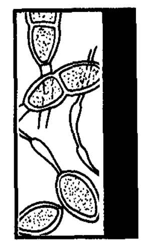
VOL.45, No.3, SEPTEMBER, 1965

CANADIAN PLANT DISEASE SURVEY



EDITOR: D.W. CREELMAN

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

VOL.45, No.3, SEPTEMBER, 1965

CANADIAN PLANT DISEASE SURVEY

RESEARCH BRANCH CANADA DEPARTMENT OF AGRICULTURE

CONTENTS

"The Canadian Plant Disease Survey is a periodical of information and record on the occurrence and severity of plant diseases in Canada. It will also accept other original information such as the development of methods of investigation and control, including the evaluation of new materials. Review papers and compilations of practical value to phytopathologists will be included from time to time. It will not accept results of original research suitable for publication in more formal scientific journals".

DETECTION OF VIRUS INFECTION IN IMPORTED CHERRY AND APRICOT CLONES[1]

Maurice F. Welsh[2] *and James May*[3]

Abstract

Apricot and cherry clones originating in European and Asian countries were indexed on the cherry, apricot, prune and peach varieties grown commercially in British Columbia. Five unfamiliar syndromes were induced on sweet cherry varieties, and gumming reactions were given by apricot and peach. Several alternative bases are suggested for the selection of indicators to serve in the post entry indexing of stone fruits, and the relative merits of such indexing host ranges are reviewed.

Introduction

The occurrence of viruses in deciduous fruit trees and scionwood, imported to North America from other continents, has been reported by Kahn et al. (5), Milbrath (6), and Welsh and Keane (11), among others.

In 1960 and 1961 the requirements of the tree fruit breeding program at the Canada Agriculture Research Station, Summerland, justified the importation of cherry and apricot varieties originating in a number of countries in Europe and Asia. Many of these were available from the Plant Introduction Station at Chico, California. Others were imported directly from research institutions on the other continents. Some of the cherry varieties at the Chico station had been partially indexed by Milbrath (6). No information was available on the virus status of the other materials to indicate whether their introduction to Research Station plantings was a safe procedure. No established quarantine and indexing facilities existed in Canada for their reception. Accordingly, appropriate facilities were prepared at Summerland. All imported clones were established in 1961 on suitable rootstocks in an isolated location, and an indexing program was initiated to determine whether release from this isolation was justified.

Indexing procedure

The number of clones to be indexed, multiplied by the number of indicators deemed necessary for adequate indexing, yielded a total demand on time and facilities that precluded the separate indexing of individual clones. We retained the full range of indicators, but resorted to bulk indexing of groups of the imported clones. The groupings of clones were based on the regions in which they had originated, whether they had been imported directly from these regions or had been acquired through the Introduction Station at Chico (Table 1). The groupings were Austria-Germany-Netherlands; France; Iran - Pakistan - Turkey; Norway - Sweden; Poland-Russia.

The indicators chosen were the varieties of commercial stone fruits most widely planted, or most confidently recommended for planting, in British Columbia in 1961. These were 'Bing', 'Sam', 'Lambert', and 'Van' sweet cherry; 'Montmorency' sour cherry; 'Italian' prune; 'Wenatchee' and 'Tilton' apricot; 'Veteran', 'Redhaven', 'Fairhaven', 'Triogem', 'Golden Jubilee' and 'Valiant' peach. 'Myrobalan B' plum was added, specifically to detect plum pox (sarka) virus (10).

Table 1. Sources of imported cherry and apricot clones, and their groupings for bulk indexing after entry.

	Numbers of clones	
	Cherry	Apricot
Group 1: Austria-Germany-Netherlands		
Germany direct	13	1
Austria direct	3	1
Austria, Germany and Netherlands via California*	10	
Group 2: France		
France direct		4
France via California*	2	
Group 3: Iran-Pakistan-Turkey		
Iran direct		4
West Pakistan direct		2
Turkey via California*	1	
Group 4: Norway-Sweden		
Norway direct	4	
Sweden direct	2	
Group 5: Poland-Russia		
Poland direct	2	14
Poland via California*	6	
Russia via California*	2	

* U. S. D. A. Plant Introduction Garden, Chico, California.

[1] Contribution No. 173 Canada Agriculture Research Station, Summerland, B. C.

[2] Plant Pathologist [3] Technician

The indicator trees were planted in an isolated plot in 1961, and inoculated in 1962. Imported clones that had grown rapidly enough to provide sufficient inoculum budwood were indexed on all indicators. Some clones, which had produced limited terminal growth, were indexed on less extensive host ranges that gave priority to 'Bing' and 'Sam' sweet cherry, 'Montmorency' sour cherry, 'Wenatchee' apricot, and 'Veteran' peach. Two trees of each indicator variety were inoculated. Each tree received two buds from each inoculum source. Four uninoculated check trees of each indicator were included in the plot. Readings were made in the summers of 1963 and 1964.

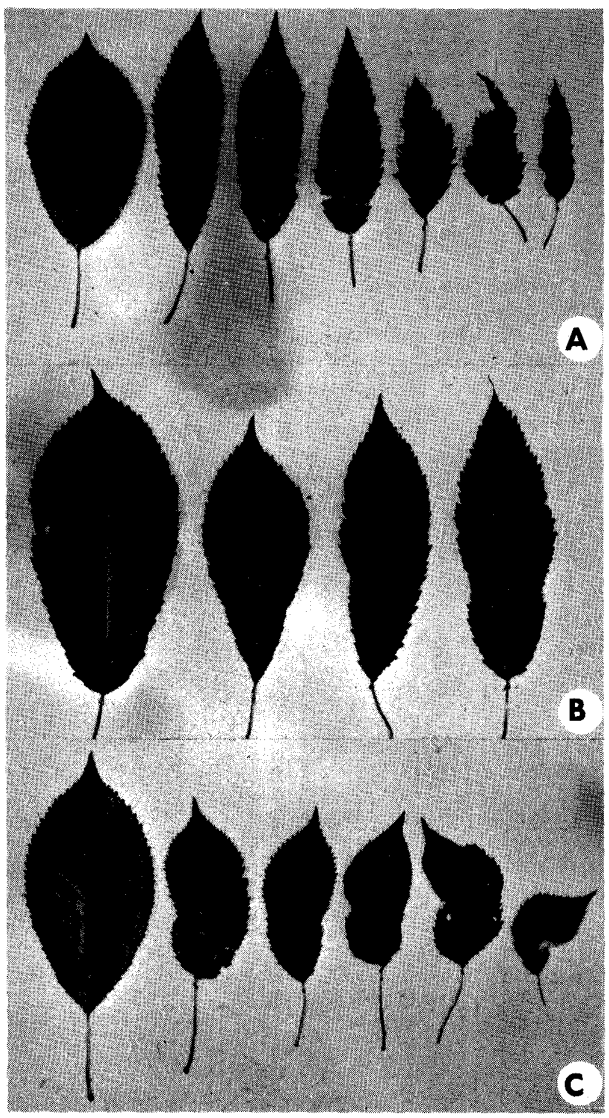


Fig .1. Symptoms on cherry test trees used for bulk indexing of cherry and apricot clones originating in France: (a) leaf narrowing and roughening symptoms on Bing; (b) similar symptoms on Sam; (c) light green areas on leaves of Bing. In each photograph the leaf at left is from an uninoculated tree.

Indexing results

Syndromes expressed in the indicators demonstrated that a number of viruses had been intercepted, including 5 that are considered especially significant because they have induced unfamiliar syndromes on commercial cherry varieties.

The indicator trees of 'Bing', 'Sam', and 'Montmorency' that indexed clones of French origin displayed symptoms in 1963 and 1964 that resembled those of Pfeffingerkrankheit (2) recently re-named rosetziekte (9). On 'Bing' the leaves were narrow, thickened and leathery, with shiny upper surfaces and roughened lower surfaces (Figure 1a). Many had feathered midribs and veins, very mild interveinal enations, and irregular serrations. The symptoms were similar but less severe on 'Sam' (Figure 1b). On 'Montmorency' only scattered branches were affected, bearing leaves that were elongated with slightly roughened lower surfaces and feathered veins. On 'Bing' in both seasons a small proportion of leaves displayed a light green sectoring, or light-colored areas (Figure 1c) resembling the "oil spots" described by Blumer (2) and Mulder (8).

In the late summer of 1963 the clones originating in Austria, Germany and the Netherlands induced moderate to severe reddening on the foliage of 'Sam' and similar but milder symptoms on 'Van'. This was accompanied by slight upcurling of the leaf margins. These are the symptoms induced in these varieties by little cherry virus. However, although these foliage symptoms recurred in 1964, the fruit symptoms of little cherry disease were not apparent in either variety.

In 1964 the 'Bing' trees indexing the Austria-Germany-Netherlands clones displayed symptoms resembling those of rosetziekte. The symptoms differed from those induced in 'Bing' by the clones originating in France only in the absence of "oil spots". However, 'Sam' and 'Lambert' indexing these sources displayed symptoms not induced by the French clones. Several limbs of each 'Sam' tree produced leaves that were small but not elongated. The serrations were irregular, the midrib and veins feathered. Affected leaves displayed mild vein-clearing, and some bore mild interveinal enations. On each of the 'Lambert' trees several limbs displayed symptoms that resembled those of cherry crinkle (Figure 2). The leaves were small with irregular serrations, feathered veins and some vein clearing. Fruiting on these branches was sparse, and the fruits were abnormally pointed. 'Montmorency' trees indexing these clones bore, on scattered branches, elongated leaves with irregular serration and feathered veins.

Clones originating in Norway and Sweden induced on 'Sam' cherry in 1963 and 1964 reactions that included interveinal leaf blotching and premature defoliation (Figure 3). In 1964 gumming blisters appeared on most terminal shoots, gradually girdling them and causing them to shrivel. A similar but milder leaf blotching was induced on 'Van' cherry.

The Norway-Sweden clones induced quite different symptoms on 'Bing' in 1963 and 1964 (Figure 4). On several branches of each tree the leaves developed necrotic lesions on midribs and main veins, sometimes in areas of the leaf lamina. Puckering of the leaves resulted. Spread through the trees appeared to be slow.

In addition to these distinctive and unfamiliar syndromes, several other virus reactions were given by the indicators. Necrotic ring spot symptoms were evident in 1963 on 'Montmorency' trees indexing clones from Austria - Germany - Netherlands, France, Norway-Sweden, and Poland-Russia. Prune dwarf appeared on 'Italian prune' indexing French clones. There was profuse gumming at the sites of a number of pairs of inoculum buds of the following: Norway - Sweden indexed on 'Bing'; Austria - Germany-Netherlands, France and Norway-Sweden on one or both apricot varieties; Austria-Germany-Netherlands, France, Iran - Pakistan - Turkey, and Poland - Russia on most varieties of peach. On most trees the gumming was localized around inoculum buds in 1963 and more generally distributed in 1964 as gumming blisters at other sites on budded limbs.

Among the 'Myrobalan' test trees only one, which was indexing two apricot varieties imported from France, displayed foliage symptoms. These were minute chlorotic flecks, associated with veins, and unlike the symptoms induced by the sarka virus.

Discussion

This virus interception program has amply demonstrated the need for post entry quarantine and indexing of imported cherry and apricot clones. The necessity to resort to bulk indexing has prevented an assessment of the proportion of the imported clones that carried viruses. However, it has demonstrated that numerous virus infections occurred in these clones, and that at least 5 of these infections induce, in commercial cherry varieties, diseases that have

Fig. 2. Leaf symptoms on Lambert cherry test trees used for bulk indexing of cherry and apricot clones originating in Austria, Germany and the Netherlands.

Fig. 3. Leaf and shoot symptoms on Sam cherry test trees used for bulk indexing of cherry clones originating in Norway and Sweden.

Fig. 4. Leaf symptoms on Bing cherry test trees used for bulk indexing of cherry clones originating in Norway and Sweden.

not been observed in Canadian plantings. Re-indexing of the individual clones, now in progress, suggests that most of the more significant infections are in the imported cherry rather than the apricot clones.

Except for the gumming reactions on apricot and peach varieties, which may be merely reactions to viruses of the necrotic ring spot group, there was no evidence of viruses serious in these hosts. The viruses causing serious European diseases of peach, apricot and plum, such as l'enroulement chlorotique des feuilles (3) apoplexie (7) and sarka (1) were not detected in these importations.

The adoption of an indicator host range that represented most of the stone fruit varieties of local importance was a means of ensuring that viruses of significance to the British Columbia tree fruit industry would be detected. The other alternatives considered were (a) the use of the standard stone fruit indicator range used by the IR-2 Tree Fruit Repository Committee (4), or (b) contriving a group of indicators selected specifically for viruses reported in stone fruits in the countries from which the imported clones originated.

Each of these selections of indicator ranges has defects. The one that was chosen would not necessarily be adequate if the imported clones were distributed from British Columbia to other fruit-growing regions on the continent where additional stone fruit varieties are grown. Indeed, already additional varieties of peach are being recommended and planted in British Columbia. The second indicator selection, which is in use for the IR-2 Repository, was devised to detect infections of the stone fruit viruses that occur already in North America; its suitability for interception of exotic viruses can be little more than fortuitous. The third alternative of adopting indicator ranges developed in source countries is applicable only if adequate research has been conducted in those countries to identify the virus infections that occur, and indicators that reveal their presence. Moreover, it might fail to detect viruses that exerted more serious effects on North American varieties than on those in the source countries.

Individual indexing of all clones on very sensitive indicators such as 'Shiro-fugen' flowering cherry, or on herbaceous indicators such as cucumber, is a logical elaboration of any indexing program. However, positive indexing on these indicators need not imply more than presence of latent viruses that are already widely distributed in domestic plantings. Therefore although such indexing provides additional useful information on the virus status of the individual clones, it does not necessarily provide justification for the rejection of importations.

Thus, full confidence cannot be placed in any of these bases for devising interception indicator ranges. The results of the indexing program that we are reporting suggest that one other approach to selection of indicators may usefully supplement those already considered. This is the identification of varieties that give evidence of sensitivity to unusually high proportions of the serious viruses that are introduced to them. Examples in our indexing program were 'Bing' and 'Sam' cherry, which between them gave reactions to all the potentially more serious virus infections detected through the full indicator range. It may be significant that both these varieties have been included in the IR-2 indicator range because of their sensitivity to infections of viruses known in North America.

The factors that merit being taken into consideration in devising indexing procedures for imported stone fruit materials include: (1) the major types and varieties of stone fruits grown in the regions where they will be distributed; (2) the indicators that have given evidence of unusually high sensitivity to viruses in previous indexing experience; and (3) varieties reported to be sensitive to the virus infections that are known to be common and serious in the source countries. Only experience in interception indexing can be expected to verify that these approaches can be reconciled in a limited range of indicators

When virus incidence is as high in imported clones as it proved to be in those we have indexed, the bulk indexing of groups of clones has value only in indicating the prevalence of virus infections, and the types of symptoms that they can produce. It has not proved effective for the prompt clearance of individual virus-free clones to the importer.

Literature cited

1. Atanasoff, D. 1932. Plum pox, a new virus disease. Yearbook Univ. Sofia Fakulty Agr. 11: 49-70.
2. Blumer, S. and Goering, J. 1950. Das Kirschbaumsterben in Baselland (Pfeffingerkrankheit) Phytopathol. Z. 16: 300-335.
3. Bovey, R. 1956. L'enroulement chlorotique, une nouvelle maladie à virus du pêcher. Rev. rom. Agr. 11: 42-43.
4. Fridlund, Paul R. 1962. IR-2, a project with a "blood bank" of virus disease-free fruit trees. Wash. State Univ. Stations Circ. 401. 12 p.
5. Kahn, R. P. et al. 1963. Detection of viruses in foreign plant introductions under quarantine in the United States. Plant Disease Reptr. 47: 261-265.
6. Milbrath, J. A. 1954. The "Eckelrader" disease or "Pfeffingerkrankheit" detected in cherry importations from Europe. Plant Disease Reptr. 38: 258-259.
7. Morvan, M. G. 1957. Mise en évidence de l'action d'un virus dans le dépérissement de l'abricotier. Compt. rend. acad. agr. France 43: 613-614.
8. Mulder, D. 1951. De Eckelrader virusziekte van zoete kersen. Mededel. Dir. Tuinb. 14. 217-228.
9. Pfaeltzer, Hillegonda J. 1961. A soil-borne virus disease of cherries in the Netherlands. Tideskr. Planteavl. 65: 73-82.
10. Schuch, Kurt. 1962. Untersuchungen uber die Pockenkrankeit der Zwetsche. Z. Pflanzenkrankh. Pflanzenschutz 69: 137-142.
11. Welsh, Maurice F. and Keane, F. W. L. 1961. Introduction of a virus to McIntosh apple from an imported clone of Granny Smith. Can. Plant Disease Survey 41: 203-209.

OCCURRENCE OF THE GOLDEN NEMATODE ON VANCOUVER ISLAND, BRITISH COLUMBIA[1]

W. R. Orchard [2]

The golden nematode, Heterodera rostochiensis Woll. 1923, was found for the first time infesting a growing crop on Vancouver Island when it was discovered June 17, 1965, by the author in a commercial 3.5-acre planting of 'Warba' potatoes on the Saanich peninsula at the southern tip of the island. Identity of the nematode was confirmed June 21, 1965, by Mr. R. H. Mulvey, Chief, Nematology Section, Entomology Research Institute, Canada Department of Agriculture, Ottawa, Canada.

At discovery, 92 days from planting, field symptoms and signs of nematode infection were sufficiently marked to indicate a well established infestation. It was learned that the infested field had a crop history of continuous potato production for the past 17 years. Infected plants occurred in roughly circular to oblong zones with the longer axes in the line of natural or tile drainage or with the direction of cultivation. The larger zones were approximately 25 feet in diameter. Infected plants were stunted and were 20 cm high compared to 80 cm for healthy plants. The foliage spread of infected plants was similarly reduced as was the root development and yield. These effects became progressively less from the center to the perimeter of infested zones.

Cysts of the nematode were very numerous on the roots of infected plants and could be seen without aided vision. They were distinguished in the white, yellow, golden, tan and in the chestnut-brown stages with the aid of a X14 hand lens. In lifting potato plants, the disturbance to the root system resulted in the detachment of many cysts which fell from the roots to the soil surface.

Soil samples taken from the root zone of infested plants and processed by the Cobb gravity screening technique yielded gravid females, cysts, males, and larvae of H. rostochiensis. Microscopically, the cysts proved to be near spherical in shape (.542 mm long x .521 mm wide, average dimensions of 20 cysts following overnight immersion in formol solution 10%. The cysts were shiny and carried a body wall pattern with punctations typical for the species as described by Franklin 1951. Examination of perineal sections showed the hatching pore to be circumfenestrate and the anal pore "V" shaped as described for H. rostochiensis by Cooper 1955. The male specimens proved typical of the genus Heterodera and were judged H. rostochiensis on the basis of spicule detail as described by Franklin.

The golden nematode of potatoes was discovered in Newfoundland in October 1962 but heretofore was not known to occur on a growing crop elsewhere in Canada.

Under the direction of the Plant Protection Division, Canada Department of Agriculture, an extensive survey is being carried out to establish the limits of occurrence of this pest in Canada.

1 Contribution No. 207, Experimental Farm, Research Branch, Canada Department of Agriculture, Saanichton, British Columbia.

2 Nematologist, Experimental Farm, Canada Department of Agriculture, Saanichton, British Columbia.

Literature cited

1. Cooper, B. A. 1955. A preliminary key to British species of Heterodera for use in soil examination. Soil Zoology: edited by D. Keith McE. Kevan, Butterworths Scientific Publications, London, 512 pp.
2. Franklin, M. T. 1951. The cyst-forming species of Heterodera. Commonwealth Agricultural Bureaux, 147 pp.

FRECKLE PIT - A VIRUS DISEASE OF ANJOU PEAR[1]

Jack M. Wilkes and Maurice F. Welsh [2]

Introduction

In 1960 freckle pit, a shallow, green-pigmented pitting was observed on 'Anjou' pear fruits in several orchards in the Okanagan Valley of British Columbia. Surveys conducted in 1960 and in subsequent years established that this disease is widely distributed in Okanagan Valley orchards. The senior author has also seen affected 'Anjou' fruits on trees in the northern interior portion of the State of Washington, which borders on the British Columbia fruit-growing region.

Symptoms of a disorder in the White Salmon district of southern Washington that were described by Kienholz (3) resemble those observed in British Columbia and northern Washington. No other published records of the occurrence of this syndrome have been found, and no tests to determine its cause have been reported.

Symptoms

Mild symptoms of the disease become apparent in late August (about 1 month before picking). They become increasingly severe toward the harvesting season, at which time affected fruits bear numerous, slightly sunken, dark green pits about 1/8-inch in diameter (Fig. 1). These pits are underlaid by a network of dark green strands that penetrate the flesh 1/8 to 1/4 inch (Fig. 2). Pitting is more concentrated and severe at the calyx end. As the fruit ripens, the green pigment in the pits becomes lighter but still can be distinguished from the golden yellow ground color of unaffected skin. During this ripening period the green strands in the flesh turn brown. In fruits that are mildly or moderately affected, the skin and flesh symptoms become less apparent, or disappear, during ripening. No foliage or tree abnormalities have been associated with these fruit symptoms.

Transmission tests

Between 1960 and 1962 budwood from 4 affected 'Anjou' trees in 3 Okanagan Valley orchards was used to inoculate healthy test trees of 'Anjou', 'Bosc', 'Bartlett', and 'Flemish Beauty', either by spring or summer budding.

Four of the 5 inoculated 'Anjou' trees displayed symptoms, three full growing seasons after inoculation. No symptoms have been observed in 5 fruiting trees of 'Bartlett', 4 of 'Bosc', and 3 of 'Flemish Beauty', all of which have been inoculated for three or more years.

Nine affected 'Anjou' trees in a commercial orchard were top-worked by the owner to 'Bartlett' in 1960. In three subsequent years of cropping none of the 'Bartlett' fruits have displayed symptoms.

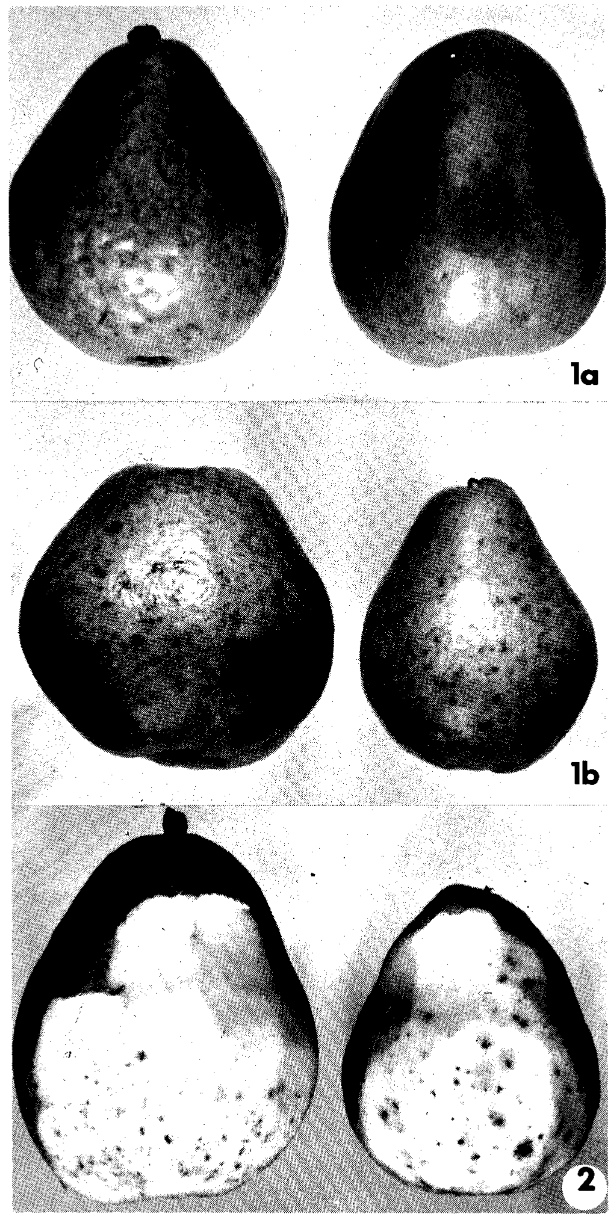


Fig. 1. External symptoms of freckle pit on Anjou pear fruit, (a) pitting on fruit at picking maturity, (b) pitting on ripening fruit, with colour differences evident.

Fig. 2. Peeled Anjou pear fruit showing the dark green strands that penetrate the flesh beneath areas affected with freckle pit.

Natural spread

Surveys have been conducted annually in 4 affected blocks of 'Anjou' pear trees for 4 to 5 years (Table 1). Symptoms have recurred each year in affected trees, but no spread has been observed.

---

1 Contribution No. 171, Canada Agriculture Research Station, Summerland, British Columbia.

2 Plant Pathologists.

Table 1. Incidence of freckle pit in grower orchards that have been surveyed annually.

Orchard No.	No. of years surveyed	No. of trees infected	Total No. of trees	% Infection	Age of trees (years)
1	5	9	67	13.4	15
2	5	46	68	67.6	14 trees about 50
					52 trees about 20
3	5	10	12	83.3	15
4	4	23	104	22.1	12-15
Totals		88	251		

Conclusions

The virus etiology of freckle pit has been demonstrated by the successful experimental transmission from 'Anjou' to 'Anjou'. Among the varieties of pears grown commercially in British Columbia, and included in the transmission tests, apparently only 'Anjou' displays symptoms. Tests are in progress to determine whether 'Bartlett', 'Bosc' and 'Flemish Beauty' are resistant or tolerant.

This disease can be distinguished from other reported virus - induced, fruit - pitting diseases of pear (1, 2) by the type of symptoms on 'Anjou', as well as by the failure of the causal virus to induce symptoms in 'Bosc', and 'Flemish Beauty'.

The absence of natural spread during a 4-5 year period, in pear plantings that total 251 trees, and that are 13% to 83% infected, provides substantial evidence that dissemination of the virus must have resulted from the use of infected pear scionwood.

Literature cited

1. Keane, F. W. L. and Maurice F. Welsh. 1960. Transmissible corky pit of 'Flemish Beauty' pear. Plant Disease Reptr. 44: 636-638.
2. Kienholz, J. R. 1939. Stony pit, a transmissible disease of pears. Phytopathology 29: 260-267.
3. Kienholz, J. R. 1943. Observations on certain pits and other blemishes of pear fruits. Proc. Wash. State Hort. Assoc. 39: 51-57.

RED STRIPED PERICARP OF CORN

R. E. Wall and C. G. Mortimore[1]

During September of 1964, a longitudinal red striping of kernels of field corn was observed in Essex and Kent Counties in southwestern Ontario. The discoloration was limited to the pericarp and varied in intensity from a faint streak on the side of the kernel to an almost complete reddening, with streaks extending over the crown (Fig. 1). The reddening was most noticeable on the round kernels near the tip of the ear. In all respects, the condition resembled the "red-stripe" reported from southern Michigan, northeastern Indiana, and northwestern Ohio in 1963 (2) and from more widespread areas in 1964 (5). The cause of the red-stripe has not been determined.

Ears with red-striped pericarp were present in most fields examined in Essex and Kent Counties. The intensity of the condition varied considerably from plant to plant, but there appeared to be no great differences in incidence among commercial hybrids. However, inspection of numerous inbreds and single crosses harvested at the Harrow Research Station revealed the absence of red-stripe in several cases. Therefore, grain samples from several genotypes, each randomized in three replicate blocks, were examined for red-stripe. The samples contained 200 to 400 kernels from the centre portions of three ears that were collected at weekly intervals during the autumn and oven-dried to determine kernel weight and moisture contents. The samples were rated for red-stripe on a 0 to 5 scale, representing no discoloration to almost complete reddening of all kernels, respectively.

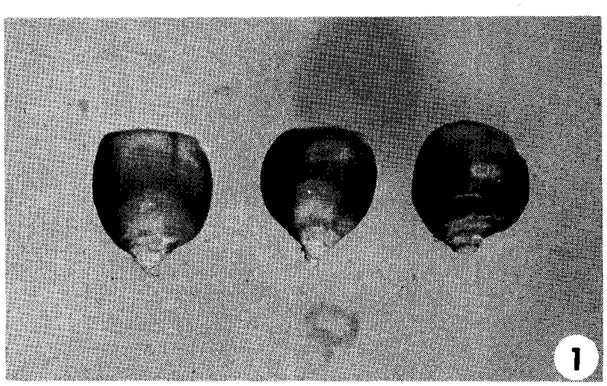


Fig. 1. Kernels of dent corn showing the pattern and range of intensity of red-stripe.

Kernel samples collected on the earliest date, Sept. 3, had very little or no red-stripe (Table 1). Apparently the discoloration did not occur until a few days to two weeks before physiological maturity.

The only single-cross of known parentage entirely free of red-stripe, CH159 x CH3, was made up of inbreds with practically no striping whereas the parents of single-crosses showing considerable striping had high red-stripe ratings (Table 1). The data would indicate that there is some genetic control over the expression of red-stripe.

In cold germination tests carried out by Hoppe's method (3), there were no differences in per cent germination between samples with and without red-stripe from the same population. Both untreated and Arasan-treated seed were tested. Seedlings from red-striped seed did not differ in appearance from those derived from normal seed.

Apparently, many factors condition the appearance of red-stripe. Its occurrence late in the growing season, shortly before maturity suggests a physiological or an environmental effect. There is also a genetic effect on the expression of red-stripe. The possibility of a transmissible pathogenic factor involved in red-stripe is suggested by its apparent "spread" from an area involving three States in 1963 to a much wider area across the United States and Canada in 1964 (2, 5).

Literature cited

1. Anonymous, 1963. MSU, USDA analyze "red-stripe" corn in Mich-Ind-Ohio. Seed Trade News 81 (24).
2. Anonymous, 1964. Three states affected by red-striping. Buckeye Farm News. February. p. 38.
3. Hoppe, P. E. 1957. The rolled towel seed tester for corn. U. S. D. A. Leaflet No. 425.
4. Kunze, R. E. 1964. Corn disease is threat to farmers in 1964. Buckeye Farm News. February. p. 18, 20.
5. Ullstrup, A. J. 1964. Private communication.

1 Research Station, Harrow, Ontario, Canada.

Table 1. Incidence of red-striped pericarp in inbreds and single-crosses grown at the Research Station, Harrow, Ontario, in 1964.

Inbred or Single-cross	Approx. date of physiological maturity[1]	Red-stripe Rating[2]				
		Sept. 3	Sept. 10	Sept. 16	Sept. 24	Oct. 1
M13	Sept. 12	0.7	2.3	2.3	3.0	--
A374	Sept. 12	0	0.3	1.3	0.3	--
W153R	Sept. 12	0	0.3	2.3	1.3	--
US153	Sept. 12	0.3	1.3	2.3	3.0	--
W37A-R1	Sept. 15	0	0	1.3	2.0	--
CH159	Sept. 20	0	0	0	0.3	--
CH160	Sept. 20	0	0	0.3	0	--
CH3	Sept. 25	--	0	--	0	0.3
CH9	Sept. 25	0.3	--	1.3	3.3	3.0
Oh51A	Sept. 28	--	1.0	2.0	2.7	3.0
M14	Sept. 28	--	0.3	1.3	1.3	2.3
A374 x CH159	Sept. 12	0	0	0.3	0.3	--
W37A-R1 x CH9	Sept. 12	0	1.0	1.0	0.7	--
W37A-R1 x W153R	Sept. 12	0	0	1.0	1.7	--
W37A-R1 x WF9	Sept. 15	0	1.3	1.0	2.7	--
W37A-R1 x Oh51A	Sept. 15	0	0.7	1.3	1.3	--
CH159 x CH3	Sept. 25	--	0	0	0	0
B14 x CH9	Sept. 28	--	1.3	1.3	1.7	1.5

1 Date of maximum kernel dry weight.

2 Based on 0 to 5 scale; 0 = no stripe; 5 = intense discoloration of all kernels.

WILLOW BLIGHT AND THE SURVIVAL OF SOME SALIX SPECIES IN NOVA SCOTIA[1]

K. A. Harrison[2]

The old French willow, (Salix alba L. var. vitellina (L.) Stoke), once one of the beautiful shade trees in Nova Scotia, is now little more than a memory of an older generation. This willow apparently was introduced early in the history of the French settlement of the province and was used extensively around buildings. Willow posts, used to mark property boundaries, often sprouted and in time grew to become impressive rows of trees along roadsides and banks of streams. Now they have almost completely disappeared because of the destructive disease, willow blight. There is no record of how or when this disease was introduced but in 1926 it caused severe defoliation of trees in many parts of the province. It seems most likely that it was introduced from Europe on nursery stock during or shortly after World War I.

Willow blight in Nova Scotia is an expression of the interaction of two diseases; willow scab caused by Pollacia saliciperda (Allesch. & Tub.) Arx, which is related to apple scab, and willow canker, Physalospora miyabeana Fukushi, first described on willow in Japan and later on basket willow in England. Both organisms overwinter in cankers on twigs and sprouts. The scab organism attacks the leaves early in the spring and progresses along the petiole into the twigs. The canker organism enters later through leaves and tissues injured by scab. Twigs and new growth are killed during the late spring and summer. Different species and varieties of willow vary in their susceptibility to each organism. They must be susceptible to both before willow blight is destructive.

---

1 Contribution No. 1207 from the Research Station, Canada Department of Agriculture, Kentville, Nova Scotia.

2 Plant Pathologist

Salix pentandra did not thrive on this site but the original clone growing in Greenwich, Kings County, is vigorous and has developed into a striking group of trees. S. blanda was very susceptible and soon died out. S. fragilis has sufficient resistance to survive attacks and is found in some parts of the province.

In 1935 cuttings of the cricket bat willow, S. alba L. var. calva GFW Mey (S. coerulae Sm.), free of water mark disease, was obtained from England through the kindness of the late Prof. W. J. Dowson of Cambridge. This species was first tested in the laboratory for susceptibility to willow blight and then under natural conditions in the field near infected willows. Blight never developed on this willow although other varieties were killed. The species is recommended for replacing blighted willows in Nova Scotia. Cuttings grow rapidly and develop rather quickly into good sized trees.

An original planting of French willows survives in the Grand Pre Memorial Park where it is kept alive by a regular program of sprays. The trees are shorter now than they were originally because of the difficulty of spraying the tops adequately to prevent the blight there. Control was obtained originally with 5 sprays of Bordeaux 8-24-100, starting when the buds first break and repeated every 10 to 14 days. Control is now maintained with 3 or 4 sprays of Phygon, ½lb to 100 gal water. The early sprays are most effective and in seasons with low rainfall 3 sprays are sufficient for control.

In 1933 a row of willows, obtained from cuttings from various sources, was planted parallel to the highway and along one side of a pastured block of dyked meadow on the Research Station, Kentville. The site was favorable and the willows grew vigorously. Several French willows were near and furnished a natural source of inoculum. Table 1 lists the species planted and the number surviving in 1965.

Table 1. Willows planted in May 1933 and records taken April 1965

Number planted	Survived	*Salix* species	Comments
1	0	*caprea* L.	No trace remaining.
1	1	*alba* L. var. *tristis* Gaudin[1]	Tree thriving.
2	0	*viminalis* L.	No trace remaining.
4	3	*alba* var. *vitellina* (L.) Stoke	Trees barely surviving, blighted.
4	3	*alba* var. *tristis* Gaudin[2]	Trees badly blighted.
4	0	*nigra* Marsh	No trace remaining.
5	4	*alba* var. *tristis* Gaudin[3]	Trees large, some blight.
3	0	*pentandra* L.	No trace remaining.
4	0	*amygdalina* L.[4]	No trace remaining.
5	5	*aurita* L. x *viminalis* L.[6]	Thicket of sprouts.
4	4	*fragilis* L.[5]	Trees large, thriving.
3	0	*blanda* Anderss.	No trace remaining.
5	3	*fragilis* L.	Trees large, some blight.
5	2	*britzensis*[7]	Trees suppressed, crowded by oaks.

1 Received as *S. niobe*
2 " " *S. vitellina* var. *pendula*
3 " " *S. babylonica aurea*
4 " " *S. triandra*
5 " " *S. fragilis besfordiania*
6 As received.
7 Wrongly named; correct name unknown.

FOLIAGE DISEASES OF VEGETABLES IN QUEBEC IN 1963 AND 1964 AND THEIR RELATIONSHIP TO RAINFALL

Thomas Simard[1], Rene Crete[2] and Jacques Simard[3]

Abstract

Observations made over a six-year period in the muck soil district south of Montreal indicate that early establishment of foliage diseases may be expected and fungicide applications required in July when the June rainfall is close to or above the 27-year average. Rainfall in June notably lower than the 27-year average indicates a late development of diseases and, therefore, no fungicide sprays should be required in July.

Résumé

Des observations échelonnées sur une période de six ans dans la région des sols organiques du sud de Montréal indiquent qu'il y a risque d'implantation hâtive des maladies foliaires lorsque le total des pluies de juin approche ou dépasse la moyenne de 27 ans. Il peut alors être nécessaire de commencer les traitements fongicides en juillet. Par ailleurs, une précipitation de juin notablement inférieure à la moyenne de 27 ans serait un indice d'un développement tardif des maladies foliaires et de l'inutilité d'avoir recours à des traitments fongicides en juillet.

Introduction

Since 1959, annual surveys of the plant diseases occurring on the principal vegetable crops grown in muck soils south of Montreal have been carried out. The object of these surveys was to follow disease development in the area and to try to relate this development to annual climatic conditions, especially rainfall. The general method used is described in an earlier report (3). The diseases observed in 1963 and 1964 and their intensity are listed in Tables 2 to 5. The annual rainfall for 1959 to 1964 and the 27-year average rainfall from June to September for the period 1938 to 1964, recorded at Ste. Clotilde, Que., were obtained from M. C. Peron, of the Research Station at St. Jean, Que, and are presented in Table 1. These figures are considered to be representative of the general situation in the muck soil district.

Table 1. Rainfall in inches at Ste-Clotilde (Chateauguay).

Year	June	July	August	September
1959	3.46	1.44	5.18	1.43
1960	3.19	1.54	1.56	4.31
1961	3.32	5.12	3.77	0.52
1962	1.93	4.76	4.59	2.76
1963	2.83	1.62	6.04	3.13
1964	1.72	2.91	3.03	1.19
27-year average	3.50	3.50	3.39	3.15

Results and discussion

In general, the results for 1963 were similar to those presented for 1962 (4). In both years, foliage diseases were observed about one month later than in 1961 and did not develop extensively. In 1964, the situation was even less critical; the diseases developed about 45 days later than in 1961 and, in general, traces only were recorded early in September.

1 Plant Pathologist, Research Division, Quebec Department of Agriculture, Montreal, Que.

2 Plant Pathologist, Research Station, Research Branch, Canada Department of Agriculture, St. Jean, Que.

3 Formerly Plant Pathologist, Research Division, Quebec Department of Agriculture, Montreal, Que.; now Training Consultant, Primary Industries, Technical and Vocational Training Branch, Department of Labour, Ottawa, Ont.

Table 2. Diseases in the Ste-Clotilde region.

CROP	DISEASES	DISEASE INTENSITY 1963	1964
CARROT	Alternaria leaf blight (Alternaria dauci)	4-tr./4 fields	1-tr./4 fields
	Cercospora leaf blight (Cercospora carotae)	4-tr./4 fields	2-sl./4 fields
	Root-knot nematode (Meloidogyne hapla)		1-tr./4 fields
CELERY	Bacterial blight (Pseudomonas apii)	2-tr./4 fields	
	Pink rot (Sclerotinia sclerotiorum)	1-tr./4 fields	
LETTUCE	Aster yellows (callistephus virus 1)	3-tr./3 fields	1-tr./1 field
	Downy mildew (Bremia lactucae)	3-tr.-sl./3 fields	1-tr./1 field
	Mosaic (virus)	3-tr. /3 fields	
	Root-knot nematode (Meloidogyne hapla)	1-tr./3 fields	
ONION	Calcium deficiency	1-tr./4 fields	
POTATO	Early blight (Alternaria solani)		1-tr./3 fields
	Late blight (Phytophthora infestans)		1-tr./3 fields
	Herbicide injury	1-tr./3 fields	
	Tip burn	3-tr.-sl./3 fields	
SUGAR BEET	Leaf spot (Alternaria tenuis)	2-tr./2 fields	

Table 3. Diseases in the Napierville region.

CARROT	Alternaria leaf blight (Alternaria dauci)	2-tr./2 fields	2-tr./2 fields
	Cercospora leaf blight (Cercospora carotae)	2-tr./2 fields	
ONION	Leaf fleck (Botrytis cinerea)	3-tr./3 fields	1-sl./1 field
	Calcium deficiency	3-mod./3 fields	
POTATO	Rhizoctonia (Pellicularia filamentosa)	2-tr./2 fields	

Table 4. Diseases in the Sherrington region.

CROP	DISEASES	DISEASE INTENSITY 1963	1964
CARROT	Alternaria leaf blight (*Alternaria dauci*)	3-tr./3 fields	1-sl./2 fields
	Cercospora leaf blight (*Cercospora carotae*)	3-tr./3 fields	1-sl./2 fields
	Root-knot nematode (*Meloidogyne hapla*)	1-tr./3 fields	
	Aster yellows (callistephus virus 1)		1-tr./2 fields
CELERY	Late blight (*Septoria apiicola*)	2-tr./ 8 fields	
	Bacterial blight (*Pseudomonas apii*) (on Utah 10-B or 1611)	3-mod./8 fields	
LETTUCE	Aster yellows (callistephus virus 1)	1-tr./ 1 field	
ONION	Fusarium basal rot (*Fusarium oxysporum* f. *cepae*)	4-tr./12 fields	
	Downy mildew (*Peronospora destructor*)	3-tr./12 fields	
	Leaf fleck (*Botrytis cinerea*)	3-mod./12 fields	
	Leaf blight (*Botrytis squamosa*)	3-tr./3 fields	
POTATO	Rhizoctonia (*Pellicularia filamentosa*)	1-sl./3 fields	
	Late blight (*Phytophthora infestans*)	2-sev./3 fields	
	Black heart (oxygen deficiency)		1-sl./1 field

Table 5. Diseases in the Farnham region.

CROP	DISEASES	1963	1964
CARROT	Alternaria leaf blight (*Alternaria dauci*)	1-sl./1 field	1-tr./2 fields
	Cercospora leaf blight (*Cercospora carotae*)	1-tr./1 field	1-sl./2 fields
	Root-knot nematode (*Meloidogyne hapla*)	1-tr./1 field	
LETTUCE	Downy mildew (*Bremia lactucae*)	1-tr./1 field	
	Aster yellows (callistephus virus 1)	1-tr./1 field	
	Mosaic (virus)	1-tr./1 field	
ONION	Leaf fleck (*Botrytis cinerea*)	3-tr./3 fields	
	Leaf blight (*Botrytis squamosa*)		1-mod.1-sev./2 fields

This seems to support the hypothesis of a correlation between June rainfall and inoculum build-up leading to the establishment of initial disease foci, put forward earlier (4).

Table 1 shows that the June rainfall in the years 1959-1961 was close to the 27-year average at Ste. Clotilde. 1961, with heavy rainfall in June, July and August, was characterized by a general spread and high intensity of foliar diseases on potato, onion and carrot. In 1961, early epidemics of potato late blight (P. infestans) on both early and late varieties, onion leaf blight (B. squamosa) and mildew (P. destructor) and carrot leaf blights (A. dauci and C. carotae) were observed (3). In 1959 and 1960, the June rainfall was high but latter parts of both seasons were dry, except in August 1959. Consequently, the foliar diseases mentioned above although generally observed, were at a low intensity (1, 2).

The years 1962-1964 were characterized by June rainfall notably lower than the 27-year average. During those three years, foliage diseases developed late; in mid-August of 1962 and 1963, and early in September of 1964. During this period, disease was most serious in 1962, when July and August rainfalls were higher than the 27-year average (4). During the six years under observation, disease development and intensity were the least serious in 1964, when rainfalls for June, July and August were below the 27-year average.

In summary, these results indicate that an early establishment of foliage disease may be expected and fungicide applications required in July, when the June rainfall is close to or above 3.50 inches, the 27-year average at Ste. Clotilde. Further spread and development of the diseases will depend on the July and August rainfalls.

A June rainfall much lower than the 27-year average indicates a late development of diseases and, therefore, fungicide sprays should not be required in July. Under these conditions, if fungicide sprays are applied in August in years when rainfall is high during July and/or August, no serious economic losses should be expected from foliage diseases.

Literature cited

1. Simard, Jacques et René Crête. 1959. Observations sur quelques maladies des cultures de légumes en terre organique du sud de Montréal. Société de Québec pour la protection des plantes, 42e rapport: 23-24.
2. Simard, Jacques, René Crête and Thomas Simard. 1960. Vegetable disease on muck soils in the Montreal area in 1960. Can. Plant Dis. Survey 40: 72-74.
3. Simard, Jacques, René Crête and Thomas Simard. 1961. Vegetable diseases on muck soils in the Montreal area in 1961. Can. Plant Dis. Survey 41: 353-356.
4. Simard, Jacques, René Crête and Thomas Simard. 1962. Vegetable diseases on muck soils in the Montreal area in 1962. Can. Plant Dis. Survey 42: 216-219.