# Factors Determining the Development of Wheat Spindle Streak Mosaic Caused by a Soil-Borne Virus in Ontario

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ABSTRACT

Wheat spindle streak mosaic, characterized by light green to yellow spindle-shaped dashes and short streaks on the younger leaves, and mosaic and necrosis on the older leaves of winter wheat in May and June, has been widespread most years since 1957 in fields in Ontario, where wheat is frequently grown. It is caused by a manually transmissible, soil-borne virus composed of slender, threadlike particles that contrast with the short rods of wheat (soil-borne) mosaic virus.

It has caused yield losses of 7-59% in experimental plots of winter wheat. All cultivars of *Triticum aestivum* and *T. durum* tested developed symptoms, but cultivars of *Secale cereale, Hordeum vulgare,* and *Avena sativa* did not.

Symptoms developed at 5-13 C on wheat grown in infective soil. Optimum conditions were 10 C with 1,000 ft-c of light 12 hr/day. Some plants developed symptoms in 31 days, but most required 60 days or more. In crop years when the disease was widespread and severe, there were more than 60 days with mean temperatures between 4.4 and 12.8 C (40-55 F).

The disease occurs on wheat grown on sandy to clay soils, but only after winter wheat has been grown several times in the same field. Soil has remained infective after 5 years either moist or dry in a greenhouse, unprotected outside during all seasons, or moist in a room at 8-12 C. Infectivity was retained in fractions of soil separated by a 0.044-mm sieve or by differential rates of sedimentation in water.

Infectivity in soil was eliminated by heating for 30 min at 52 C, drenching with solutions containing mercuric chloride, captan, ethyl alcohol, formalin, or 2,4,5-trichlorophenol, and by fumigating with preparations containing 1,3-dichloropropene and 1,2-dichloropropane, methyl bromide or sodium methyl dithiocarbamate at rates normally recommended for soil disinfection.

Fertilizers used at normally recommended rates did not reduce disease development, but the following amendments mixed with soil at the rates indicated eliminated or greatly reduced disease development: urea or uric acid at 0.5 g, ammonium nitrate at 5 g, chicken manure (without litter) at 10 g, turkey manure (with litter) at 50 g, and sucrose at 20 g/liter. Phytopathology 60:319-331.

Mosaic symptoms attributed to a soil-borne virus were first noticed on winter wheat in southern Ontario in 1957. Subsequent observations indicated variations in prevalence and severity in different districts and different years (15, 16, 18). In 1961, the disease was present almost universally on winter wheat in districts where wheat fields were most common, but was scarce or absent every year in fields where winter wheat was seldom grown.

Usually wheat that showed severe mosaic symptoms in early May appeared to be only mildly affected by mid-June, and at maturity produced high yields of good quality grain. From such observations, it was at first assumed that the disease was of little consequence, but observations and measurements made more recently indicate otherwise. The disease has caused a small but widespread reduction of yield most years in a high percentage of fields in areas where wheat is commonly grown, and in addition has caused severe reductions in yield in specific instances.

Mosaic diseases of wheat caused by soil-borne viruses have been recognized in the U.S.A. (8, 9, 13), Japan (6, 21), and Italy (1, 2). Comparative tests showed that the Ontario disease differed from the mosaic caused by wheat (soil-borne) mosaic virus (WMV) from soil collected in Illinois. Unlike the latter, the mosaic on wheat in Ontario includes distinct chlorotic and necrotic dashes and short streaks; it does not affect rye or barley (16), and it does not develop at temperatures above 15 C. The Ontario virus, like WMV, is manually transmissible, but the virus particles in dip preparations are slender and threadlike in contrast to the short, thick rods of WMV (Fig. 1). The Ontario virus is designated wheat spindle streak mosaic virus (WSSMV) to differentiate it from other soil-borne viruses of cereals. Symptoms described by McKinney (10) on wheat at Ithaca, New York, in 1946 and 1948, may have been caused by the same virus.

This paper reports characteristics of wheat spindle streak mosaic disease and factors affecting its occurrence and severity in Ontario.

MATERIALS AND METHODS.—Surveys for the distribution and severity of mosaic diseases in winter wheat in southern Ontario were usually made in early May when most of the wheat was less than 30 cm high, and sometimes in early June when the wheat was in the boot-to-heading stage. Farmers were consulted in special instances to determine the histories of fields with unusual presence or absence of disease. Samples were usually collected by digging diseased plants and placing the roots with adhering soil in polyethylene bags in which the soil could be kept moist. Sometimes plants were transplanted with field soil directly into wooden boxes (15 cm deep) and watered to keep the plants alive. After transport to the laboratory, the plants were placed in a growth room at 8-12 C if continued development of symptoms was desired.

Tests for soil transmission of the virus were done by

growing wheat in soil collected from fields where the disease was found. Symptoms did not develop in initial tests done at temperatures between 15 and 20 C, which are suitable for the development of wheat (soil-borne) mosaic (13). The first successful tests were done outside by sowing wheat in boxes of soil during late September or early October and leaving the plants exposed during the major part of the winter. In late February, the boxes were moved to a cool greenhouse (5-15 C), and symptoms were recorded in late April or early May. Most tests were done in a room with the temperature at 8 to 12 C and with about 1,000 ft-c of light 12 or 16 hr/day.

Little difference in disease development occurred at moisture levels that varied from just above the wilting point to saturation. Therefore, for most experiments the soils were kept moderately moist for optimum plant growth. At 10-day intervals, a complete fertilizer supplement (20-20-20) was included in the watering schedule.

Earlier tests (16) showed that high levels of infection occurred if one portion of highly infective soil was mixed with up to 15 portions of noninfective soil, or if a 1-cm layer of infective soil was deposited on top of noninfective potting soil mixture in which seeds were sown. Therefore, to conserve infective soil, tests were usually done with a layer of infective soil (3-4 cm) on top of a potting soil mixture.

For most manual transmission tests, leaves from diseased plants grown at 8-12 C were ground with distilled water (about 1 g:4 ml), Celite or 600-mesh Carborundum powder was added, and the mixture was rubbed on leaves of test plants in the two-leaf stage. The test plants were placed in a growth room at 8-12 C immediately after inoculation. Some plants usually developed symptoms in 4-8 weeks, but results were unreliable, indicating instability of infectivity. Neutral phosphate or borate buffers did not improve the infectivity of inoculum, hence water alone was used as diluent for most tests. Later, buffers at pH 8.5 to 9.0 were found to be beneficial. Most attempts to transmit the virus from plants collected in the field were unsuccessful, probably because most plants tested were too mature or had experienced too much warm weather to maintain high concentrations of stable infective virus. The virus has been transmitted readily from young plants collected when symptoms were most pronounced during cool weather in early May.

Most varieties of wheat tested were susceptible, but Kent winter wheat (*Triticum aestivum* L.), which is moderately resistant to mildew, was used as the standard test cultivar. Later, Ramsey (*T. durum* Desf.) was used in some manual and soil transmission experiments.

RESULTS.—*Symptoms in the field.*—In Ontario where winter wheat is sown in mid- to late September, symptoms of wheat spindle streak mosaic are not observed in the fall, but when growth resumes in the spring, infected crops appear yellowish brown in patches or over the entire field. This discoloration results from severe mosaic and necrosis of the earlier leaves. These leaves appear slightly rolled, narrower, and more erect than normal. They soon become hidden by new green leaves. The newest leaf on each shoot is usually symptomless until nearly fully expanded, especially if it develops during warm weather. Light green to yellow spindle-shaped dashes parallel to the leaf axis develop throughout the leaf blade or in areas anywhere from the tip to the base of the leaf. If conditions are favorable, the chlorotic markings increase, diffuse, and coalesce into a mosaic pattern that can be confused with wheat streak mosaic or *Agropyron* mosaic. Intensely chlorotic dashes and blotches become necrotic and may be mistaken for *Septoria* leaf blotch. Chlorotic to necrotic patches of tissue, light yellow to light brown in color, develop and extend from areas anywhere from near the

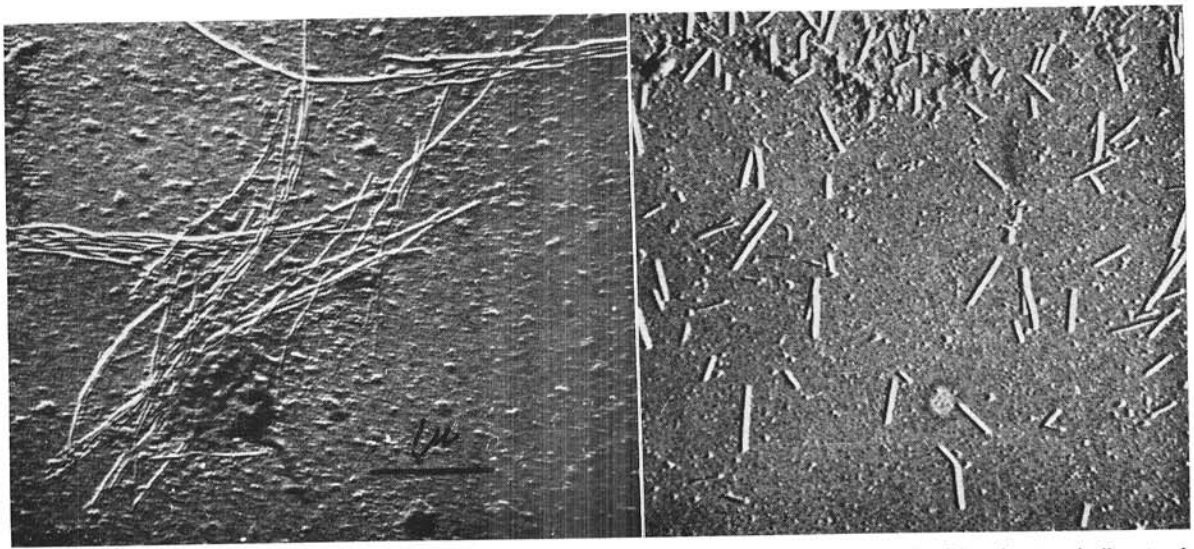

**Fig. 1.** Virus particles from dip preparations from Pawnee wheat plants (left) infected with wheat spindle streak mosaic virus from Ontario, and (right) with wheat (soil-borne) mosaic virus from Urbana, Illinois, at equal magnifications.

tip to the base of the leaf. In a typical symptom syndrome, on tillers in the six- to seven-leaf stage (Fig. 2, 3), the diseased plants are only slightly stunted, the youngest leaf is symptomless or has light green spindle-shaped streaks, the second youngest leaf has light green to yellow spindle-shaped dashes and short streaks throughout, and the third to fifth leaves have a patchy light green to yellow mosaic with spindle-shaped streaks of yellow in the green, or green in the yellow areas, and with streaks or blotches of necrotic tissue. The older leaves are largely or entirely necrotic. If warm summer temperatures develop quickly after mid-May, the milder chlorotic symptoms may disappear, and new leaves may fail to develop symptoms, leaving little evidence of the disease. However, if temperatures remain predominantly cool through May and early June, the mosaic symptoms chlorosis and necrosis may continue to develop on all leaves, and the affected plants become moderately stunted. After maturity, there are no foliage symptoms to distinguish diseased from

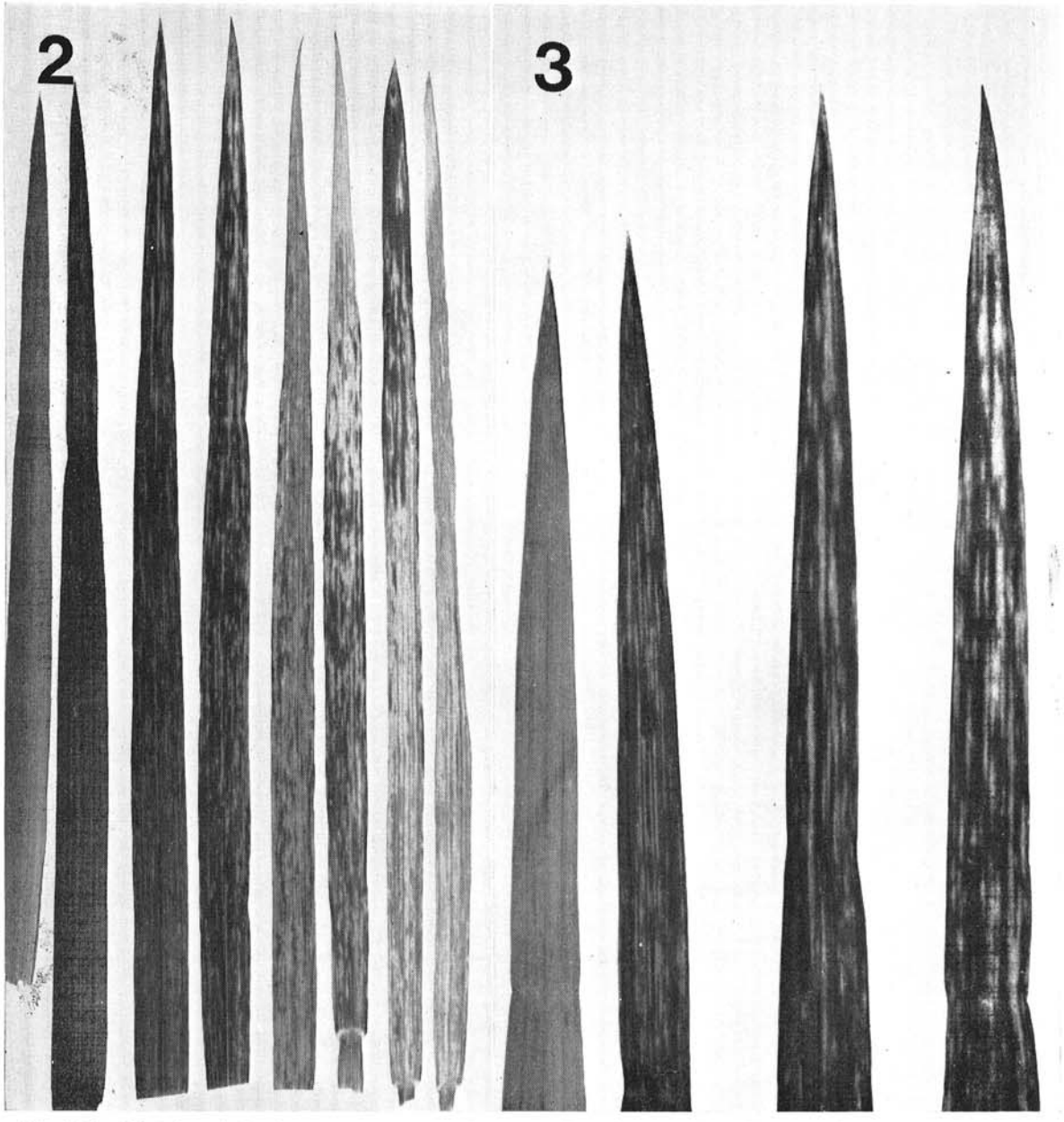


**Fig. 2-3.** **2)** Pairs of the four youngest leaves from two diseased plants. Note that one of the youngest leaves (left) is symptomless, the other has light green, spindle-shaped dashes near the tip. The second-youngest leaves have spindle-shaped dashes and streaks throughout; the third and fourth pairs have light yellow mosaic with dashes, blotches, and patchy areas of light-colored necrotic tissue. **3)** An enlargement of the tips of the pairs of the two youngest leaves from Fig. 2, showing the spindle-shaped chlorotic dashes characteristic of wheat spindle streak mosaic.

healthy plants, but comparisons made between plants that were marked before maturity have shown that the disease reduces the numbers and heights of tillers, the numbers of tillers that produce heads, and the numbers of florets that set seed.

*Symptom development in growth rooms.*—When wheat is sown in infective soil or inoculated by the leaf rub method and grown in a growth room at 8-12 C, a light green mottling including spots and dashes may appear on some plants in 4-8 weeks. Additional plants may develop symptoms for 2 or 3 months. As with plants observed in the field, the initial symptoms on subsequent new leaves include light green spindle-shaped dashes and streaks, followed by mottling including dashes or elongated blotches, some of which become yellow to light brown and necrotic. The leaves tend to roll adaxially sufficiently to cause them to appear narrower and more erect than normal. Older leaves become completely necrotic and light brown in color. Affected plants are stunted to varying degrees. At 3 months after manual inoculation, the infected plants in one experiment were 31% shorter, and the above-ground parts 68% lighter than healthy controls. Four months after seeding in infective soil, diseased plants were 23% shorter and 57% lighter than plants that escaped infection in the same soil.

The effect of higher temperatures on disease development was demonstrated with plants that became severely diseased at 8-12 C after manual inoculation or by growing in infective soil as described above. These were transplanted into outdoor plots in a sheltered location where temperatures were predominantly between 15 and 30 C. As the plants grew at the warmer temperature, symptoms diminished on younger leaves and new growth was symptomless. At maturity the diseased plants were equal to or taller than the disease-free plants, but plants that were infected from soil produced fewer tillers.

*Effects on yield.*—Farmers have seldom complained about yield losses attributable to the disease. In one field in which the disease appeared to be moderately severe in early May 1961, the symptoms were only mild in mid-June, and after harvest in July the farmer reported a yield of 60 bu/acre, which was considered an excellent yield for wheat in the area. Nevertheless, in 1968 severe mosaic symptoms were still evident in several fields in mid-June, and the farmers were concerned. In one field, the farmer reported a yield of 30 bu/acre. Normally he would have expected a yield of 40 to 60 bu/acre.

To measure the effects of spindle streak mosaic on the height and yield of wheat under field conditions, a stake 2 m long was placed in the center of each of six small patches of mosaic diseased wheat in a field in Huron County in early May 1963. Similar markers were placed in adjacent patches where little or no mosaic was evident. When the wheat was ripe, the average height of plants in the mosaic-free patches was 111.5 cm as compared with 96.0 cm in the mosaic-diseased areas, a reduction in height of 14%. The average grain yield in square yard areas (0.83 $m^2$) was 292 g for mosaic-free as compared with 172 g for the mosaic-diseased areas, a reduction of 41.2%.

Similar yield measurements made in four fields in Essex County by L. F. Gates, Research Station, Department of Agriculture, Harrow, Ontario, in 1967 showed yield losses of 7.1%, 10.4%, 12.8%, and 20.1%.

Possible errors attributable to variations in soil fertility in different patches selected for this type of comparison were avoided in measurements of loss made on wheat grown in a small plot of infective soil. Kent wheat was sown 27 October 1967, so late that only a few seedlings emerged before winter. By 15 May 1968, mosaic symptoms were evident on only about 5% of the plants, indicating that most plants had escaped infection because of late germination of seeds in the fall. In pairing diseased and symptomless plants, no allowance was made for stunting of growth that could have been caused by the disease up to this time. When the plants were mature, comparisons were made between 10 pairs of plants that were not damaged as a result of other causes. The diseased plants were 14.3% shorter than the symptomless plants; they had 40% fewer heads and produced 58.6% less grain.

*Hosts and varietal reactions.*—The susceptibilities of several varieties of wheat, rye, and barley were compared in plots sown in a field where the disease had occurred previously and in boxes of infectious soil. All seedings were done in late September or early October. After seeding, the boxes of soil were kept outdoors, where they were partially protected from sudden changes of temperature until early March; they were then moved into a small greenhouse with temperatures between 5 C and 15 C until the outside temperatures exceeded 15 C in May.

All wheat cultivars tested in the field and in soil boxes developed mosaic symptoms, but no symptoms developed on the winter barley (*Hordeum vulgare* L.) cultivars Hudson and Kenate, or on Horton rye (*Secale cereale* L.). Of the cultivars of wheat (*Triticum aestivum* L.) that have been grown in Ontario, Cornell, Dawbul, Genesee, Kent, Richmond, and Talbot developed moderate to severe mosaic symptoms and were moderately stunted, but few plants of Rideau developed symptoms, and these had milder symptoms than the other varieties. Symptoms also developed on Concho wheat selected for resistance to wheat (soil-borne) mosaic in Kansas, and on Pawnee, Bison, and Michigan Amber known to be susceptible to wheat (soil-borne) mosaic in Illinois, Kansas, and Nebraska (14).

Other experiments in which different cultivars of wheat were grown in infective soil kept in a growth room at 8-12 C showed that the winter wheat cultivars Minter, Nebred, Harvest Queen, Kharkov 22 M.C., and Winalta, and the hard red spring wheats Marquis, Thatcher, and Selkirk, were susceptible. In addition, the *T. durum* cultivar Ramsey was highly susceptible.

*Field surveys.*—Following the first observation of wheat spindle streak mosaic in a few fields in Kent County in June 1957, the disease was found during limited surveys in June 1958 in Kent, Huron, Perth, and Oxford Counties. More extensive surveys during

May in most subsequent years showed that the disease occurred in all areas examined where winter wheat was commonly grown in southern Ontario. In 1965, no symptoms of the disease were found in any area surveyed, but Essex and Kent Counties were not included in the survey. In all other years, symptoms were found, but varied from an average of 13.3% of the plants in all fields observed in 1964 to 65.1% in 1967 (Table 1, Fig. 4 and 5).

*Relationship of frequency of wheat-cropping to disease development.*—The disease was usually found in a higher percentage of fields in districts where fields of wheat were more numerous, regardless of the texture of the soil. Even in years in which the disease was prevalent, fields of disease-free wheat were sometimes found adjacent to fields in which all plants were diseased. When farmers were consulted about the histories of fields, it was always found that fields with the disease had grown wheat at 1 to 6 year intervals in rotation with other grains or forage crops. Usually any adjacent fields without the disease had grown alfalfa or forage grasses for several years, and had not grown a crop of wheat for 8 or more years. The disease has not been found in farmers' fields in Renfrew or Carleton Counties in eastern Ontario, where there are few fields of wheat and where wheat is seldom grown in the same field more than once in 10 years. However, in 1967 and 1968, plants with mild symptoms were found on the Central Experimental Farm, Ottawa, Carleton County, but only in plots in which winter wheat has been grown every 3 years for 12 or 15 years. These observations indicate that WSSMV may be harbored in the soil in most areas of Ontario where winter wheat is grown, but the disease does not develop unless wheat is grown frequently on the same land.

*Variations in infectivity from soil.*—All wheat plants were diseased in many of the fields examined most years. The most severe symptoms of WSSM were observed in fields in which wheat had been grown most frequently in recent years. When the infectivity of soils from different fields was tested by growing Kent wheat in pots of the soils at 8-12 C, usually the highest ratios of diseased plants occurred in soils from fields in which the disease symptoms were most severe. Such results indicate that the quantity of inoculum in the soil may affect severity as well as incidence of symptoms in the field, perhaps because the plants become infected more quickly when inoculum is abundant.

*Relation of temperature and light to the development of WSSM.*—In earlier tests, symptoms of wheat spindle streak mosaic did not develop on inoculated plants grown in infective soil at temperatures of 16 C or higher.

The effects of temperatures ranging from 2 to 15 C were tested in a series of six cabinets and rooms. Light, supplied by fluorescent tubes, varied in intensity from 220 ft-c at 2 C to 1,000 ft-c and 2,500 ft-c at 15 C. The plants were observed for disease development until 4 months after the date of inoculation or seeding (Table 2).

TABLE 1. Prevalence of winter wheat plants with spindle streak mosaic in fields surveyed in Ontario, 1958-1968

| Year | No. fields according to % diseased plants | | | | Total fields | Mean % infection |
|---|---|---|---|---|---|---|
| | No. symptoms | Tr. to 20% | 21 to 80% | 81 to 100% | | |
| 1958 | 3 | 2 | 1 | 2 | 8 | 31.2[a] |
| 1960 | 25 | 5 | 4 | 9 | 43 | 24.7 |
| 1961 | 25 | 6 | 7 | 68 | 106 | 61.6 |
| 1962 | 29 | 9 | 5 | 7 | 50 | 19.4 |
| 1963 | 5 | 13 | 6 | 15 | 39 | 45.6 |
| 1964 | 25 | 3 | 1 | 4 | 33 | 13.3 |
| 1965 | 24 | 0 | 0 | 0 | 24 | 0.0 |
| 1966 | 12 | 4 | 6 | 19 | 41 | 50.0 |
| 1967 | 19 | 8 | 26 | 87 | 140 | 65.1 |
| 1968 | 33 | 11 | 6 | 46 | 96 | 47.4 |

[a] Calculated by averaging the estimated percentages of diseased plants in all fields examined regardless of the sizes of the fields.

TABLE 2. Effects of temperature and light intensity on the development of spindle streak mosaic in Kent wheat inoculated by the leaf rub method or sown in infective soil

| Temp | Light | | Plants grown in noninfective soil | | | Plants grown in infective soil | |
|---|---|---|---|---|---|---|---|
| | | | Not inoculated | Inoculated manually | | | |
| | | | Dis/total | Dis/total | Incubation | Dis/total | Incubation |
| *C* | *ft-c* | *hr* | | | *days* | | *days* |
| 15 ± 1 | 2500 | 16 | 0/14 | 1/29 | 45 | 0/39 | |
| 15 ± 1 | 1000 | 16 | 0/12 | 5/20 | 45-78 | 0/41 | |
| 13 ± 1 | 600 | 16 | 0/15 | 3/30 | 55-78 | 24/50 | 56-97 |
| 10 ± 3 | 200 | 8 | 0/15 | 7/30 | 55-78 | 30/53 | 56-78 |
| 8 ± 3 | 1250 | 12 | 0/14 | 3/27 | 70-100 | 22/53 | 60-120 |
| 5 ± 1 | 600 | 12 | 0/15 | 4/31 | 90-120 | 2/49 | 85-97 |
| 2 ± 2 | 220 | 8 | 0/13 | 0/26 | | 0/51 | |

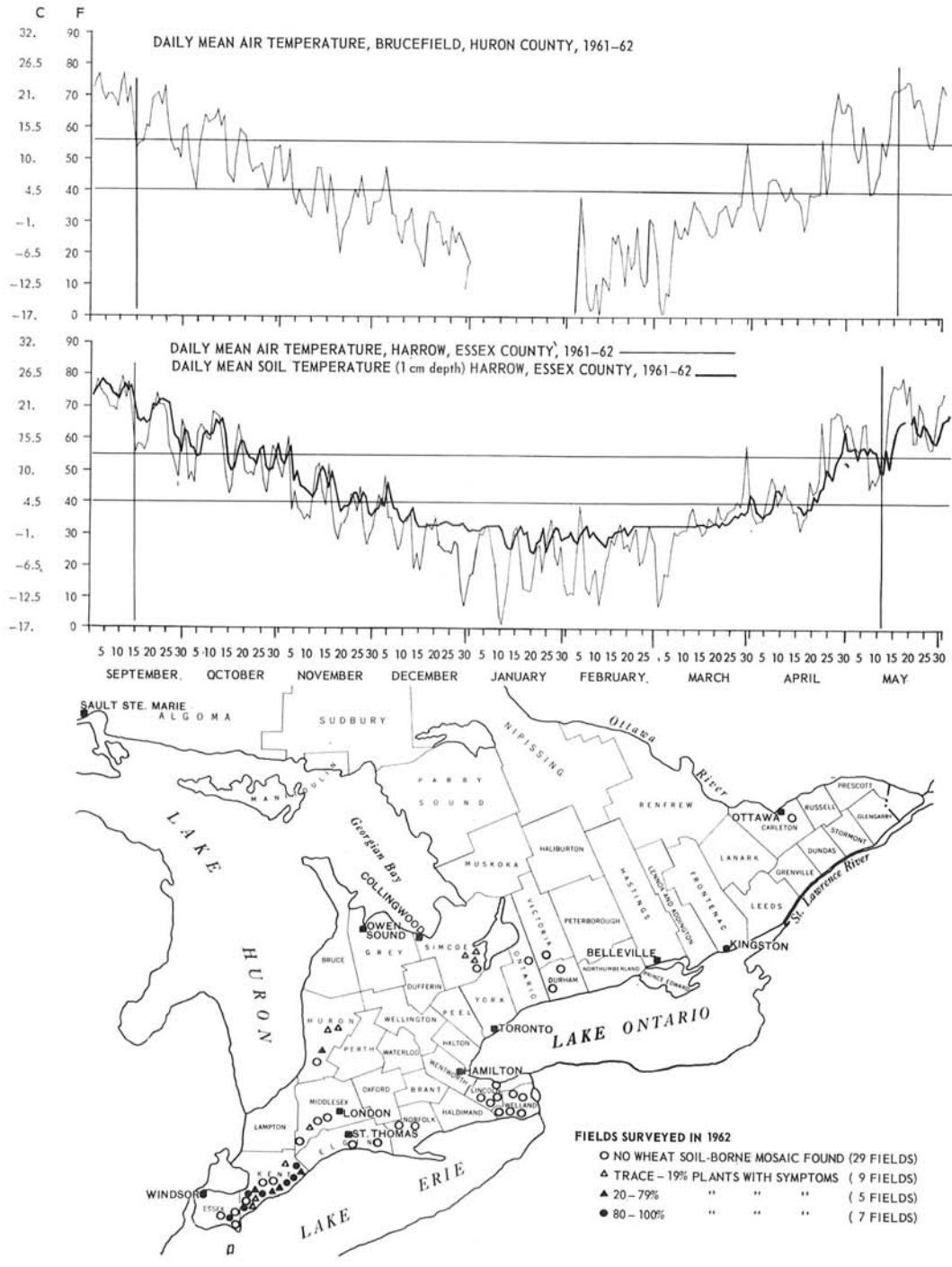


**Fig. 4.** Daily mean temperatures at locations in two wheat growing districts in Ontario from 15 Sep., about seeding time, to the survey date in May, in relation to the incidence of wheat spindle streak mosaic in the 1961-62 wheat crop. Note the period in the fall, and particularly the short period in the spring, with temperatures at 40-55 F (4.4-12.8 C), in relation to the low incidence of disease.

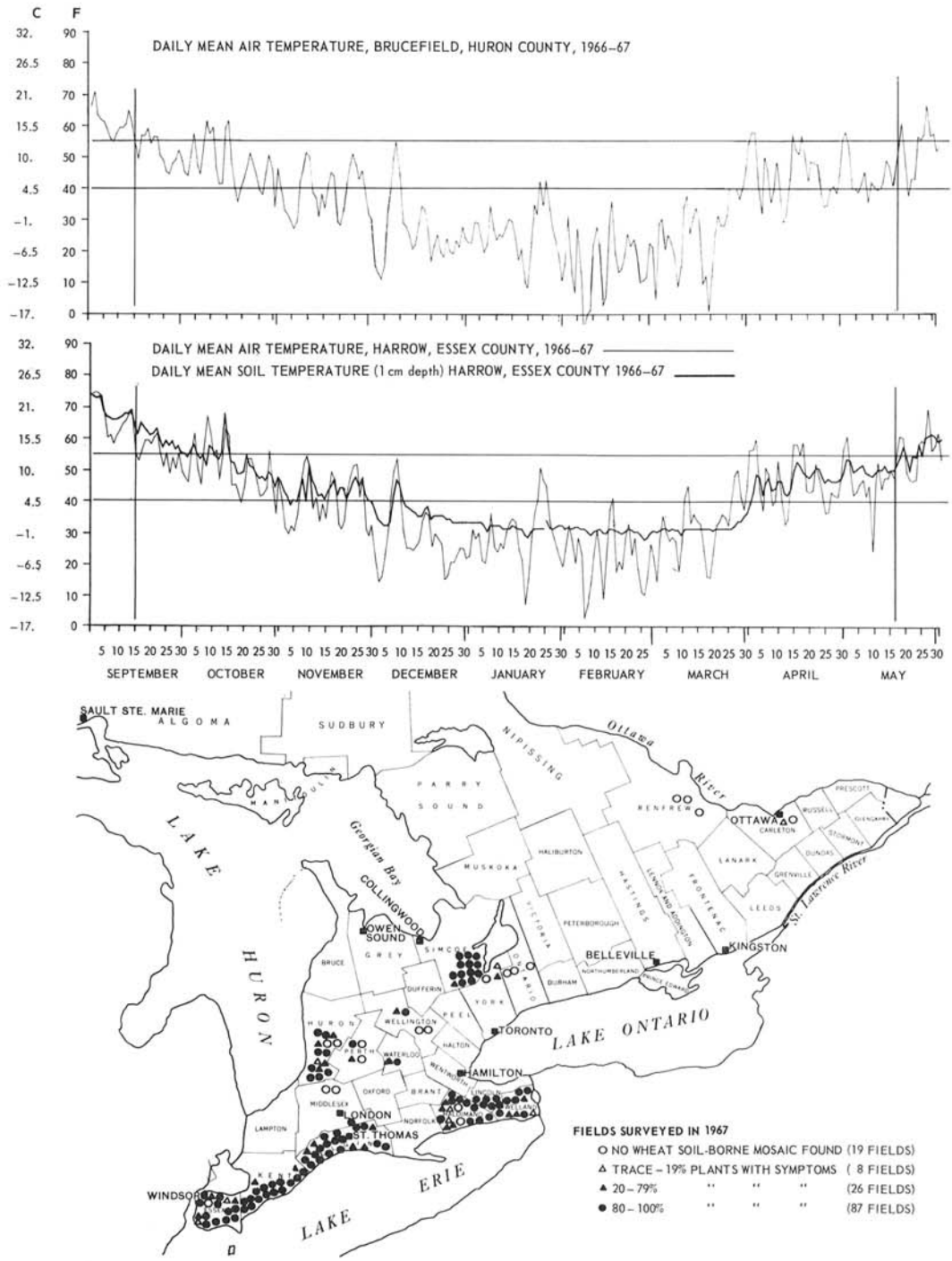


**Fig. 5.** Daily mean temperature at locations in two wheat-growing districts in Ontario from 15 Sep., about seeding time, to the survey date in May, in relation to the incidence of WSSM in the 1966-67 wheat crop. Note the relatively long periods in the fall, and particularly in the spring, with temperatures at 40-55 F (4.4-12.8 C), in relation to the high incidence of disease.

Some of the plants from seed sown in infective soil developed symptoms at temperatures of 5 to 13 C, but not at 15 C. Manually inoculated plants developed symptoms at 5-15 C. More plants developed symptoms after either method of inoculation when kept at 10 C than at higher or lower temperatures. Symptom development was faster at 10 and 13 C than at lower temperatures, but even at these temperatures, the incubation period for symptom development on plants varied from 45 to 97 days.

The effects of daylight-type fluorescent light at intensities of 500, 1,000, 1,500, and 2,000 ft-c for 12 hr/day on development of WSSM on plants grown from seed in infective soil were compared in cabinets regulated at 10 ± 1 C. Symptoms developed in 31 to about 104 days under all light intensities tested, but more plants developed symptoms at 500 and 1,000 ft-c (51/257 and 58/277) than at 1,500 and 2,000 ft-c (36/269 and 33/258). At 500 ft-c, the plants tended to be spindly, and developed severe mildew infection. At 1,000 ft-c, the symptoms on plants infected either manually or naturally were most distinct and plant growth was optimum, hence this light intensity appeared to be optimum for disease development at 10 C.

The effects on symptom development of soil temperatures of 5, 10, 15, and 20 C were tested in Wisconsin-type temperature tanks in a room with air temperature regulated at 10 C and light at 1,000 ft-c for 12 hr/day (Table 3).

At all temperatures tested, symptoms developed on some of the plants grown from seed sown in infective soil. Since this was the first time symptoms were observed on plants grown in infective soil at 15 or 20 C, the 10-C air temperature must have favored symptom development. On manually inoculated plants, symptoms developed at soil temperatures of 5, 10, and 15 C, but not at 20 C. However, percentage infection was low at all temperatures, indicating that the inoculum was not highly infective.

As indicated by these experiments, different percentages of plants became infected at different temperatures from 5 to 13 C, but some plants escaped infection at each temperature. However, in several experiments, all plants developed symptoms when grown in infective soil in the winter in a greenhouse in which the temperature was usually 8-12 C, but fluctuated from about 0 to 18 C. Although a prolonged exposure to temperatures between 5-13 C is necessary for symptom development, it is possible that fluctuations in temperature may play a major role in infection and disease development.

TABLE 3. Effect of soil temperature at a constant air temperature of 10 C on the development of wheat spindle streak mosaic in Kent wheat inoculated by the leaf rub method or grown in infective soil

| | Plants grown in noninfective soil | | |
|---|---|---|---|
| Soil Temp. | Not inoculated (Dis/total) | Inoculated manually (Dis/total) | Plants grown in infective soil (Dis/total) |
| *C* | | | |
| 20 ± 1 | | 0/29 | 1/39 |
| 15 ± 1 | 0/19 | 1/31 | 9/49 |
| 10 ± 1 | 0/21 | 2/29 | 32/55 |
| 5 ± 1 | 0/13 | 3/24 | 11/57 |

*Correlation of seasonal temperatures with disease incidence.*—Temperature records were examined to determine if there were more days with mean temperatures in the critical range of 5-13 C between seeding time (about 15 September) and the survey date in May in years when the disease was prevalent and severe, than in years when it was scarce and mild or absent. Since the official records are in Fahrenheit, the temperature range 40-55 F (4.4-12.8 C) was chosen. The records examined were of daily mean air-temperatures at Harrow for the Essex and Kent County area, at Brucefield for Huron County, at Bradford or Newmarket for the Simcoe and northern York County area, and at Welland for Welland County. Isolated days with temperatures of 4.4 C or higher between 15 December and 15 February were not included because the soil remained too cool for plant growth. Since WSSMV is soil-borne, the records of soil temperature at about seeding depth (3-5 cm) would have been preferred, but soil temperature records were available only for 1 and 10-cm depths, and these only for the Harrow Station. Records for the 1-cm depth at Harrow were used for comparison with air temperatures. Comparisons of the weather records and survey data for several years (Fig. 4, 5, Table 4) show that the daily mean air temperature was in the 4.4 to 12.8 C range for 30 to 53 days in the autumn, then for an additional 15 to 38 days during the spring period prior to the survey dates in May. The total time in the critical range for the two periods was 65 or more days at all locations in the crop seasons of 1960-1961, 1966-1967, and 1967-1968, when disease development was prevalent in all areas. Air temperature was in the critical range for only 51 to 62 days in the 1961-62 and 1964-65 crop seasons, when disease development was light or absent in most areas. Records at the Harrow Station show that in the years selected, the mean soil temperature was in the 4.4-12.8 C range for more days than was the mean air temperature, but the contrast between years of disease scarcity and of disease abundance were greater. In the 1961-62 crop year, when there was little disease, the soil temperatures were in the critical range for 62 days as compared with 84 to 102 days in the years when the disease was prevalent in Essex and Kent Counties.

Although there had already been 46 days with mean soil temperatures in the 4.4 to 12.8 C range in the fall of 1967 at Harrow, symptoms of WSSM were not found when fields in the area were examined by L. F. Gates about 10 November. However, severe symptoms were prevalent in many of the same fields the following May. Wheat sown in infective soil so late in the fall of 1967 that plants did not emerge before winter did not develop symptoms the following spring. These observations indicate that the symptoms seen in the spring result from infection in the fall. If plants become infected in the spring, they do not have time to develop

TABLE 4. Numbers of days with mean temperatures between 40 F and 55 F in relation to the incidence of wheat spindle streak mosaic in different areas of southern Ontario in different years

| Temperature recorded at | Days with mean temp. 40-55 F (4.4-12.8 C) | | | | | | Field Surveys | | |
|---|---|---|---|---|---|---|---|---|---|
| | 15 Sep. to winter | | Winter to survey date | | Total 15 Sep. to survey date | | | | |
| | Air | Soil[a] | Air | Soil | Air | Soil | Date | Counties | % Disease |
| 1960-61 crop year | | | | | | | *May* | | |
| Harrow | 37 | 41 | 35 | 43 | 72 | 84 | 11 | Essex, Kent | 77.7 |
| Brucefield | 44 | | 26 | | 70 | | 10 | Huron | 82.7 |
| Bradford | 43 | | 24 | | 67 | | 9 | Simcoe, York | 48.9 |
| 1961-62 crop year | | | | | | | | | |
| Harrow | 30 | 39 | 21 | 23 | 51 | 62 | 16 | Essex, Kent | 22.5 |
| Brucefield | 36 | | 15 | | 51 | | 16 | Huron | 17.5 |
| Bradford | 34 | | 22 | | 56 | | 15 | Simcoe, York | 7.5 |
| Welland | 32 | | 23 | | 55 | | 17 | Welland | 0.0 |
| 1964-65 crop year | | | | | | | | | |
| Brucefield | 42 | | 20 | | 62 | | 17 | Huron | 0.0 |
| Newmarket | 35 | | 18 | | 53 | | 18 | Simcoe, York | 0.0 |
| 1966-67 crop year | | | | | | | | | |
| Harrow | 47 | 53 | 38 | 49 | 85 | 102 | 17 | Essex, Kent | 66.4 |
| Brucefield | 50 | | 37 | | 87 | | 18 | Huron | 58.3 |
| Newmarket | 53 | | 29 | | 82 | | 18 | Simcoe, York | 75.0 |
| Welland | 51 | | 34 | | 85 | | 16 | Welland | 65.2 |
| 1967-68 crop year | | | | | | | | | |
| Harrow | 48 | 46 | 34 | 38 | 82 | 84 | 8 | Essex, Kent | 52.9 |
| Brucefield | 37 | | 31 | | 68 | | 8 | Huron | 56.0 |
| Newmarket | 34 | | 31 | | 65 | | 9 | Simcoe, York | 45.3 |
| Welland | 46 | | 36 | | 82 | | 7 | Welland | 0.0 |

[a] Soil temperatures were measured at a depth of 1 cm.

symptoms before temperatures rise above the critical range.

*Longevity of infectivity of soil stored under different conditions.*—To determine conditions favorable for storage of infective field soil, portions of a composite mixture of soil collected from several fields in southwestern Ontario in May 1963 were placed in boxes stored under different conditions including unsheltered outside, moist in a cool room (8-12 C), and either moist or dry in a greenhouse.

In October each year, samples of soil from each storage location were tested for infectivity by sowing Kent wheat in four 12-cm pots of each of the four lots of soil. After 4 months at 8-12 C in the growth room, counts of infected plants were recorded (Table 5). In spite of considerable variations among treatments and replicates, all samples remained infective for the 5 years of the test. The sample with the most consistently high infectivity was that stored dry in the warm conditions of the greenhouse.

*Infectivity of different physical fractions of field soil.*—Wheat spindle streak mosaic occurs in wheat grown in soils with textures varying from clay to sand. To determine if infection was associated only with certain particles in the soil, infective loam soil was passed through sieves with openings of 0.86-mm, 0.28-mm, 0.14-mm, 0.074-mm, and 0.044-mm sizes. Soil that passed each sieve was tested for infectivity by placing a 3-cm layer of the soil on top of noninfective potting soil in clay pots, then seeding wheat and maintaining the pots at standard conditions in the growth room. All fractions of soil, including that which passed through the 0.044-mm sieve, were infective.

In further tests, four 50-g portions of soil that had

TABLE 5. Retention of wheat spindle streak mosaic infectivity by field soil after different periods of storage under different conditions

| Date Kent wheat sown for test | Years stored | Storage conditions | | | |
|---|---|---|---|---|---|
| | | Outside at Ottawa, all seasons | Moist in growth room at 10 ± 2 C | Greenhouse 22 ± 8 C | |
| | | | | Moist | Dry |
| 1 Oct. 1963 | 0 | 87.7[a] | | | |
| 20 Oct. 1964 | 1 | 34.6 | 0.0 | 68.1 | 61.2 |
| 5 Oct. 1965 | 2 | 8.2 | 23.2 | 9.4 | 24.5 |
| 2 Dec. 1966 | 3 | 1.2 | 21.1 | 70.6 | 63.1 |
| 16 Oct. 1967 | 4 | 7.8 | 22.0 | 39.7 | 50.9 |
| 18 Oct. 1968 | 5 | 31.6 | 62.7 | 10.8 | 20.8 |

[a] % Infected out of 50 to 85 Kent wheat plants grown at 8-12 C for about 4 months after seeding. Plants similarly grown as controls in normal potting soil preparations did not develop symptoms.

passed through the sieve with 0.044-mm pore size were separated into sand, silt, and clay fractions by rate of sedimentation in water (3). The mean wt of the fractions from each 50-g portion was 19.16 g sand, 23.15 g silt, and 7.69 g clay. Each fraction from each portion of soil was mixed with the top 3-cm portion of noninfective potting soil mixture in a 12-cm clay pot. Wheat was grown in the soil mixtures to test for infectivity. The total numbers of plants that developed infection in association with the soil fractions were 12/40 with the sand, 6/31 with the silt, 11/41 with the clay, 7/20 with nonfractionated diseased soil, and 0/20 with noninfective potting soil. Infectivity was carried about equally with all fractions tested. It appears that infectivity is associated with clay particles or with organic material that was not separated from any of the fractions by the procedures used.

*Disinfection of soil.*—The effects of selected chemicals and heat on infectivity were tested on a mixture of soils from infected fields. The soil was passed through a 5-mm sieve, thoroughly mixed in a slightly moist condition, and treated in 10-kg lots. Most chemicals were mixed undiluted with the soil, but some were diluted in 2 liters of water and added as a drench. Chemicals were used at rates about equal to and at four times the commercially recommended rates. After treatment with such chemicals, the soil lots were enclosed in polyethylene bags for 1 week, then exposed for aeration and drying for another week. For the heat treatments, each lot of soil in a flask was slowly heated in a water bath and stirred until the entire sample reached the required temperature. It was maintained at this temperature for 30 min, then spread thinly in a pan to cool at room temperature and exposed to the air for 1 week. To test for infectivity, each lot of treated soil was placed on top of 7 cm of noninfective potting soil in a box 15 cm deep by 40 cm square. About 80 seeds of wheat were sown in each, and also in noninfective soil, and the boxes were kept in the required cool growth conditions for development of disease symptoms.

Infectivity was eliminated in soil heated for 30 min at 52 C or higher, but not at 45 C. It was also eliminated by treatments containing mercuric chloride, captan, ethyl alcohol, formaldehyde, Nurelle, methyl bromide, DD, and Vapam (Table 6). The treatments containing ethylene dibromide, No Damp, and the insecticides dieldrin, lindane, and Zectran were not effective at the rates applied.

Without having data on the effects of the above treatments on the virus, and knowing that each of the treatments may affect nematodes as well as fungi and other organisms that could function as vectors, the results do not give a specific indication of the nature of a possible vector in the soil. However, the similarity

TABLE 6. Elimination of wheat spindle streak mosaic virus infection in soil by treatment with chemicals

| Treatment for 10 kg of infected soil | | Plants Infected/total |
|---|---|---|
| No treatment | | 25/65 |
| Drenched with 2 liters aqueous solution containing | | |
| Water only | | 16/41 |
| Calochlor [60% mercurous chloride + 30% mercuric chloride] | 2.5 g | 0/48 |
| Calochlor [60% mercurous chloride + 30% mercuric chloride] | 10.0 g | 0/68 |
| Captan, 50-W [*N*-([trichloromethyl]thio)-4-cyclohexene-1,2-dicarboximide 50%, WP] | 5.0 g | 7/78 |
| Captan, 50-W [*N*-([trichloromethyl]thio)-4-cyclohexene-1,2-dicarboximide 50%, WP] | 20.0 g | 0/76 |
| Ethyl alcohol | 30% | 31/53 |
| Ethyl alcohol | 95% | 0/21 |
| Formalin [40% formaldehyde] | 2% | 0/59 |
| Formalin [40% formaldehyde] | 8% | 0/15 |
| No Damp [8-hydroxyquinoline benzoate, 2.5%] | 20 ml | 9/57 |
| No Damp [8-hydroxyquinoline benzoate, 2.5%] | 80 ml | 34/76 |
| Nurelle [2,4,5 trichlorophenol, 18.2%] | 5.0 g | 0/45 |
| Nematicides and fungicides applied undiluted at the rate of: | | |
| D.D. [1,3 dichloropropene + 1,2-dichloropropane] | 1 ml | 37/61 |
| D.D. [1,3 dichloropropene + 1,2-dichloropropane] | 4 ml | 0/82 |
| Telone [1,3 dichloropropene + related $C_3$ hydrocarbons] | 1 ml | 23/72 |
| Telone [1,3 dichloropropene + related $C_3$ hydrocarbons] | 4 ml | 0/70 |
| Ethylene dibromide | 0.9 ml | 16/53 |
| Ethylene dibromide | 3.6 ml | 25/67 |
| Methyl bromide | 3.0 ml | 0/66 |
| Methyl bromide | 12.0 ml | 0/64 |
| Vapam [sodium methyl dithiocarbamate, 31%] | 4.0 ml | 0/68 |
| Vapam [sodium methyl dithiocarbamate, 31%] | 16.0 ml | 0/70 |
| Insecticides applied undiluted | | |
| Dieldrin [endo, exo-1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1, 4:5,8-dimethanonaphthalene 85%] | 0.5 g | 42/72 |
| Dieldrin [endo, exo-1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1, 4:5,8-dimethanonaphthalene 85%] | 2.0 g | 38/76 |
| Lindane [γ-1,2,3,4,5,6-hexachlorocyclohexane 99%] | 0.08 g | 46/77 |
| Lindane [γ-1,2,3,4,5,6-hexachlorocyclohexane 99%] | 0.32 g | 8/65 |
| Zectran [4-dimethylamino-3-5-xylyl-*n*-methylcarbamate] | 0.5 g | 12/67 |
| Zectran [4-dimethylamino-3-5-xylyl-*n*-methylcarbamate] | 2.0 g | 30/75 |
| Control (noninfective potting soil mixture only) | | 0/69 |

in effects of several chemicals used in these tests and in tests reported with soil infested with wheat mosaic and oat mosaic viruses in the U.S.A. (7, 11) suggest that similar agents may be involved in the transmission of the three viruses.

Two of the chemicals most effective in the initial tests, Telone (1,3 dichloropropene + related $C_3$ hydrocarbons) at 30 liters/hectare and metasodium (sodium methyldithiocarbamate 31%) at 120 liters/hectare were tested in replicated plots of naturally infective soil near Clinton, Ontario, but neither treatment reduced infection.

*Effects of fertilizers and other amendments on soil infectivity.*—There has been no indication that normal use of commercial fertilizers reduces the incidence of disease in farm fields. Similarly, several fertilizers and soil amendments mixed with infective soils have increased plant growth without reducing the percentage of plants infected even when used at high rates. These include superphosphate (20% phosphate) at 1.2 g and 2.4 g, potassium sulphate (50% potash) at 0.6 g and 1.2 g, 5-10-10 fertilizer at 2 g, 20-20-20 fertilizer at 2 g, ammonium sulphate at 2 g, ground limestone at 100 g, lime at 5 g/liter of soil, and peat moss at 1:3 by volume. However, some amendments used at high rates have eliminated infectivity. These include bloodmeal at 4 g/liter but not at 2.5 g, urea at 2 g, and sucrose at 50 g and 20 g but not at 5 g/liter.

In 1968, several fields were observed in Simcoe and Essex Counties in which applications of turkey manure at about 8-12 tons/acre appeared to have eliminated the development of WSSM. The disease was present in other areas of the same fields where less manure had been applied. In other fields, inconsistent effects were associated with applications of other animal manures. The effects of five types of manure were therefore compared with the effects of several nitrogen-containing chemicals. Fresh, litter-free chicken, horse, cow, and sheep manure, and dry turkey manure including litter, were used at the rates of 10 g and 50 g/liter of infective soil in pot tests under controlled conditions. The chemicals, including Uramite (urea formaldehyde), urea, uric acid, and ammonium nitrate were used at 0.5 g and 5 g/liter of soil. After treatment, the pots of soil were kept at 18-25 C for 1 week; then wheat was sown and the pots were placed in the growth room at 8-12 C for disease development (Table 7). Disease incidence was increased in soil amended with either dosage of cow, horse, or sheep manure or the smaller dosage of Uramite. It was decreased with the lower dosages of chicken manure or uric acid or the higher dosage of Uramite and eliminated in soil amended with the higher dosages of chicken or turkey manure, ammonium nitrate, or uric acid or with either dosage of urea. Correlation of these results with the quantities of N in the treatments shows that increases in disease were generally associated with the smaller additions of N, while reductions in disease were associated with the larger additions of N.

DISCUSSION.—When the symptoms of WSSM were first observed on winter wheat in southern Ontario, the characteristics of the symptoms and circumstances of their occurrence immediately suggested that the disease was caused by a soil-borne virus. No intensive effort was made to study other pathogens, because there were no consistent clues to incriminate anything else as a primary causal agent. Initial attempts to transmit a virus manually and by growing plants in infective soil failed, but later attempts were successful once the requirement for low temperatures was realized.

The disease resembled wheat (soil-borne) mosaic in many respects, including factors related to soil infectivity and disease development. The presence of spindle-shaped markings in early phases of symptom development on most leaves, failure to develop on rye or barley, and failure to develop at temperatures above 15 C distinguished the Ontario disease from wheat mosaic, but such differences could reflect merely a strain difference. However, the slender, sometimes threadlike particles found with difficulty in dip preparations from plants with the Ontario disease were so different from the short, thick rods so readily found in plants infected with

TABLE 7. Incidence of spindle streak mosaic in wheat grown in infective soil treated with manures and nitrogen chemicals

| Manures applied to soil | Rate of application/liter of soil | | | |
|---|---|---|---|---|
| | 10 g | | 50 g | |
| | *g N* | *% mosaic* | *g N* | *% mosaic* |
| Chicken | 0.11[a] | 3.4[b] | 0.55 | 0.0 |
| Turkey | 0.22 | 19.2 | 1.12 | 0.0 |
| Cow | 0.06 | 24.0 | 0.32 | 48.5 |
| Horse | 0.07 | 43.7 | 0.35 | 24.1 |
| Sheep | 0.10 | 35.5 | 0.50 | 41.9 |
| Nitrogen chemicals applied to soil | *0.5 g* | | *5.0 g* | |
| Ammonium nitrate | 0.16 | 17.2 | 1.66 | 0.0 |
| Uramite (urea formaldehyde) | 0.23 | 56.6 | 2.30 | 10.3 |
| Urea | 0.23 | 0.0 | 2.33 | 0.0 |
| Uric acid | 0.16 | 3.3 | 1.66 | 0.0 |
| Nontreated control, 15.8% | | | | |

[a] Nitrogen content of manures from samples analysed by Animal Research Institute, Canada Department of Agriculture, Ottawa.

[b] % Infected out of 25 to 33 Kent wheat plants.

wheat mosaic virus that it was concluded that the virus was distinct.

Wheat spindle streak mosaic is one of three mosaic diseases recognized on wheat in Ontario. *Agropyron* mosaic, which was also recognized in Ontario in 1957, is widely distributed on *Agropyron repens* and sometimes develops on wheat growing near patches of *A. repens,* but its incidence on wheat has been very low and its effects mild (17). Wheat streak mosaic, destructive in the plains states, Alberta, and since 1965 in neighboring Ohio (22) and Michigan, was not detected in Ontario until 1964 (12, 18, 19). It has been found only in a few fields in Middlesex, Kent, and Essex Counties, where its perpetuation appears to depend on an association between maize and wheat.

Wheat spindle streak mosaic has been the most consistently recurring and prevalent of the three diseases. Yield loss measurements indicate that it causes substantial losses in some circumstances. Its phenomenal prevalence in some years throughout the areas of most intensive wheat production, and the occasional known examples of severe effects on yield, demonstrate that this disease causes a significant reduction in wheat production in Ontario. There have probably been instances in which WSSMV has caused severe losses that have been attributed to other factors. Certain changes in agricultural practice, or weather conditions particularly favorable for the disease, could accentuate its destructiveness.

Temperature appears to be the main climatic variable affecting the development of WSSM in wheat grown in infected fields. The usual requirement of at least 60 days at temperatures between 5 and 13 C makes the development of the disease on spring wheat in Ontario unlikely, because after seeding in late April or early May, the required temperatures for disease development do not persist long enough. Winter wheat, on the other hand, is exposed to suitable temperatures for periods in the fall and in the spring which usually total 60 days or more. If the seeding date was late enough in the fall to minimize exposure to the critical temperatures, however, symptoms might not develop in the spring, because the minimum incubation period would not be completed before the warm weather of late May and early June.

Symptoms of WSSM are suppressed at temperatures above 15 C. Plants that were severely diseased after 3-4 months at 8-12 C recovered when moved to summer temperatures (15-30 C). In the field, wheat that appears severely diseased in early May usually recovers remarkably during the warmer weather of late May and early June, but it still produces fewer heads and less grain than healthy wheat. The severe mosaic and necrosis occasionally observed at heading time depends on continued cool temperatures (about 15 C or lower) during late May and early June or until the plants are headed. Early maturing varieties that complete most of their vegetative growth during the earlier, cooler part of the season may be more severely damaged than equally susceptible late-maturing varieties which complete development in the later, warmer part of the season.

A striking feature of the distribution of WSSM is its almost universal presence in some years throughout wheat fields that have grown several previous crops of winter wheat, and its absence in fields that have not grown wheat for many years. The disease has not been found in farmers' fields in the Ottawa Valley where wheat is seldom grown in the same field twice in 10 years. However, at Ottawa it has been found in experimental plots in which wheat has been grown every 3rd year. Other examples indicate that the disease is more severe in fields that have grown wheat frequently, especially if wheat was grown the previous year. These examples indicate that the virus may be present in most agricultural soils in southern Ontario, but it remains latent until wheat has been grown several times on the same land.

A high level of infectivity still evident in soil stored under different conditions for 5 years discourages prospects for eliminating infection in soil simply by increasing the intervals between wheat crops. However, since the most severe damage from the disease has been observed on wheat in fields in which wheat was grown the preceding year, it is logical to suggest that wheat should not be grown on the same land in successive years. In years when the disease was prevalent, the only wheat free from the disease was in fields that had grown alfalfa or pasture grasses for 8 or more years since the last crop of wheat, or in fields that had received heavy applications of poultry manure.

Experiments with pots of infective soil have confirmed an effect of poultry manure, and have shown that heavy applications of other substances high in nitrogen, including urea and uric acid, and applications of sucrose also prevented infection. Such results, as well as results with chemicals, indicate that some crop plants that could be used in rotations, soil amendments, or pesticides, may eventually be found economically useable to reduce or eliminate soil infection. In addition, there is considerable hope that economically desirable varieties of wheat can be developed readily from resistant or tolerant lines already being tested (20), and hence control the disease.

The stability of infectivity of soil contrasts with the instability of infectivity of juice from diseased plants, as indicated by unreliable results from manual inoculation. The stability in soil could result from an intimate association, with a persistent resting stage of a vector. The elimination of infectivity by certain chemicals probably also results largely from interference with a vector rather than a direct effect on the virus. None of these treatments is specific for one group of organisms. Even sucrose, which can destroy nematodes osmotically (5), would affect other organisms. A fungus such as *Polymyxa graminis* could be a vector of this virus, as has been suggested for wheat soil-borne mosaic virus in Nebraska (4) and Italy (2).

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