Windborne Dispersal of *Colletotrichum truncatum* and Survival in Infested Lentil Debris

L. Buchwaldt, R. A. A. Morrall, G. Chongo, and C. C. Bernier

First, third, and fourth authors: Department of Plant Science, University of Manitoba, Winnipeg, R3T 2N2 Manitoba, Canada; and second author: Department of Biology, 112 Science Place, University of Saskatchewan, S7N 5E2 Saskatchewan, Canada.
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ABSTRACT

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Different windborne materials were examined as potential sources of inoculum of *Colletotrichum truncatum*, the cause of anthracnose of lentil. Dust generated during harvest of anthracnose-infested lentil crops was shown to be a source of inoculum that could be dispersed by wind at least 240 m from a combine. Plant debris and soil dispersed by high winds in the fall from fields with infested lentil stubble also were sources of inoculum. Infectivity of dust, debris, and soil samples was determined by inoculating lentil assay plants. Samples that had overwintered outside also caused anthracnose on inoculated assay plants. Microsclerotia were on windborne lentil debris, but it was not possible to identify *C. truncatum* microscopically in the collected dust and soil samples. It appears that diseased lentil crops can be a source of inoculum for subsequent lentil crops planted at a distance. Dispersal of *C. truncatum* by wind has likely contributed to the spread of lentil anthracnose in western Canada. *C. truncatum* forms microsclerotia on infected lentil plants. A 4-year survival study of the pathogen showed that infectivity of infested debris placed on the soil surface declined during the first 12 months of exposure, whereas infectivity of buried debris remained high, until a rapid decline occurred at 48 months. The long-term survival of *C. truncatum* in buried lentil debris may explain the high level of infestation found in many fields in the province of Manitoba, although windborne inoculum may also be a source of infection.

Additional keywords: *Lens culinaris*.

Lentil (*Lens culinaris* Medik.), a traditional crop in the Middle East, North Africa, and Asia, has been grown in western Canada since the early 1970s. The acreage has increased substantially over the past 15 years to more than 350,000 ha annually and, consequently, fungal diseases such as anthracnose, caused by *Colletotrichum truncatum* (Schwein.) Andrus & W. D. Moore, have become increasingly important. Anthracnose was first reported in Manitoba in 1987 (11) and in Saskatchewan in 1990 (13). Annual surveys demonstrated that anthracnose occurs frequently at high levels in the major lentil production areas of Manitoba, especially in seasons with above average rainfall, and that it is widespread, but less severe, in Saskatchewan (3,4,12,14).

The first symptoms of anthracnose in the field are superficial lesions on leaves and stems of young lentil plants; the crop, however, appears healthy until early flowering. At this time, more lesions develop on lower leaflets, and a premature leaflet abscission begins. This is followed by development of severe lesions on the stem base that gradually move upwards on the stem. During rainy periods, acervuli are formed in lesions on the plant and on abscised leaflets on the ground, and conidia are splash-dispersed to the upper canopy and neighboring plants. As stem lesions enlarge, the plants may wilt and die. Microsclerotia are formed on all infected plant parts, and these survival structures remain in the field after harvest, either on lentil debris on the soil surface or buried in the soil by tillage.

It is unlikely that the wide distribution of anthracnose in western Canada is because of spread by seedborne *C. truncatum*. In field trials, seed from severely infected lentil plots have shown up to 2% infection with *C. truncatum* (L. Buchwaldt, *unpublished data*); however, similar levels of infection are rare in seed samples, even in years optimal for anthracnose development (4, 12–14). Furthermore, transmission of the pathogen from seed to seedling does not readily occur, if at all (8; R. A. A. Morrall, *unpublished data*).

One objective was to explain the rapid spread of lentil anthracnose in western Canada by examining different windborne materials as potential sources of inoculum. These materials included dust produced during lentil harvest and plant debris and soil spread by wind from fields containing infected lentil stubble. Since *C. truncatum* forms microsclerotia on infected lentil plants, another objective was to examine the longevity of the pathogen on debris either on the soil surface or buried in soil.

MATERIALS AND METHODS

Collection of windborne dust at harvest. Dispersal of *C. truncatum* in dust generated during harvest of diseased lentil crops was studied in three commercial fields in 1991 (fields 1, 2, and 3) and two in 1992 (fields 4 and 5). Before harvest, 50 plants were sampled randomly in each field by walking in a half circle of 100 to 200 m in radius. Anthracnose severity was rated on each plant using the following severity class values: 0 = healthy; 1 = a few small, superficial lesions on the stems; 2 = deep lesions on up to $^2/_3$ of the stems, but some stems still healthy; and 3 = deep, girdling lesions on all stems, the plant dying. For each field, a disease severity index (I_D) was calculated as 100 × [sum of (number of plants/severity class) × (severity class value)/150], in which the denominator is the 50 plants in the sample multiplied by the maximum severity class value, resulting in a disease severity index ranging from 0 to 100.

Fields 1, 2, and 3 were harvested from 12 to 16 August 1991 and fields 4 and 5 on 29 September 1992. During combining, dust

Corresponding author: L. Buchwaldt
E-mail address: buchwaldt@bldgagric.lan1.umanitoba.ca

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was generated and dispersed by wind. Samples of dust were collected on 1-m^2 sheets of polyethylene placed on the ground in two replicate linear transects downwind from the working combine. The first sheet in each of the two transects was placed 20 m from where the combine made its nearest pass and the others every 20 m out to 240 m, with a total of 12 sheets in each transect. At one location (field 3 in 1991), six sheets were placed in each of four replicate transects at 20-m intervals out to 120 m. There were 10 m between each transect at all locations. The sheets were held in place with metal stakes and exposed for at least 6 h while the combine was harvesting. The sheets were then folded to contain the trapped dust. Within an indoor area, the dust was removed using the following vacuum system. An in-line filter holder (no. 1119; Gelman Sciences Inc., Ann Arbor, MI) mounted with a 47-mm cellulose prefilter (no. 15403-47-N; Sartorius GmbH, Göttingen, Germany) was attached to a vacuum pump with a 1-m-long rubber tube. The area of suction was enlarged by attaching a 78-mm glass funnel to the filter holder using a 0.2-m-long rubber tube. To ease the movement of the glass funnel over the sheet, felt was glued to the rim of the funnel. Each sheet was vacuumed, and dust deposited on the filter was weighed. To prepare dust for microscopic examination, clear adhesive tape was drawn across the dust. The tape was then stained with lactophenol-cotton blue and examined microscopically (400×).

In 1991, dust was pooled over all collection distances within each of fields 1, 2, and 3. Infectivity of the three dust samples was tested in a host plant assay (described below) 2 months after collection. In addition, portions of the dust samples from fields 1 and 2 were enclosed in bags of water-permeable Miracloth (Calbiochem-Behring Corp., La Jolla, CA), placed outside on the soil surface at the University of Manitoba campus, and tested for infectivity in the host plant assay after 7-month overwintering. In 1992, dust samples from every second collection distance in fields 4 and 5 were tested for infectivity 4 months after collection, while samples from alternate collection distances were placed outside, as in 1991, and tested for infectivity after 6-month overwintering.

Collection of windborne lentil debris and soil. After harvest of lentil fields 1, 2, and 3 in August 1991, cone-shaped nets of glass fiber mesh (2.3 mm^2) were set up to trap plant debris and soil dispersed by wind during a period from 12 September to 17 October. The nets, which were 0.8 m long with a 0.25-m-diameter opening, were placed 50 mm above ground and 1 to 3 m from the edge of the harvested crops. One net was placed along each of the four sides in the three fields with infested lentil stubble. A 0.3-liter plastic cup was placed at the bottom of each net, and the cups were emptied weekly for 6 weeks. The samples were air-dried and passed through a 4-mm^2 sieve that separated the material into two fractions, one of large plant debris and the second of soil and small plant particles. The two fractions for each collection site, date, and field were weighed. Subsequently, for each of the three fields, plant debris fractions were pooled over collection date and site; soil fractions were pooled similarly. The plant debris fractions were examined microscopically (60 to 300×) for the presence of *C. truncatum*, and the soil fractions were examined after staining with lactophenol-cotton blue (400×).

Infectivity of the plant debris and soil fractions was examined in two different host plant assays (described below). In addition, a part of the soil fractions from fields 1, 2, and 3 were enclosed in Miracloth, placed outside, and tested for infectivity after 6-month overwintering. Samples of plant debris were not placed outside for overwintering, as they were too small and because survival of *C. truncatum* on debris was examined in a separate study described below.

Survival of *C. truncatum* in infested lentil debris. In 1991, 2 to 3 weeks before harvest, anthracnose-infected lentil plants were collected from commercial fields in southern Manitoba planted to the highly susceptible cultivar Eston. Microsclerotia of *C. truncatum* were present on stems, leaflets, and pods at the time of collection. Approximately 40 g of dried plant material was enclosed in each of 36 bags (0.3 × 0.4 m) of glass fiber mesh (2.3 mm^2). In August, the bags were placed at two sites 500 m apart on the University of Manitoba campus; one site had a clay soil and

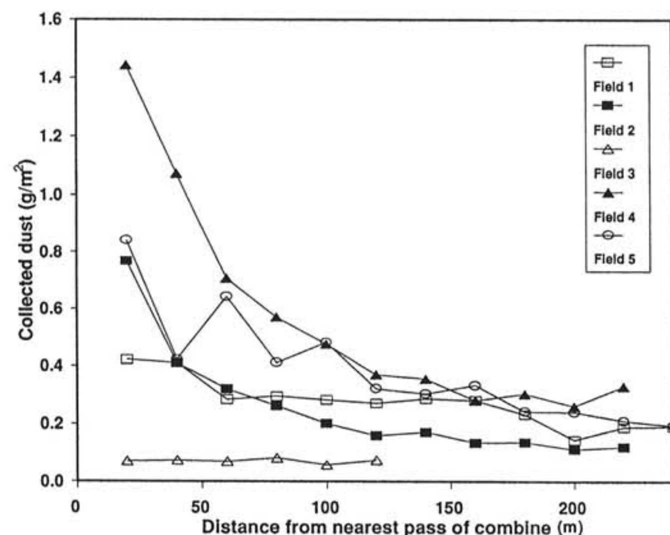

Fig. 1. Amount of windborne dust collected during harvest of five lentil crops in western Canada in 1991 to 1992. The average daily wind speeds were 13.6, 9.0, and 6.0 km/h in fields 1 to 3, respectively, and 15.6 km/h in fields 4 and 5.

TABLE 1. Percent assay plants infected with *Colletotrichum truncatum* after inoculation with wind-dispersed dust samples collected during combining of anthracnose-infested lentil crops in western Canada in 1991 to 1992

Field no.	I_D[a]	Distance from nearest pass of combine (m)	Infectivity of dust samples: Before overwintering	After overwintering
1991				
1	52	20–240[b]	100	37 ± 9
2	64	20–240	74 ± 13[c]	40 ± 6
3	95	20–120	95 ± 4	…[d]
1992				
4	68	20	20 ± 11	41 ± 19
		40	6 ± 6	25 ± 5
		60	11 ± 11	43 ± 7
		80	0	35 ± 24
		100	11 ± 11	46 ± 24
		120	0	40
		140	5 ± 5	100
		160	18	…
		180	0	62
		200	11	…
		220	11	55
		240	28	…
5	95	20	36 ± 13	41 ± 8
		40	3 ± 3	53 ± 9
		60	22 ± 12	60 ± 10
		80	0	80 ± 20
		100	69 ± 19	36 ± 14
		120	5 ± 5	45 ± 5
		140	8 ± 8	70
		160	11 ± 11	54
		180	22	60
		200	27	50
		220	…	70
		240	…	66

[a] Disease severity index of anthracnose in the lentil crop before harvest.
[b] Dust pooled over collection distances in 1991.
[c] Percent infected assay plants and standard error for inoculation of dust samples onto two or three replicate pots of lentil assay plants. No standard error for inoculation of one pot.
[d] Sample too small for inoculation.

the other a silt loam. At each site, half of the bags were buried at a 0.10 to 0.15 m depth, and half were placed on the soil surface. Both areas were left fallow, except for occasional hand-weeding. Every spring and fall for 4 consecutive years, one bag from the surface and one buried bag were recovered from each site. The bags were rinsed under running tap water, air-dried, and the contents weighed. Lentil debris recovered after 2, 8, 12, 20, 24, 32, 36, 44, and 48 months of exposure was tested for infectivity in a host plant assay (described below). Pieces of debris also were incubated on moist filter paper in a 90-mm petri dish for 2 to 4 days and examined microscopically (60 to 300×) for the presence of *C. truncatum*.

Host plant assays. Ten to 15 seeds of 'Eston' were sown in a soilless mixture (Metro mix; W. R. Grace & Co. Canada Ltd., Ajax, Ontario) in 0.5-liter pots. After 3 to 4 weeks in a growth room (20/16°C day/night temperatures with a 16-h photoperiod), the plants were inoculated with either dust or soil samples. A 0.5-g sample of each type of material was suspended in 10 ml of sterile, distilled water and brushed onto lentil stems in three pots. If dust at a single collection distance was less than 0.5 g, as in 1992, two pots of lentil plants were inoculated with 0.3 g of dust in 7 ml of water or one pot was inoculated with 0.2 g of dust in 5 ml of water. Noninoculated pots served as controls. All plants were incubated for 24 h in a plastic covered tent at 100% relative humidity, obtained with a humidifier set for constant, low-moisture production. The plants were then transferred to a greenhouse supplemented with 12 h of light per day provided by high-pressure sodium lamps. The windborne plant debris collected in the fall was assayed for infectivity by placing 0.5 g of debris on the soil surface of each of three pots containing 3- to 4-week-old lentil plants. Noninoculated plants served as control. The pots were placed in a greenhouse with good separation between pots and watered vigorously every second day to ensure splash-dispersal of inoculum from the debris onto the plants. Samples of lentil debris from the survival study were assayed similarly, except that four pots of lentil were each infested with a 0.5-g sample. For both methods of inoculation, the assay plants were examined microscopically (60 to 300×) 2 to 3 weeks after inoculation for the presence of stem lesions with sporulating *C. truncatum*. Identification of the pathogen was based on the presence of acervuli, setae, and microsclerotia. Plants without stem lesions, or with stem lesions but without fungal structures, were placed in plastic bags on a moist paper towel and sprayed with a solution of the herbicide paraquat at 0.3% a.i. (Sweep; ICI Chipman, Stoney Creek, Ontario, Canada) while inside the bag. Paraquat desiccates the plant tissue and enhances sporulation of *C. truncatum* (6). Paraquat-treated plants were reexamined after 4 days of incubation. The incidence of infected assay plants was calculated to include plants that showed signs of *C. truncatum* either before or after paraquat treatment. The standard error was calculated for the mean infectivity of each debris, dust, and soil sample, except when only one pot of lentil was inoculated, as with some dust samples from separate collection distances in 1992.

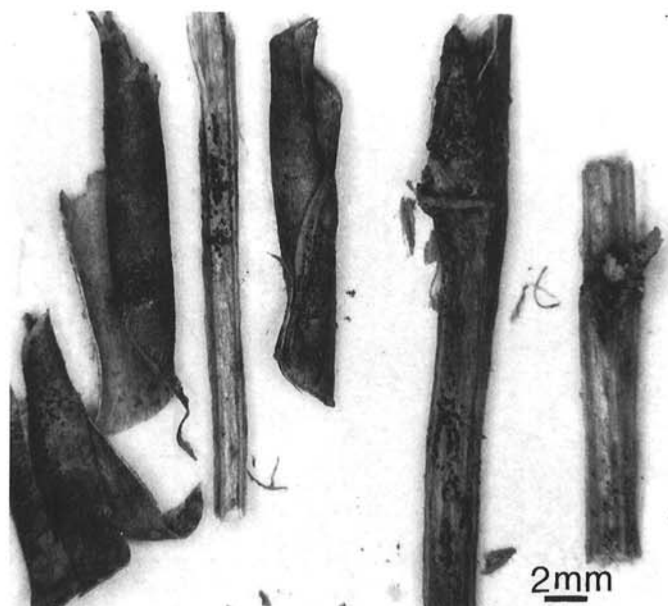

Fig. 2. Microsclerotia of *Colletotrichum truncatum* on lentil pod walls and stem pieces spread by wind in the fall from anthracnose-infested lentil fields in western Canada.

Relationship between disease incidence and inoculum concentration. To relate the incidence of infected assay plants to inoculum concentration, lentil plants grown and incubated under the same conditions as assay plants were inoculated with different spore concentrations of a single virulent isolate (JPPTNL 882) of *C. truncatum*. Eight seeds of 'Eston' were sown per pot and thinned to five plants after 3 to 4 weeks. Four replicate pots were inoculated with 20 to 24 ml of each spore concentration, incubated for 24 h at 100% relative humidity, and placed in a greenhouse in a complete randomized block design. Concentrations of 0, 1, 2, 5, 10, and 20 × 10^3 conidia/ml and 0, 20, 40, 60, 80, and 100 × 10^3 conidia/ml were tested and repeated two and three times, respectively. Percent infected plants were scored after 4 weeks. Regression analyses of percent infected plants on log (inoculum concentration) were performed.

RESULTS

Infectivity of windborne dust at harvest. Dust collected from field 3, harvested on a day with mean wind speed of 6.0 km/h, averaged 0.80 g/m^2 at all distances up to 120 m from the working combine (Fig. 1). The other four lentil crops were harvested at mean daily wind speeds of 9.0, 13.6, and 15.6 km/h, and dust was deposited in a gradient that averaged 0.42 to 1.44 g/m^2 at the collection distance nearest the combine to 0.12 to 0.33 g/m^2 at the collection distance located 240 m downwind (Fig. 1). Microscopic examination of the dust showed that it comprised fungal spores, primarily conidia of *Alternaria* species, plant, and soil particles. It was not possible to detect *C. truncatum* microscopically in these samples.

The disease severity index (I_D) for anthracnose in the five lentil crops before harvest ranged from 52 to 95 in 1991 and from 68 to 95 in 1992 (Table 1). Incidence of infected assay plants inoculated with dust collected during harvest in 1991 ranged from 74 to 100% before overwintering of the dust and from 37 to 40% after overwintering. Incidence of infected assay plants inoculated with dust from separate collection distances in 1992 ranged from 0 to 69% before overwintering and from 25 to 100% after overwintering (Table 1). Noninoculated assay plants did not develop symptoms of anthracnose.

TABLE 2. Percent assay plants infected with *Colletotrichum truncatum* after inoculation with windborne lentil debris and soil collected from anthracnose-infested lentil crops in western Canada

Source of inoculum	Field no.	Infectivity of samples: Before overwintering	Infectivity of samples: After overwintering
Windborne lentil debris	1	52 ± 30[a]	...[b]
	2	69 ± 14	...
	3	89 ± 6	...
Windborne soil and plant particles	1	1.9 ± 1.9	0.9 ± 0.9
	2	3.0 ± 2.1	2.1 ± 1.3
	3	1.4 ± 1.4	0

[a] Percent infected assay plants and standard error for inoculation with debris and soil samples.

[b] Sample too small for inoculation.

Infectivity of windborne lentil debris and soil. In 5 of 6 weeks from September to October 1991, the mean daily wind speeds varied from 10 to 35 km/h. Under these conditions, 0 to 10 g of plant debris and soil were trapped in each of fields 1, 2, and 3 (four collections sites combined). In 1 week, 3 days with a mean daily wind speed of 39 to 45 km/h occurred, and this resulted in trapping from 20 to 78 g of plant debris and soil.

After separation of the trapped material into two fractions, the first fraction comprised lentil pod walls and stem pieces and small amounts of straw from previous cereal crops; the second fraction comprised soil and plant particles less than 4 mm^2. Microscopic examination of the lentil debris showed the presence of microsclerotia of *C. truncatum* on pod walls and stem pieces (Fig. 2). Inoculation with this fraction resulted in 52 to 90% incidence of anthracnose-infected assay plants (Table 2). The other fraction, which consisted of soil and small plant particles, resulted in 1.4 to 3.0% incidence of infected assay plants before overwintering and up to 2.1% after overwintering (Table 2). Control plants did not develop anthracnose.

Survival of *C. truncatum* in lentil debris. After 1 year outside, the weights of lentil debris in the survival study were reduced to 35% of the original amount. The amount of debris declined to 20% of the original weight after 2 and 3 years and to about 10% at the end of the 4-year period. There were no differences in rate of decomposition between debris in buried bags and bags on the surface. Microsclerotia and setae characteristic of *C. truncatum* were recognizable on incubated pieces of lentil debris retrieved at regular intervals over that period (Fig. 3).

After 2 months outside, infectivity of lentil debris placed in bags on the soil surface averaged 98% disease incidence in the host plant assay. Infectivity of this material declined rapidly to 20 ± 10% after the first 12 months of exposure and varied from 5 ± 3% to 41 ± 7% in the following period, until no infectivity could be detected after 48 months (Fig. 4). In contrast, buried lentil debris remained highly infective and caused from 47 ± 9% to 87 ± 9% infected assay plants for 44 months, but, after 48 months, infectivity declined to 9 ± 6% (Fig. 4). None of the noninoculated control plants showed symptoms of anthracnose, indicating that inoculum from debris in inoculated pots was not splash-dispersed onto neighboring plants during watering.

Inoculum concentration and disease incidence. Incidence of infected lentil plants averaged 30% after inoculation with 1×10^3 conidia/ml, the lowest inoculum concentration evaluated. Incidence increased to 100% at the two highest conidial contractions of 8×10^4 and 1×10^5 conidia/ml (Fig. 5). There was a strong linear relationship between percent infected plants and the logarithm of inoculum concentration with a coefficient of determination $R^2 = 0.96$.

DISCUSSION

Estimates of the amount of infectious inoculum of *C. truncatum* in windborne dust, lentil debris, and soil were obtained by inoculating lentil assay plants with fixed amounts of each sample. No other pathogen of lentil such as ascochyta blight (*Ascochyta fabae* f. sp. *lentis*) was detected in the samples. However, interactions with saprophytic fungi and other microorganisms may have occurred, as under natural conditions. To aid detection of infected assay plants, plants with no visual structures of *C. truncatum* were treated with paraquat, which enhances the formation of conidia, setae, and microsclerotia (6). The linear relationship between percentage infected plants and logarithmic inoculum concentration (Fig. 5) was developed so that it would be possible to relate disease incidence in the host plant assay to an approximate conidial concentration of *C. truncatum* in the samples.

Several sources of inoculum of *C. truncatum* were identified in the present study: dust generated during harvest of anthracnose-infested lentil crops and debris and soil dispersed by wind from fields with infested lentil stubble. Combining of lentil in windy conditions above 9 km/h was accompanied by large amounts of dust that could be dispersed over distances more than 240 m from the combine. The presence of *C. truncatum* in the dust could not be determined microscopically, since the pathogen has small (12 to 16 × 4 to 6 μm), indistinguishable conidia and microsclerotia of a few hundred melanized cells that could not be differentiated from soil particles by staining. The presence of the pathogen became evident after inoculation of the dust onto lentil assay plants. Infectivity of the 1991 pooled dust samples before overwintering

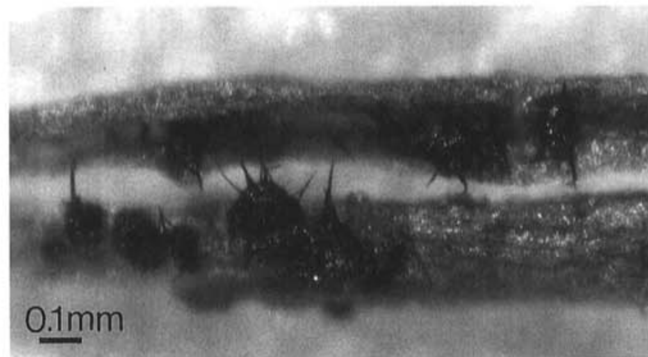

Fig. 3. Pieces of lentil debris with microsclerotia and setae of *Colletotrichum truncatum* retrieved after being buried for 4 years at a soil depth of 0.10 to 0.15 m.

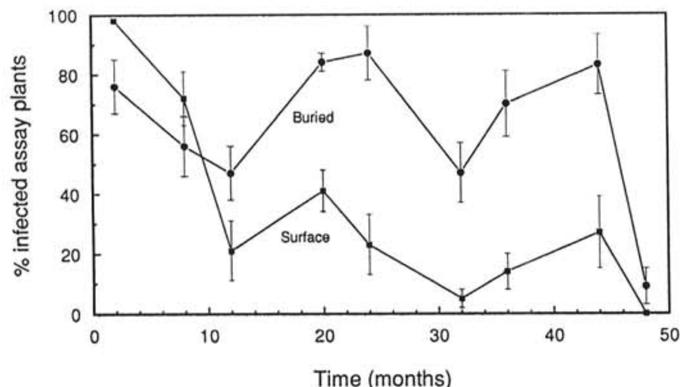

Fig. 4. Infectivity of lentil debris infected with *Colletotrichum truncatum* after burial in field soil or placed on the soil surface and retrieved after 2, 8, 12, 20, 24, 32, 36, 44, and 48 months of exposure. Infectivity tests were conducted by inoculation of lentil assay plants in a greenhouse.

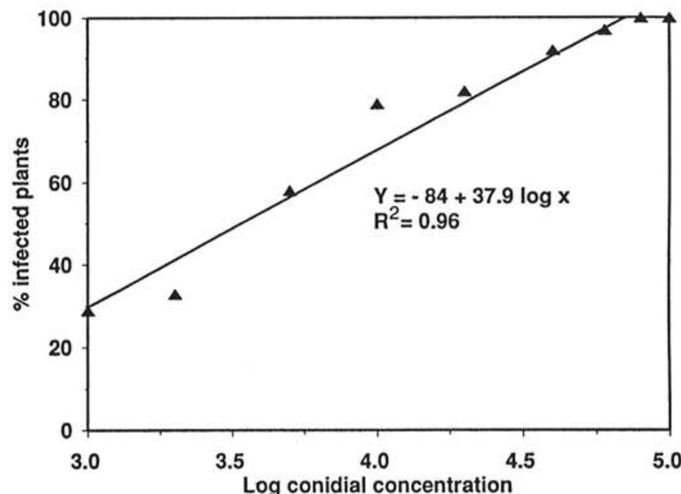

Fig. 5. Relationship between conidial concentration of *Colletotrichum truncatum* and percent lentil plants with anthracnose symptoms.

was equivalent to inoculation with 14.7 to 71.5 × 10^3 conidia/ml and to 1.8 × 10^3 conidia/ml after overwintering. Infectivity of the 1992 nonpooled dust samples before overwintering was equivalent to inoculation with up to 10.2 × 10^3 conidia/ml and to 1.8 to 71.5 × 10^3 conidia/ml after overwintering. There was no relationship between infectivity and distance from the working combine in 1992. However, inoculation with dust from separate collection distances in 1992 showed a larger variability in infectivity than inoculation with dust pooled over all collection distances in 1991 (Table 1). This may be due, in part, to variation in inoculum concentration at different distances and, in part, to variability in the assay, since small amounts of dust collected at distances furthest from the combine only allowed inoculation of one or two replicate pots of assay plants. The 4-month period from collection to the time of the assay before overwintering in 1992 may have reduced infectivity of these samples.

Dust clouds moving vertically to high altitudes were often observed in conjunction with working combines. Although collection of this potential source of inoculum was not possible, dispersal of anthracnose-infested dust over distances greater than 240 m probably occurs. Whether dispersal of inoculum in dust generated during harvest is unique to lentil anthracnose or also is important in dispersal of other pathogens needs to be examined.

High winds above a daily average of 39 km/h were able to disperse lentil pod walls, stem pieces, and soil from fields with lentil stubble. Infectivity of lentil debris varied between fields and corresponded to inoculation with 3.8 to 33 × 10^3 conidia/ml of *C. truncatum*. Since microsclerotia were found on much of this material, they likely account for the high infectivity (Fig. 2). Soil from the same fields caused infection in the host assay corresponding to inoculation with 200 conidia/ml. Infectivity of this fraction is probably because of the presence of microsclerotia, either associated with small plant particles (less than 4 mm^2) or detached.

It appears that lentil crops severely diseased with anthracnose can be a source of inoculum for subsequent lentil crops sown at a distance. This also has been noticed in annual disease surveys (12, 14). Consequently, lentil growers should place new lentil crops at a substantial distance from previously infected crops. The widespread prevalence of lentil anthracnose in western Canada has most likely been facilitated by wind dispersal of inoculum in dust generated at harvest and in lentil debris and soil dispersed by high winds in the fall. Wind dispersal may also explain the occurrence of anthracnose in fields not previously grown to lentil (9).

Microsclerotia of *C. truncatum* were apparent on anthracnose-infested lentil plants collected in commercial fields and used in the pathogen survival study. The ability of *C. truncatum* to form microsclerotia on legume hosts was first described by Andrus and Moore in 1935 (1) and later by Tiffany and Gilman in 1954 (17). Recently, isolates of *C. truncatum* producing microsclerotia on soybean (*Glycine max*) have been reported (10), and formation of microsclerotia on pea (*Pisum sativum*) artificially inoculated with *C. truncatum* has also been described (15). To our knowledge, there has been no report on the longevity of *C. truncatum* on lentil or other legume hosts.

Our study showed that *C. truncatum* was able to survive up to 44 months on buried lentil debris, after which a rapid decline occurred by 48 months, whereas viability was reduced to low levels after 12 months on the soil surface (Fig. 4). Infectivities of debris left on the soil surface, as measured in the host plant assay, were 21 and 23% after 12 and 24 months, respectively. In contrast, infectivities of buried debris were 47 and 87% on the same dates. Infectivity of 0.5 g of surface material corresponds to inoculation with 1.6 × 10^3 conidia/ml and buried material to 2.8 to 3.2 × 10^3 conidia/ml. Microsclerotia of *C. truncatum* could be identified on pieces of lentil debris retrieved during the 4-year period, and infectivity was probably related to the presence of these survival structures (Fig. 3). The persistence of *C. truncatum* in soil is probably a result of prolonged periods of low temperatures characteristic of western Canada, which are inhibitory to parasitism of the microsclerotia by other organisms. The relative short survival of *C. truncatum* in lentil debris on the soil surface is possibly because of exposure of microsclerotia to repeated wetting and drying, temperature extremes, and UV radiation.

C. coccodes has long been known to produce microsclerotia on host species such as tomato and potato, and two studies (2,7) have shown no decline in survival of these resting structures over test periods of 52 and 84 weeks, respectively. *C. graminicola*, the cause of sorghum anthracnose in Texas, survived 18 months in sorghum residue on the soil surface, but not as long when buried in the soil; microsclerotia on the residue were found to be the primary source of inoculum (5). The difference between the longevity of the lentil and sorghum pathogens in soil may be attributable to higher microbial activity and to faster breakdown of plant residues in the warmer soils of the southern United States compared with western Canada. However, microsclerotia of *Microdochium panattonianum*, the cause of lettuce anthracnose, was reported to survive almost unchanged in the soil for 3 years in California, after which populations rapidly declined (16).

A preliminary study of host range found that faba bean (*Vicia faba*) and vetch (*Vicia sativa*) were highly susceptible when inoculated with *C. truncatum* isolates from lentil; whereas lupin (*Lupinus angustifolius*), field bean (*Phaseolus vulgaris*), alfalfa (*Medicago sativa*), soybean (*Glycine max*), and round-leaved mallow (*Malva pusilla*) showed an incompatible reaction (8). Pea (*Pisum sativum*) developed superficial and nonspreading necrotic lesions on stems and leaves with little or no detrimental effect to the plants (8), indicating that *C. truncatum* from lentil has relatively little effect on pea. Since faba bean is only grown on a small acreage in western Canada, legume crops other than lentil are probably of limited importance for the buildup of inoculum of *C. truncatum*. Thus, it seems that the major source of inoculum is anthracnose-infested lentil debris left in the field after a diseased crop. The rapid decline in infectivity of *C. truncatum* on debris left on the soil surface indicates that the best approach to reduce the amount of viable inoculum in commercial lentil fields would be to leave infested stubble for 1 year before burial by tillage.

The presence of microsclerotia and the longevity of *C. truncatum* on lentil debris can explain the occurrence of anthracnose, even in lentil crops in a 4-year crop rotation as observed in annual disease surveys (9,14), although windborne inoculum from nearby fields also may be a source of infection.

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