Evaluation of a Method Used to Estimate Loss in Yield of Potatoes Caused by Late Blight

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Contribution No. 262 and 232 for Ottawa and Charlottetown Research Station, respectively, Canada Department of Agriculture, Ottawa, Ontario.

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The authors gratefully acknowledge the technical assistance at each of the trials, and also thank the Chevron Chemical Company for providing samples of Difolatan. Grateful acknowledgment is expressed to the Cartography Section, Soil Research Institute, for preparing the illustrations.

Accepted for publication 15 April 1971.

ABSTRACT

Principles of a disease assessment method, developed in England and Wales to estimate loss in potato tuber yield caused by late blight, *Phytophthora infestans*, were evaluated in eastern Canada. Field trials were conducted at three locations in 1969 and two in 1970, using the cultivars Green Mountain and Katahdin. The fungicide Difolatan 4 Flowable [*N*-([1,1,2,2 -tetrachloroethyl]-sulfenyl)-*cis*-4-cyclohexene-1,2-dicarboximide] which did not affect tuber yield except by controlling late blight, was used to influence three different epidemics by operating various spray schedules. All the disease progress curves were not satisfactorily defined when the data were transformed using $\log_{10} \frac{x}{1-x}$ and regression lines fitted. The disease assessment method assumes that tuber production stops when defoliation by late blight reaches 75%. The loss is calculated by ascertaining the time when this point is reached and using this datum in conjunction with a tuber development curve for Green Mountain. Actual losses, 42, 33, 52, 17, and 26% derived by weighing, were in poor agreement with the estimated losses, 23, 10, 16, 3, and 0%, respectively, computed by the method. Possible reasons for the divergence in results are discussed. Phytopathology 61: 1471-1476.

Late blight of potato (*Solanum tuberosum* L.) caused by *Phytophthora infestans* (Mont.) d By., is one of the most intensively studied diseases of major food crops. However, only one method (14) has been developed to predict or estimate the loss in tuber yield. The method utilized the growth or bulking curve for tubers in a healthy crop (Fig. 3) and information on the amount of blight on the foliage before tuber growth stops; these observations were obtained by periodic tuber sampling of healthy and diseased crops throughout the season, and the latter were also assessed for disease. In England and Wales, tuber growth stopped when late blight affected 75% of the foliage of the cultivars, Majestic and King Edward (14). The principle of the method involves ascertaining when 75% of the foliage is affected by late blight, then reading off the percentage of the potential yield normally accumulated after this date from a bulking curve for the tubers, this reading being equivalent to the percentage loss in total tuber yield resulting from late blight. The method was tested (2) and later verified in various spraying trials (9, 14, 15, 17) by weighing the tubers from sprayed and unsprayed plots and relating the yield to the progress curves of the disease. Using the prediction method described above, estimated gains from spraying were compared with actual gains. It was reported (9) that the "...method proved to give mean estimates of defoliation losses at least as accurate as those by weighing the produce from sprayed and unsprayed plots". The success of the method depends on the fact that "...a mean bulking curve must be obtained for the variety in the region for which loss assessments are to be made" (9).

The objective of our experiments was to test the applicability of this method in Canada, where the growing period is ca. 40 days shorter than in England, and where different cultivars and cultural practices are used, under different environmental conditions. It is important to note that the mean bulking curves for the region suggested and used by Cox & Large (9) have been employed for the calculation of estimated losses for experiments reported in this paper.

MATERIALS AND METHODS.—Cooperative field trials were organized at Charlottetown (Prince Edward Island), Fredericton (New Brunswick), and Ottawa (Ontario) during 1969, and at Charlottetown and Ottawa in 1970. All experiments were conducted in a similar manner, and materials used were identical except where stated otherwise. A randomized block design was used. Three treatments, replicated 6 times, resulted in 18 plots. Each plot was 50 ft long and consisted of four rows 36 inches apart; the seed pieces were planted 12 inches apart. A buffer row was left between plots. The cultivar, Green Mountain, was used in each experiment except at Fredericton, where Katahdin was used. The planting, inoculating, top-killing, and harvest dates for each experiment are shown in Table 1. A water suspension containing spores of several races of *P. infestans* was applied to the buffer rows in the evening.

The three treatments represented varying fungicide spray schedules designed to produce three epidemics: (A) No fungicide spray was applied, to simulate an early infection; (B) fungicide spray from mid-July to mid-August, to simulate a late infection; (C) fungicide spray from mid-July to mid-September, to give a low level of disease or, preferably, complete control. The

TABLE 1. Planting inoculation, topkilling, and harvest dates for potato field trials at three locations in eastern Canada in 1969 and 1970

Trial	Year	Planting	Inoculating[a]	Topkilling	Harvesting
Charlottetown	1969	6 June	25 July	23 Sept.	20 Oct.
Fredericton	1969	28 May	16 July	22 Sept.	30 Sept.
Ottawa	1969	10 June	16 July	20 Sept.	27 Oct.
Charlottetown	1970	12 June	24 July	18 Sept.	9 Oct.
Ottawa	1970	26 May	15 July	1 Oct.	8 Oct.

[a] Buffer rows inoculated with a spore suspension of *Phytophthora infestans*.

fungicide, Difolatan 4 Flowable [*N*-([1,1,2,2-tetrachloroethyl] sulfenyl)-*cis*-4-cyclohexene-1,2-dicarboximide], was chosen because of its good performance in screening tests (6, 7, 8). The fungicide was applied at 0.8 imperial quarts (1.00 lb. active ingredient) in 120-150 gal of water/acre (1.12 kg active ingredient/hectare), using a tractor-sprayer unit except at Ottawa, where a 30-ft boom sprayer was used capable of spraying all the plot area from the perimeter of the experiment, thus avoiding any wheel damage to the foliage. In 1970, the amount of Difolatan applied was increased by 50% to 1.5 lb. active ingredient/acre (1.68 kg active ingredient/hectare). Insects were controlled by spraying the experimental area with endosulfan (1,4,5,6,7,7 - hexachloro-5-norbornene-2,3-dimethanol cyclic-sulphite) when required.

Disease assessments on the two center rows in each plot were recorded at 3- to 7-day intervals after the epidemic started. The British Mycological Society assessment key (4) developed by Large (13, 14) was used to record the percentage infection and defoliation caused by late blight. If necessary, interpolations were made between the fixed percentages in the key. One observer made some assessments at all three locations and compared results with the local observer. The check showed that assessments by different observers of a particular plot were comparable.

The foliage was killed by sodium arsenite, and the tubers from the center two rows of each plot were lifted, graded, and weighed (Table 1). A small area of Green Mountain was planted near the main experiment in Ottawa in 1970, and tubers were sampled at 2-week intervals to obtain data on the growth of tubers. The experimental procedures were identical to those employed in the main experiment, and tubers from a block of 20 plants were harvested at each sampling date.

RESULTS.—Successive assessments for late blight were plotted against time to produce progress curves depicting the progress of late blight for the three treatments at four trials (Fig. 1). The standard errors for some treatment means (untransformed) are shown for the successive dates. Alternatively, the epidemic may be described by $\log_{10} \frac{x}{1-x} = a + bD$, where x = the proportion of foliage affected by late blight and D = time (19). The rate of infection is equivalent to the slope of the line (Fig. 2). Regression lines were fitted to the points in each treatment, but the percentage of total variability explained by the regression was low for some treatments. The percentage explained for treatment A at Fredericton in 1969 and treatments A, B, and C at Charlottetown in 1969 and 1970 was lower than that for other treatments, indicating that the fit was not as satisfactory. This is also expressed in the higher standard errors of the b values for these treatments (Table 2).

The total yield (including large, small, and blighted tubers) associated with each of the epidemics is shown in Table 3, with treatment means and their corresponding standard errors given in the same table. The average of the C treatments is equivalent to 12.0 tons/acre (29 metric tons/hectare), which is considered comparable to potato yields on commercial farms in eastern Canada. The yields of treatments A and B have also been calculated as a percentage of the corresponding C treatment in each case; the difference between this figure and 100% is equivalent to percentage loss in yield, and is recorded in Table 3 as actual yield loss.

Bulking curves (Fig. 3) for the cultivars Green

TABLE 2. Regression coefficients for disease progress curves, and percentage variability explained by regression lines[a]

Location, year, and treatment[b]	b Value or infection rate	% Variability explained by regression
F 1969 A	.094 ± .018	84
F 1969 B	.070 ± .003	99
F 1969 C	.052 ± .006	93
C 1969 A	.130 ± .024	83
C 1969 B	.070 ± .012	85
C 1969 C	.060 ± .014	75
C 1970 A	.140 ± .023	86
C 1970 B	.165 ± .046	72
C 1970 C	.074 ± .014	84
O 1970 A	.046 ± .002	97
O 1970 B	.099 ± .013	95
O 1970 C	.046 ± .013	92

[a] Regression equation for the transformed disease data is $\log_{10} \frac{x}{1-x} = a + bD$, where x = proportion of foliage affected and D = time.

[b] First letter refers to location: F = Fredericton; C = Charlottetown; O = Ottawa; second letter refers to treatment with fungicide (see Fig. 1).

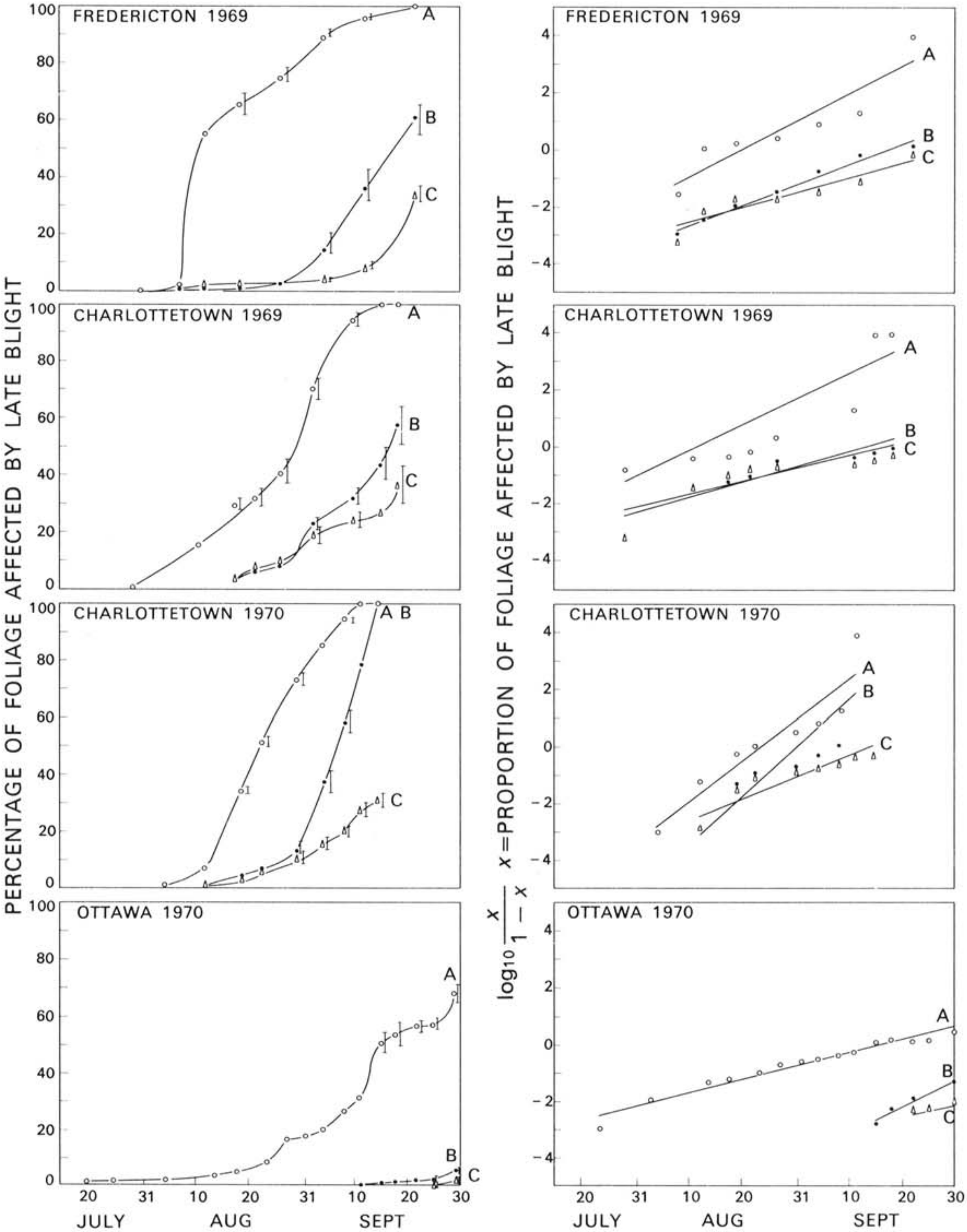

Fig. 1-2. 1) Progress curves for late blight on potatoes in four field trials where three fungicide spray schedules were operated. ○——○ A = no fungicide applied; ●——● B = fungicide spray applied from mid-July to mid-August; △——△ C = fungicide applied from mid-July to mid-end September. The length of the vertical line represents the standard error (SE) on either side of the mean. Each SE was calculated from six disease assessments, one from each replicate. Standard errors were not calculated for disease assessments made earlier in the season, or when the values for all replicates were identical. **2)** Regression lines fitted to the data in Fig. 1. using the transformation $\log_{10} \frac{x}{1-x}$. 100% disease (x = 1.00) is taken as 99.99% (x = 0.9999).

TABLE 3. Yield of tubers from sprayed and unsprayed plots with comparisons of estimated and actual losses in tuber yield

Location, year, and treatment[a]	Yield of tubers (lb.) Means with SE	Yield loss expressed as % of treatment C	
		Actual	Estimated
F 1969 A	103.3	42	23[b]
F 1969 B	177.9 ± 2.7	1	
F 1969 C	179.5		
C 1969 A	120.4	33	10[c]
C 1969 B	190.4 ± 2.4	(6)[d]	
C 1969 C	180.2		
O 1969 A	69.7	(4)	
O 1969 B	71.4 ± 4.1	(7)	
O 1969 C	66.7		
C 1970 A	83.3	52	16[c]
C 1970 B	143.6 ± 4.1	17	3[c]
C 1970 C	172.2		
O 1970 A	215.4	26	0[c]
O 1970 B	283.0 ± 8.7	3	
O 1970 C	292.4		

[a] First letter refers to location: F = Fredericton; C = Charlottetown; O = Ottawa; second letter refers to fungicide (see Fig. 1).
[b] Based on Katahdin bulking curve produced at Maine (see Fig. 3).
[c] Based on Green Mountain bulking curve produced at Maine (see Fig. 3).
[d] Figures in parentheses represent an increase in yield.

Mountain and Katahdin were derived from the data of Akeley et al. (1) for potatoes grown in Aroostook County, Presque Isle, Maine, USA, for the 3 years 1950-1952; this area is adjacent to the potato-growing area in New Brunswick. The remaining two bulking curves in Fig. 3 represent the cultivar, Majestic, grown in England (10), and the cultivar, Green Mountain, grown in Ottawa in 1970.

The actual losses (Table 3) associated with the different epidemics were compared with the computed estimated losses. The estimated losses were calculated using the method described by Large (14), basing the calculation on the bulking curve for Green Mountain and Katahdin grown in Maine (Fig. 3). The date on which the progress curves of treatments A and B reach 75% disease on the foliage is taken at the point when tuber growth stops (9). Using this date, the percentage loss of tuber production is read off the right-hand scale (Fig. 3) of the bulking curves; this percentage is the estimated loss. In each case, the potential or 100% yield is equivalent to treatment C, although it has disease in some cases. If the estimated losses are calculated by using the bulking curve for Green Mountain produced at Ottawa in 1970 (Fig. 3), the results are similar. In each trial, the estimated loss is much smaller than the actual loss.

DISCUSSION.–Most disease assessment research involves a stage with field experiments where the disease is allowed to develop in some plots, but is controlled by fungicide in others. When the corresponding plot yields are compared, there is always the possibility that the decrease or increase in yield in the sprayed plot can be attributed either to phytotoxicity (18) or beneficial effect of the fungicide over and above its effect on disease, such as that reported for potato crops sprayed with zinc-containing fungicides (3, 5, 11, 12). Rosser (17) attributed a yield loss of

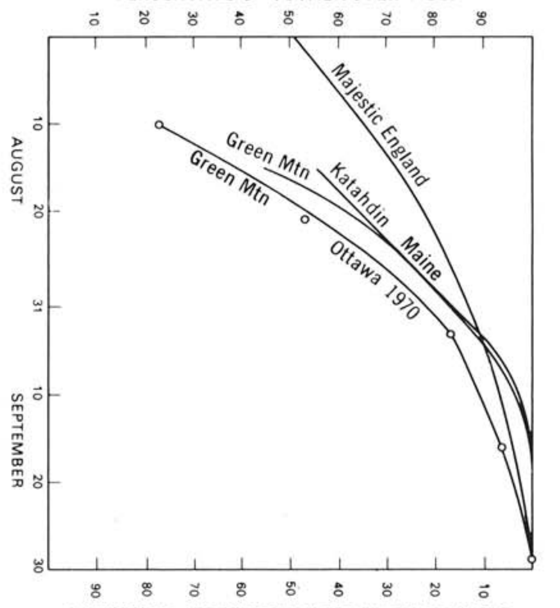

Fig. 3. Growth curves for tuber production in potato crops unaffected by late blight, showing estimated losses in tuber yield when the disease reaches 75% during August and September.

3% to copper toxicity and an additional 3% loss to wheel damage to the foliage. In our experiments we used Difolatan 4 Flowable, which was shown to be nonphytotoxic (6, 7, 8) and contains no nutritive element. No disease was present on either sprayed or unsprayed plots in the 1969 Ottawa trial, and there was no significant difference in the corresponding yields (Table 2). Wheel damage was eliminated by measuring the yield of only the center two rows, and driving the tractor through all plots whenever some plots were sprayed. At Ottawa, this type of injury was prevented by the use of a boom sprayer capable of spraying any plot in the experimental area from the perimeter. Yield results thus did not require the corrections that earlier workers found necessary (9). Data on total tuber yield have been used in evaluating the prediction method because they were the bases for the original calculation (9). However, late blight did affect both the quality and grade of tubers in our experiments. Some tubers from treatment A were blighted and were smaller than tubers from the other two treatments.

Van der Plank (19) gave samples of progress curves for late blight which were reasonably well described by transforming the data using $\log_{10} \frac{X}{1-X}$, but examples were reported in this paper where the fit is not so adequate. The above transformation is well suited for sigmoid curves, which may or may not be characteristic of an epidemic where the natural progress of late blight is affected by fungicide-spraying programs. In practice, most estimates on yield loss concern sprayed crops. Therefore, if the quantitative relationship between the amount of blight and loss in yield is to be determined more accurately, better characterization of the progress curves may be necessary.

The bulking curve produced for Green Mountain at Ottawa in 1970 is similar to the corresponding curve produced in Maine (Fig. 1), but differs markedly from the curve for Majestic in England (10). The success of the prediction method requires that a mean bulking curve must be obtained for the cultivar in the region for which loss assessments are to be made. Accordingly, the bulking curves for Green Mountain and Katahdin at Maine were used; these were suggested and used by Cox & Large (9) for this region. The calculations showed (Table 3) that the estimated loss is a gross underestimate of the actual loss in each case. The actual losses derived by weighing, 42, 33, 52, 17, and 26%, were in poor agreement with the estimated losses, 23, 10, 16, 3, and 0%, respectively, computed by the method. At Ottawa in 1970, it was possible to calculate the estimated loss using the bulking curve determined for that particular experiment, thereby ensuring that the characteristics of the bulking curve used exhibited the growth of the tubers in the crop exactly. However, the estimated loss was 0% calculated from these data, whereas the actual loss was 26%. Therefore, even when a bulking curve relating to a particular crop is used for calculating yield losses associated with the corresponding disease levels, the prediction method underestimates the loss. Furthermore, the actual loss in these experiments is based on a potential corresponding to treatment C, which had ca. 40% disease at the end of the season at both Fredericton and Charlottetown. Considering that the progress curve for treatment A, which only reached 68% disease at Ottawa in 1970, was associated with a 26% loss in yield, the potential yield used for treatment C at the other three trials is almost certainly an underestimate. Consequently, had it been possible to keep treatment C completely free from disease in all cases, the difference between estimated and actual losses would probably be greater than reported here.

The prediction method depends on the assumption that tuber production stops when 75% of the foliage is destroyed by blight. Any slight increase of tubers after this stage is offset by the slowing down of tuber growth before the 75% stage (9). However, the results of Radley et al. (16) for the cultivars, Majestic and Ulster Torch, suggest that tubers stop developing when 40 to 50% of the foliage is destroyed. According to Van der Plank (19), tuber production may not stop at any fixed percentage defoliation, but is probably related to the infection rate: the higher the value of infection rate, the higher percentage late blight one should take as a criterion for cessation of tuber production. The use of bulking curves from healthy rather than blighted crops will also lead to an underestimate of loss. Most of the predictions in the literature (9) are based upon comparisons between sprayed and unsprayed plots, where both progress curves show some degree of parallelism, and where they have exceeded 75% defoliation. In such predictions, the estimated losses in yield were calculated by deducting from the sprayed plot the yield that had accumulated between the time the disease level in the unsprayed plot exceeded 75% and that when the sprayed plots reached the same level. However, the estimated losses in yield calculated here are for situations where lower levels of disease are present in the sprayed plots, and where the progress curves for the two treatments do not display the same degree of parallelism reported in the literature. If the relationship between loss in yield and disease differs for various levels of disease, this could explain some of the divergence in results.

Because of the lack of agreement between estimated and actual losses, the method cannot be used to estimate yield losses in Canada. However, it emphasizes the need for better characterization of disease progress curves and their quantitative relationship to loss in tuber yield. The results also indicate that the losses in tuber yield attributed to late blight in sprayed potato crops in eastern Canada may have been underestimated. Similarly, the benefit of operating a successful spray program to keep late blight at a very low level may have been underrated.

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