The 1979 blue mold epidemic of flue-cured tobacco in Ontario and disease occurence in subsequent years[1]

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The incidence, spread, and severity as well as factors that contributed to the severity of the 1979 blue mold epidemic are documented. Losses in subsequent years were negligible. The origin of the 1979 epidemic in Ontario is discussed.

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L'incidence, l'btendu et la sévérité de même que les factaurs ayant contribubs à la sévérité de l'bpidbmie de moisissure bleue de 1979, sont documentbs dans cette article. Les pertes les années suivantes furent nbgligeables. L'origine de l'bpidbmie ontarienne de 1979 est discutbe.

Introduction

Downy mildew of tobacco, commonly known as blue mold, and caused by ***Peronospora tabacina*** Adam, was first recorded in Ontario in **1938.** The incidence and severity of the disease up to **1950** were well documented by Stover and Koch **(12).** From **1951** to **1966** blue mold which appeared sporadically in greenhouse seedbeds causing little economic loss **(3),** was considered a seedbed disease in both the U.S.A. and Canada **(8).** The disease was not recorded in Ontario from **1966** to **1978 (3).** In the present publication, the incidence and severity of the disease as well as the factors that influenced the epiphytotic are documented. The source of the causal pathogen ***P. tabacina*** in Ontario is also discussed.

The Progress and Severity of the Epidemic

In **1979,** blue mold caused by ***P. tabacina*** was first reported on **12** July in a field **3.2** km east of Tillsonburg, Oxford County, on tobacco plants started from container-grown seedlings (speedlings) imported from Florida. Yellow circular chlorotic spots, **12-20** mm in diameter, were observed on the adaxial surface of the lower leaves, with a corresponding greyish-blue downy growth of ***P. tabacina*** on the abaxial surface. On the same date and in the same field blue mold infection was also noticed on tobacco plants grown from seedlings produced by the grower in his own greenhouse. Plants from both sources were similar in size and color, but the blue mold lesions were fewer and smaller on the leaves of local plants. By 20 July, blue mold was reported on five more tobacco farms in the Haldimand-Norfolk Region, again on plants raised from Florida seedlings. The number of farms reporting the disease increased daily and by the end of July, blue mold was reported on hundreds of farms in the main tobacco area in Ontario (Table 1). Following the early appearance of blue mold symptoms on the lower leaves (Fig. 1), diseased spots coalesced forming large necrotic areas (Fig. 2). Gradually, leaf spots of ***P. tabacina*** appeared on the middle and even on the tip leaves of many tobacco plants at the end of the season.

Systemic infection symptoms were totally absent at mid-July in tobacco plants including those from imported seedlings. Early systemic infection causes stunting of the plant as well as twisting and malformation of leaves **(7).** However, another form of systemic infection was noticed in mature plants. The plants were not stunted at this stage, but the pathogen was able to move within the vascular tissue of the leaf (Figs. **3** and 4) and reached the stem. Accordingly, tobacco plants in severe cases of systemic infection were weakened and toppled by the wind or by the weight of their foliage (Fig. **5).** Necrosis of the vascular cylinder was evident when these plants were cut longitudinally (Fig. **6).** In Australia and the U.S.A. this type of systemic infection is referred to as "stem infection" (7, **9).** In August **1979,** it was common to notice twisting and puckering of the tip leaves of tobacco plants although such leaves were visually free from leaf infection (Fig. **7**). Similar symptoms were reported in France **(12).**

Factors that Favored the 1979 Epidemic

About **82%** of the Ontario flue-cured tobacco crop in **1979** was grown within a 50 km radius of Delhi. Approximately **75%** of the farms grew the cultivar Virginia 115; most of the remainder grew the cultivar Delhi **76.** Although both cultivars are susceptible to blue mold, symptoms on the latter were more advanced and sporulation of ***P. tabacina*** was more active on the larger leaves of this cultivar. Such concentration of farms growing cultivars susceptible to the disease provided the pathogen with a vast area of susceptible host, a factor that contributes to epidemics **(13).**

In Ontario, tobacco plants were young in early July and it is known that young, growing plants are susceptible to blue mold **(7).** Moreover, the tobacco plants were tender and therefore more susceptible to the pathogen **(14).**

Downy mildew epidemics including those of ***P. tabacina*** are influenced by weather conditions **(11, 12).** In Ontario, at the end of June **1979,** the weather was cloudy, cool, and humid (Fig. **8).** Although dry weather prevailed during July, the tobacco leaf was wet for several hours daily, with dew. Dew has been proven to provide sufficient moisture for the germination of conidia of maize downy mildew **(2).** Moreover, most growers sprinkle irrigated their fields during July, which probably assured additional moisture for conidial germination. Periods of

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cool nights were recorded between 18-22 July and 25-28 July (Fig.8). Thus weather conditions in July were favorable for blue mold infection and sporulation. The cool, cloudy weather in August was ideal for continued severity of the disease and random winds during the season contributed to the spread of the pathogen.

Sporulation of the pathogen was very active on tobacco leaves particularly the larger leaves of cv Delhi 76. It was common to notice sporulation of *P. tabacina* on both surfaces of the leaf. Since the pathogen can produce as many as one million conidia per square centimeter of sporulating leaf surface (8), spore load in the air in the main tobacco area was very high during the season. At the end of the season in mid-September, viable conidia were collected from lawns and foliage of trees; and during October floating conidia in the air gained entrance to a greenhouse and infected healthy tobacco seedlings.

In 1979, there were no fungicides registered in Canada to protect tobacco plants in the field from blue mold infection or to stop its spread, and tobacco companies made it clear that they would not accept any tobacco that had been treated with unregistered fungicides.

Table 1. The approximate accumulative number of tobacco farms that recorded blue mold in the main tobacco counties in Ontario at different periods during the 1979 epidemic.

Period between	Haldimand-Norfolk	Oxford	Brant	Elgin	Middlesex
10-20 July	5	1	0	0	0
21-29 July	15	10	0	3	0
30 July-13 Aug.	682	180	154	85	16
14-22 Aug.	822	201	164	190	17
23-30 Aug.	1048	210	184	241	61

Total: 1744 farms

Blue Mold in Ontario 1980-1984

In 1980, blue mold was active in certain tobacco areas in the U.S.A. causing considerable losses particularly in Georgia, Kentucky, North Carolina and Virginia (5). However, in 1980, there was no trace of blue mold in the main tobacco area in Ontario which had suffered badly from the 1979 epidemic. The disease was only reported on 5 August 1980 on one farm near Mount Brydges in Middlesex County and on a few farms 230 km to the east in Northumberland County (Fig. 10). The pattern of blue mold distribution in Ontario in 1979 (Fig. 9) was totally different from that in 1980 (Fig. 10). In those counties where the disease was recorded in 1980, tobacco growers did not adequately protect their crops with Ridomil sprays, whereas in the main tobacco area, growers were keen to protect their crops with the systemic fungicide. Fortunately crop losses were negligible in Ontario in 1980. In 1981, blue mold was reported in early August on one farm near Straffordville, Elgin County (6) and 3 farms in 1983 near Silver Hill, Haldimand-Norfolk Region, without measurable loss. In the 1982 and 1984 seasons, blue mold was not recorded in Ontario.

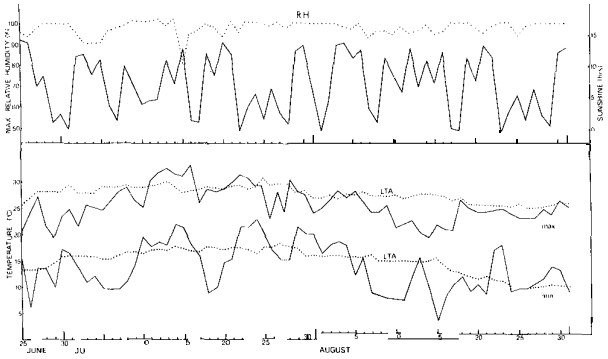

Figure 8. Maximum relative humidity, hours of sunshine, maximum and minimum temperature in the summer of 1979. (LTA = long term average).

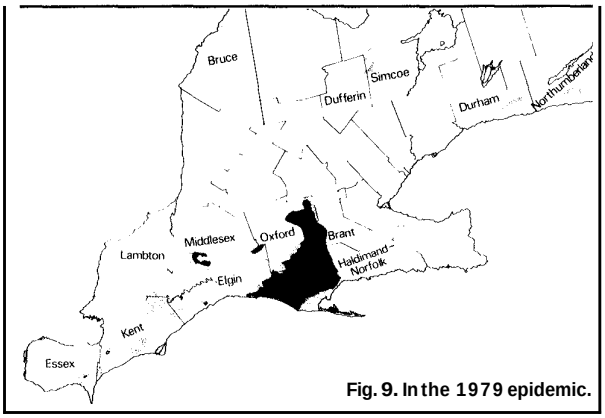

Fig. 9. In the 1979 epidemic.

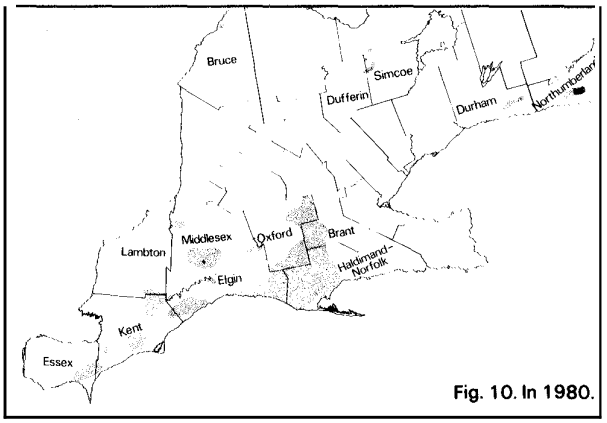

Fig. 10. In 1980.

Figures 9-10. Distribution of blue mold in the tobacco growing areas in Ontario. Dotted areas are the tobacco-growing areas, dark-shaded areas indicate the blue mold infected areas.

Discussion

For the past forty years, the source of P. tabacina infecting tobacco in Ontario has been a matter of speculation. It was believed that conidia from Kentucky and Ohio, carried by prevalent southwest wind, were the most logical source for infecting tobacco in Ontario (12). In the 1943 and 1944 seasons, it was reported that blue mold occurred in Ontario but not in Kentucky and Ohio (12). In both years, the nearest source for P. tabacina conidia was in Virginia, which the authors thought was too far away to be the source of spore showers that resulted in blue mold infection in Ontario. Although evidence was lacking, they attributed blue mold infection in both years to oospores of P. tabacina from previous crops which had overwintered in tobacco soil in Ontario (12).

Similarly in 1979, blue mold infection started in Ontario before Ohio and Kentucky (4). This probably lead tobacco specialists to believe that blue mold was introduced to Ontario on those seedlings imported from Florida and that the spread of the epidemic was mainly the result of these local infections (4).

The facts are against the theory that the disease was carried on the imported seedlings for the following reasons:

1. The lack of early systemic infections which are common in the seedling stage. The systemic infections in the 1979 epidemic were only on mature plants.

2. The rapid spread and the devastating nature of the epidemic cannot be logically attributed to spread of local infections. This observation supports the view that spore showers from U.S.A. were the source of the epidemic in Canada (7). This assumption was clarified when the Department of Environment was contacted and Bhartendu (1) computed backward air trajectories and indeed he speculated that conidia from Virginia had arrived in the main tobacco area in Ontario on 29 and 30 June 1979. Similarly, air-borne conidia form Virginia might have been the source of blue mold in 1943 and 1944. There is no evidence to demonstrate tobacco infection with oospores of *P.* tabacina. Even after the 1979 epidemic, all trials to infect tobacco with oospores were unsuccessful (10).

Still, a question remains unanswered: Why blue mold in 1979 in Ontario tended to show first on plants from imported tobacco seedlings as compared to those from local seedlings? According to the importers of the Florida seedlings, imported seedlings when received were much smaller than their own local seedlings and almost yellow in color as they had been raised in medium very poor in nitrogen. On July 12, on the farm where blue mold was first noticed in 1979, both plants from the grower's greenhouse and from imported seedlings were similar in size. This suggests that the imported plants were growing at a faster rate, probably as a result of having a better developed root system than those seedlings produced locally in greenhouse ground beds. There is evidence that rapidly growing plants develop more tender tissue which is susceptible to *P.* tabacina.

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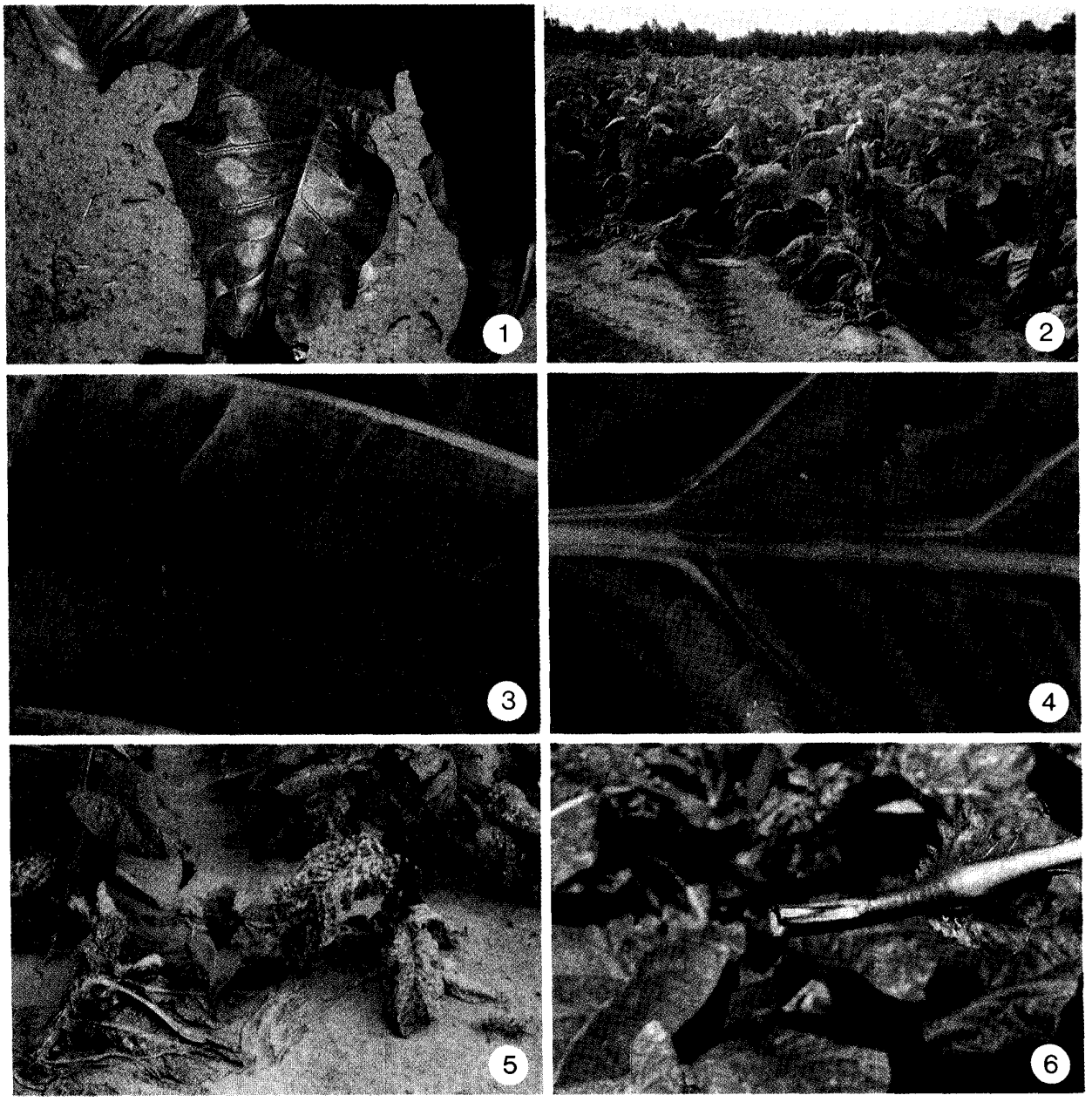

Figs. 1-7. Disease symptoms during the 1979 blue mold epidemic of tobacco in Ontario.

1. Yellow circular lesions on the adaxial surface of a lower tobacco leaf.
2. Field infected with blue mold showing large necrotic areas on the leaves.
3. Conidia of *P. tabacina* on the abaxial surface; the pathogen infected the vascular tissue of the leaf.
4. Midrib of a leaf infected with *P. tabacina*.
5. Weak and wind-toppled plants due to infection of the vascular tissue of the stem.
6. Stem of tobacco cut longitudinally to demonstrate necrosis of the vascular cylinder.
7. Puckered tobacco tip leaf.