably depending upon the variety affected. Some varieties, such as Norfolk Giant, show a severe leaf curling, others show only a ringspot marking of the foliage and a few, such as Lloyd George, appear to be immune. The virus is distinguished from other soil-borne viruses affecting raspberry by the symptoms induced on a series of herbaceous test plants and by serological and plant protection tests.

Tomato black ring (Smith, 1946)

In 1957, Harrison (23) described a soil-borne virus that occurred naturally in several crops and weeds in Scotland. The virus named "beet ring spot" was later shown to be a strain of tomato black ring (24). Raspberry was at first thought to be immune but recently the virus has been isolated from some raspberry varieties (27). Symptoms induced by this virus in susceptible varieties of raspberries differ very little from those caused by raspberry ringspot virus. Diagnosis depends upon the symptoms induced on herbaceous hosts, and upon serological and plant protection tests.

Arabis mosaic (Smith, 1944)

A soil-borne virus of raspberry, strawberry, blackberry and several weed species was described by Harrison (26) in 1958 and provisionally assigned the name "raspberry yellow dwarf". Recent serological and plant protection tests have shown that the virus is the same as Arabis mosaic (28). The virus seems to be widespread in England and rare in Scotland. In raspberry, the virus has been isolated from the variety Malling Exploit, where the characteristic symptoms are vein yellowing and stunting. It was also isolated from a Himalaya blackberry plant showing a yellow mosaic pattern in its leaves.

D. Vector Undetermined

Black raspberry streak (Wilcox, 1922)

This disease of black raspberry was described in 1922 (47) but the virus nature was not determined until 1948 (29). The disease is recognized by the discolored streaks that develop on the canes. The intensity of the streaking varies considerably, which suggests the existance of strains. An insect vector has usually been assumed but no vector has been demonstrated.

Blackberry dwarf (Zeller, 1927)

In 1927, Zeller (50) described a disease of Phenomenal berry and loganberry in Oregon which he called blackberry dwarf. Infected plants had thin canes with short internodes and an increased number of buds at each node. Leaflets were small, distorted, and mottled. A natural vector was not determined, although one of the rose aphids, Capitophorus tetrarhodus, was able to transmit the disease under caged conditions.

Yellow-blotch curl (Chamberlain, 1938)

This virus disease was described from Ontario, Canada, in 1938 (18). Diseased plants showed reduced vigour with the leaves curled down and blotched.

Necrotic fern-leaf mosaic (Chamberlain, 1941)

This virus disease was described from Ontario, Canada, in 1941 (19). Symptoms were mottled, necrotic leaves, retarded foliation, and general stunting.

Raspberry decline (Zeller & Braun, 1943)

Decline is a disease of red raspberry in Oregon (51). Diagnosis depended upon field observations, where infected plants progressively deteriorated over a maximum of about 3 years. A virus was thought to be responsible for this deterioration.

Blackberry variegation (Horn, 1948)

Blackberry variegation occurred in a single wild blackberry plant in Maryland (29). The virus was transmitted by grafting to blackberry and black raspberry. In both hosts, varying degrees of chlorosis developed on the leaves of affected plants.

Loganberry dwarf (Wilhelm et al., 1948)

This disease primarily affects loganberry and related hybrids in California (48). Diseased plants have dwarfed canes, weak fruiting laterals, and precociously developed basal buds, giving the plants a bunchy appearance late in the season.

Curly dwarf (Prentice & Harris, 1950)

Curly dwarf was described in 1950 (38), occurring as a latent virus in Lloyd George raspberry in England. The virus was detected by grafting to the indicator variety Baumforth B, in which leaf curling and dwarfing were induced.

Ringspot (Vaughan et al., 1951)

Ringspot is a virus disease of red raspberry in Washington and Oregon (46). Infected plants show ringspot markings of the foliage. There is no indication that this virus is related to the soil-borne ringspot viruses of Scotland.

Bushy dwarf (Cadman & Harris, 1951)

Bushy dwarf was the name applied to a disease of Lloyd George raspberry in Britain (7). Symptoms are dwarfing of the canes and down-curling and chlorosis of the leaves.

Raspberry yellow-blotch (Cadman & Harris, 1952)

This name was applied to a disease of Lloyd George raspberry in Scotland in 1952 (16). Affected plants showed a coarse yellow blotching of the lower leaves of the young canes and necrosis of the fruiting canes. The causal virus was thought to be transmitted by Amphorophora rubi but it is now considered doubtful that this aphid is in fact a vector (Cadman, personal communication).

Blackberry mosaic (Alcorn et al., 1955)

This disease of Himalaya blackberry was described from California in 1955 (1). Infected plants showed a marked reduction in size and number of leaflets, the length and diameter of the canes. Leaves exhibited a variety of mosaic symptoms. The authors suspected that at least two component viruses were involved.

Loganberry degeneration (Legg, in press)

Loganberry degeneration is the name proposed by Legg (35) for a virus detected in weak loganberry plants in England. It causes symptoms in R. henryi but not in other indicator hosts tested.

III VIRUS-VECTOR RELATIONSHIPS

Aphid-transmitted viruses may be usefully divided into three groups: nonpersistent, semipersistent, and persistent, but the modern concept is that the virus-vector relationships form a continum (44). The nonpersistent viruses can be acquired and transmitted in a few seconds; the semipersistent and persistent viruses require minutes or hours to acquire and transmit. The nonpersistent viruses are rapidly lost by the aphids, usually within minutes; the semipersistent viruses are retained for hours instead of minutes, and the persistent viruses are retained for days or weeks. In order to determine vector relations, virus, vector, and test plant have to be combined so that a reasonable degree of transmission can be achieved. For many of the aphid-transmitted bramble viruses this combination has not been determined with the result that their virus-vector relations are not known.

In North America, the vector relations of raspberry leaf curl, transmitted by Aphis rubicola, is not well understood. Bennett (3) reported transmission experiments which showed that the virus was acquired by the aphids within two hours and that aphids remained viruliferous for a considerable time, possibly for the life of the aphid. In determining the retention of the virus, aphids were held for varying periods on a raspberry variety that was thought to be immune to the virus. The possibility exists that some multiplication occurred in the apparently immune host and that the aphids lost their virus in a relatively short time and then acquired a new charge of virus. Thus, although the variety may have been immune, further work should be done before drawing conclusions on the vector relations of the virus.

The vector relations of the other virus known to be transmitted by aphids belonging to the genus Aphis, raspberry vein chlorosis, is also not well known, mainly because red raspberry, which has been used as a test host, is difficult to infect. In Scotland this virus was shown to be transmitted by Aphis idaei but no information was obtained about its vector relations.

For those viruses transmitted by Amphorophora rubi, the evidence suggests that most, and possibly all, are semipersistent. The mild or latent viruses have approximately the same vector relations. One that has been studied in detail, black raspberry necrosis virus, is acquired by the aphid within 30 minutes, inoculated within 2 minutes, and persists in the

feeding aphid for only a few hours. When the aphids are starved, they can retain the virus for at least one day at 20°C and at least 4 days at 3°C. Fasting before acquisition has no effect on the length of the acquisition feed required. The vector relations of the yellows type viruses do not differ appreciably from the latent viruses. Yellow mosaic is acquired within 2 hours and retained by some aphids for more than 12 hours but less than 24 hours (5). Vector relations of raspberry yellows are not known. Yellow-net is acquired within 1 hour, transmitted within 15 minutes, and usually persists less than 4 hours. Starved aphids may retain the virus for 1 day when held at 20°C. and 4 days when held at 3°C. The following table summarizes the vector relationships and other features of a heat labile and heat stable virus transmitted by Amphorophora rubi.

Table 1

CHARACTERISTICS OF TWO APHID-TRANSMITTED VIRUSES

Property	Black raspberry necrosis virus	Rubus yellow-net virus
Heat tolerance	labile	stable
Tissue affected	mesophyll	phloem
Incubation time	1 week	3 weeks
Vector relationships		
Acquisition feeding time	<30 minutes	<1 hour
Effect of preliminary fasting	none	none
Latent period	none	none
Inoculation time	2 minutes	<15 minutes
Retention-feeding	1-2 hours	1-2 hours
fasting (at 20°C)	1 day	1 day
(at 3°C)	4 days	4 days

The bramble viruses that are transmitted by Amphorophora rubi agree in most essential details with the characteristics of the semipersistent viruses as outlined by Sylvester (44). The only difference is in the retention of the virus while the aphid is fasting. With the bramble viruses the virus is retained considerably longer in the fasting aphid than in the feeding aphid

whereas with beet yellows, the semipersistent virus investigated by Sylvester, the reverse was true.

One problem in the study of bramble virus diseases is that the viruses transmitted by Amphorophora rubi have approximately the same vector relations, with the result that virus complexes are not readily separated. Many field diseases are caused by the combined effect of two or more unrelated viruses and mass transfer of aphids to test plants do not result in a separation of the component viruses. An example of this is raspberry mosaic, which in British Columbia was shown to be caused by the combined effect of black raspberry necrosis virus and rubus yellow-net virus. When several aphids are transferred from a mosaic diseased plant to a black raspberry indicator plant, the two viruses are simultaneously transmitted and the symptoms of black raspberry necrosis virus, which develop in 5 to 7 days, completely obliterate the symptoms of rubus yellow-net, which require about three weeks to develop. However, when single aphids are transferred to a series of test plants the two viruses may be separated by chance.

The vector relations of the leafhopper transmitted virus, Rubus stunt, have been investigated by de Fluiter and van der Meer (22). Preliminary work indicates that the virus has a latent period of more than 8 days and persists in the vector for more than a month.

IV PROBLEMS IN VECTOR IDENTIFICATION

A knowledge of the biology of the vectors is essential in devising sound control procedures yet our understanding of the vectors of the bramble virus diseases is deficient in many respects. In fact, for about one half of the diseases that have been described no vector has been determined. Admittedly some of these diseases are of local significance and minor economic importance but others, such as black raspberry streak, are of major concern. Thus one of the greatest needs is for the discovery of the vector of those viruses whose mode of spread is unknown, not only from the control aspect but also as an aid in the identity of the causal virus and its relationship to other known bramble viruses.

Much remains to be learned about the taxonomy and biology of the aphids that are known to be important as vectors. Most vector studies have been undertaken by plant pathologists so that purely entomological aspects have been neglected. A weakness of much early work lies in the doubtful identity of the aphids tested. For example, in early tests the large raspberry aphid was not distinguished from the small raspberry aphid with the result that Aphis rubicola was for several years erroneously considered to be the vector of mosaic. We now recognize the prime importance of accurate identification but taxonomic studies on the Rubus-inhabiting aphids must

proceed further before definite determinations are possible.

Speciation in the bramble aphids appears to have proceeded further in North America than in Europe, so that identification is more of a problem here than in Europe. Twelve species of bramble aphids of the genus Amphorophora have been described in North America whereas in Europe only one is recognized. Some of the North American species are now placed in Masonaphis, and the validity of others was recently questioned by MacGillivray (36). According to the literature, A. rubi occurs on many wild and cultivated species of Rubus. Evidence from British Columbia suggests that this is not so. Amphorophora rubi is almost entirely restricted to cultivated forms of raspberry and blackberry, but other aphids, morphologically similar, occur on wild Rubus species. One in British Columbia, Amphorophora rubitoxica, occurs on black raspberry, loganberry, and the wild trailing blackberry but will not colonize red raspberry. Another, Amphorophora parviflori, is restricted to the wild thimbleberry. Neither of these aphids is able to transmit the common raspberry viruses.

In the genus Aphis, two species are recognized in North America, rubicola and rubifolii. These two species are morphologically so similar that the diagnostic character used to separate them is the number of antennal segments; rubicola has 6 while rubifolii has 5. Some authors consider that rubicola is the raspberry aphid and does not occur on blackberry, whereas rubifolii occurs on blackberry but not on raspberry. Other authors, however, report collections of both species on red raspberry. This contradictory evidence may be explained by unreliable morphological characteristics used to separate the two species. According to Hille Ris Lambers (personal communication) a separation of these two species based upon the number of antennal segments is not reliable.

A more detailed study of the taxonomy, host-range, and vector potential of the various bramble aphids is essential. Our progress has been hindered in the past because plant pathologists and entomologists have worked in isolation. Surely a teamwork approach is called for.

V TRENDS IN CONTROL OF BRAMBLE VIRUSES

The development of sound measures to control the spread of bramble viruses depends upon an understanding of the mode of transmission and the biology of the vector. Particular attention has been paid to controlling raspberry mosaic, probably the most widespread and destructive of the bramble virus diseases. Certification programs have been established in most raspberry growing regions to provide propagators with relatively clean stock. These schemes have been only partially effective, however, and indiscriminate propagation of planting stock is still commonly practiced. More lasting control involves breeding varieties resistant to the aphid or to infection by

the aphid. This is generally recognized as the most satisfactory method of control.

In North America, the variety Herbert was used as a parent in raspberry breeding as early as 1922, since this variety had an obviously low incidence of mosaic in commercial plantings. It was later demonstrated by Winter (49) that the vector, Amphorophora rubi, did not feed or reproduce readily on this variety. Similarily, Lloyd George has remained relatively free of mosaic since its introduction into North America from Europe in 1926, a characteristic that has made this variety a favored parent in raspberry breeding. Schwartze and Huber (39) demonstrated that Lloyd George was resistant to Amphorophora rubi and that the factors controlling resistance were carried by 2 or more genes.

In Britain, workers at East Malling have recently undertaken a thorough study of the genetics of resistance to Amphorophora rubi in the raspberry. Resistance in the variety Baumforth A was shown to be controlled by a single dominant gene, designated A_1 (33). The American variety Chief was shown to carry six dominant genes for resistance, which were designated A_2 to A_7 (34). Work presently in progress has shown that the American black raspberry R. occidentalis L. provides a valuable source of resistance and that a number of other sources are available (Knight, personal communication). Thus in Britain there is every indication that genes for aphid resistance can be incorporated into new varieties.

The Lloyd George variety has remained relatively free from mosaic in North America whereas the same variety is very susceptible to mosaic in Europe. At first this led to speculation that the viruses involved in the disease in North America were distinct from those in Europe. However, it was noted that the aphid vector refused to breed on Lloyd George in North America although in Britain large populations were found on this variety; hence it was concluded that two strains of the aphid exist, one in North America and one in Europe. These were the only strains of Amphorophora rubi known on raspberry until 1958 when, as an important by-product of the raspberry breeding project in England, three strains were discovered (6). More recently, a fourth strain has been discovered in England. Sources of resistance are known against all of these strains. (Briggs, personal communication).

Obviously the success of a control program based upon varieties resistant to the vector depends upon the stability of the vector. If A. rubi is capable of mutating and developing new strains that would attack varieties that were resistant when released, a control program based on breeding for resistance would be of little lasting value. Data available suggests that the aphid is stable and that resistance will withstand the test of time. The best example is the Lloyd George variety which has been grown in the Pacific Northwest for over thirty years and has remained resistant to Amphorophora rubi.

Resistance of raspberry varieties to infection with viruses carried by Amphorophora rubi is not dependent solely on resistance to the vector. In addition, a true resistance to infection is recognized, although the nature of this resistance is not understood. In Scotland, Cadman and Fiskin (17) found that raspberry varieties differed greatly in susceptibility to infection by aphid-transmitted viruses and that the differences were not correlated with aphid resistance. In North America most varieties that are reported to escape mosaic infection are either fully immune or partially resistant to the aphid vector. A variety such as Washington, however, supports a moderate aphid population and yet plants of this variety are rarely infected in the field and are difficult to infect in the greenhouse where viruliferous aphids are transferred directly from the diseased source plants. The possibility of utilizing resistance to infection as opposed to resistance to the vector is being explored at East Malling by Knight and Keep (32).

Limited evidence suggests that the genes for resistance to the American strain of Amphorophora rubi have their origin in the European red raspberry, Rubus idaeus, whereas resistance to the European strains have their origin in the American red raspberry, Rubus idaeus sub-species strigosus. In North America, all varieties that have been reported to be resistant are of European origin while in Europe resistant varieties are derived in part at least from the American red raspberry.

Another technique that may serve a useful function in controlling aphid-transmitted viruses is heat therapy. Chambers (20) has reported inactivation of latent viruses in Scotland by holding raspberry plants from 1 to 4 weeks at an air temperature of 32°C. Similar results have been obtained in British Columbia. Black raspberry necrosis virus and other latent viruses in red raspberry plants have been successfully eliminated from plants held at an air temperature of 37°C for periods ranging from 5 to 10 days. Two other viruses, rubus yellow-net and raspberry vein chlorosis were not eliminated from plants so treated for periods up to 3 months. This limited evidence suggests that bramble viruses may be usefully divided into two groups, the heat labile and the heat stable, depending upon their ability to withstand heat treatment. This technique is particularly valuable for virus identification and for obtaining a virus-free clone of some of the older varieties that have become universally infected with latent viruses.

Breeding for resistance has not been attempted to control the leafhopper-borne virus disease, Rubus stunt. In the Netherlands, however, satisfactory control has been achieved with insecticides. In fact, the rapid increase in the incidence of Rubus stunt beginning about 1945 has been correlated with a change in spraying materials. Prior to 1945, raspberries were sprayed with dormant tar oils to control a variety of insects and this material must have destroyed the eggs of the vector. Subsequently DDT replaced tar oils and, although it controlled other pests, it had little effect

on M. fuscula with the result that high populations developed and the virus spread proportionately. Spray programs are now directed against the leafhopper and this has resulted in a marked reduction in the incidence of Rubus stunt (22).

Too little is known about the distribution and means of spread of the soil-borne viruses but they pose difficult problems not encountered in the control of viruses with aerial vectors. Rotating crops and propagating virus-free stock under isolated conditions are not effective since the viruses are carried in a wide variety of crop and weed hosts. The most promising measure involves planting varieties that appear to be immune. Lloyd George, for instance, is immune from raspberry ringspot and tomato black ring but susceptible to Arabis mosaic. Malling Jewel is immune from Arabis mosaic but susceptible to the other two. Sources of resistance are thus known and it is probable that the genes for resistance could be combined in breeding resistant plants.

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EVIDENCE OF SOIL-BORNE MOSAIC OF WHEAT IN ONTARIO

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Light green to yellow mosaic, including spots and indistinct streaks was first observed on winter wheat in Ontario in June, 1957 in several fields in southern Kent County. That same year the disease was again observed in one field in each of the following counties: Bruce, Huron, Oxford and Perth.

A survey carried out in May and early June of 1960 in southwestern Ontario revealed that the condition was more common and widespread than previously believed. Wheat showing the mosaic symptoms was found in 19 of 43 fields examined in Essex, Kent, Huron, Wellington, Peel, York and Simcoe counties.

Usually all the leaves of affected plants showed the symptoms, but the plants were not noticeably stunted. In some fields only a few affected plants were found and diseased plants usually occurred in patches. Mosaic symptoms appeared on nearly 100 percent of the plants in 6 fields.

When the condition was first observed, tests were made to determine whether or not the cause of the disease was soil-borne. Winter wheat of the varieties Cornell, Richmond, Genesee, Harvest Queen, Michigan Amber and Red Winter Spelt was planted in boxes of soil collected from a field in which the mosaic symptoms had appeared. Similiar seedings were made in greenhouse potting soil.

No symptoms developed in wheat grown at temperatures ranging from 50-75°F. In a second test wheat was sown on September 29th and the plants left outside during the winter. On March 15th they were moved into a cool (60-65°F) greenhouse. As the plants grew mosaic symptoms similiar to those observed in the field developed in all varieties grown in soil from the affected field.

Attempts to transmit a virus by rubbing sap from diseased plants onto healthy wheat have failed.

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EPICOCCUM NIGRUM ON LEAVES OF WINTER WHEAT

W. E. Sackston[1]

On June 23 and 24, while visiting the Research Station at Harrow, Ont., Dr. D. J. Samborski and I examined several fields and some experimental plots of winter wheat, looking for rust. We found very light infections of leaf rust, but no stem rust.

Leaf lesions, apparently caused by Septoria infection, were numerous, especially on the lower leaves. Some of the lesions bore rows of black bodies, which under a hand lens appeared to be small and somewhat atypically grouped pycnidia.

When I examined the lesioned leaves critically at Winnipeg, I found that the "black bodies" were not pycnidia, but sporodochia of Epicoccum nigrum Lk. ex Wallr. Numerous typical, dark, muriform conidia were present.

Although E. nigrum does not seem to have been reported previously from wheat leaves in Canada, it is frequently isolated from seeds and roots of various crops at the Winnipeg Laboratory. Freeman Weiss reported Epicoccum from Triticum as follows: "Epicoccum nigrum Lk. ex Wallr., glume spot, smudge, (saprophytic). Del., Ill., Ohio, Pa. Various other names as E. purpurascens Ehr., E. vulgare Cda. are probably synonyms."[2]

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IDENTITY OF AN INCITANT OF ANTHRACNOSE IN SWEET PEPPERS

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The recent finding (2) that the bulk of anthracnose in tomato fruit is caused by the ubiquitous, sclerotium-forming organism widely known as Colletotrichum atramentarium (Berk. & Br.) Taubenh. raises the question of the possibility of similar etiology in diseases of related fruits. Since Vermicularia atramentaria Berk. & Br. (1850) is antedated by Wallroth's Chaetomium coccodes of 1833, we must accept Hughes' combination (1), Colletotrichum coccodes (Wallr.) Hughes, as the legitimate name for this fungus under the rules of nomenclature.

Dr. C.D. McKeen kindly sent two samples of several cultures which he isolated from anthracnose lesions of field-grown sweet pepper fruits in Essex County, Ontario, in September, 1959. The two cultures which were received proved to be morphologically and culturally similar to isolates from tomato anthracnose and potato dartrose. Spores from both, under moist incubation, have proved capable of causing infection in intact, greenhouse-grown, green tomato fruit. These fruit, upon ripening, developed lesions typical of C. coccodes and yielded isolates of this fungus similar to those providing the conidial inoculum.

It would appear that, under suitable field conditions, the tomato anthracnose fungus, C. coccodes, is capable of inciting anthracnose in sweet pepper fruits as well.

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Dr. Waldermar E. Sackston, who has since 1958, been Head, Plant Pathology Section, Canada Department of Agriculture Research Station, Winnipeg, Manitoba, is leaving the Department on September 12th after 19 years of service to accept the post of Professor of Plant Pathology at Macdonald College, McGill University. Dr. Sackston's research with the Department has been focused on the diseases of oil-seed crops, and he has gained an international reputation as a specialist in the diseases of sunflowers and flax.

Dr. Harold A. Senn, Director, Plant Research Institute, Ottawa left the Canada Department of Agriculture on August 31st., after 22 years service, to join the staff of the University of Wisconsin, Madison, Wisconsin, as Director of the Biotron and Professor of Botany. Dr. Senn is recognized internationally as an authority on the design and operation of plant growth rooms and greenhouses in which environmental conditions are precisely controlled.

Dr. Jens J. Nielson, Goettingen, Germany, has accepted a post-doctorate fellowship to work with Drs. R. Rohringer and D.J. Samborski at the Canada Department of Agriculture Research Station, Winnipeg, Manitoba, on the obligate parasitism of the rust fungi. Dr. Nielson recently received his Ph.D. degree at the University of Goettingen for his studies on the obligate parasitism of Peronospora brassicae in cabbage under Prof. Dr. W. H. Fuchs, Director of the Institut für Pflanzenpathologie.

Dr. Gregoire L. Hennebert, Louvain, Belgium, has also accepted a post-doctorate fellowship to work at the Plant Research Institute, Ottawa. He will study, with Dr. S.J. Hughes, the taxonomy of the Hyphomycetes, particularly of genera related to Botrytis and their host specificity. He was awarded the D.Sc. degree by the Catholic University of Louvain this year. His thesis concerned a revision of the genus Botrytis and was completed at the Laboratory of Plant Pathology under Prof. Dr. V. Estienne and Prof. Dr. P. Staner.

Dr. Ilkka Kukonnen, University of Turku, Finland, returned to Finland at the end of August after working 13 months with Dr. D.B.O. Savile at the Plant Research Institute, Ottawa, on a post-doctorate fellowship. Dr. Kukkhonen is engaged in a taxonomic and experimental study of the smut genus Cintractia. In addition to working through the abundant material in

the herbarium at Ottawa he has done extensive field work near Ottawa, in the arctic and sub-arctic, and in the Yukon and northern British Columbia.

Dr. John T. Slykhuis, Head, Plant Pathology Section, Plant Research Institute, Ottawa, has accepted the invitation of the Australian government to make observations on cereal virus diseases in that country for a two and a half month period. During his absence from Ottawa he will also visit Japan, the Phillipines, New Zealand, India, West Pakistan, Iraq, Jordan and Egypt.