

Bacterial Blight of Soybeans: Epidemiology of Blight Outbreaks

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ABSTRACT

The relationship of wind-rain storms to the initiation of bacterial blight outbreaks incited by *Pseudomonas glycinea* on leaves of soybean (*Glycine max*) was investigated. Initiation of outbreaks on young leaves occurred during three wind-rain storms. Lesions were often associated with obviously injured areas. Even though inoculum was present, little disease was initiated in the absence of storms or during rain without wind. Inoculum was transferred from plants with leaves showing lesions to healthy leaves of the same and adjacent plants during simulated and natural storms. This was shown by

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the development of new lesions and the presence of the pathogen in leaf surface water. The wind component of storms was an essential factor in disease initiation. When closely grouped greenhouse and field plants were agitated by wind and immediately subjected to inoculum in the form of gently falling mist, abundant blight developed on leaves. During natural or simulated wind-rain storms, lesions were not initiated on all leaves; only one to two of the youngest open leaves of a plant were infected.

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The epidemiology of blight incited by *Pseudomonas glycinea* (Coerper) Stapp on soybean, *Glycine max* (L.) Merr., is incompletely understood. Blight is one of the most prevalent bacterial diseases of soybean, and is the most prevalent in Ohio. It occurs in Ohio in varying degrees on all cultivars, and leaf-spotting is the major symptom.

Symptom characteristics on leaves of field plants have been described by Wolf (17) and Coerper (3). Usually lesions are distributed in a field with no apparent pattern. However, under our conditions an outbreak of blight usually is distinctive, and is characterized by lesioned leaves grouped horizontally in a "layer" pattern; one or a few leaves of the same age on all or most of the plants within a large area of a field show lesions (14).

The disease was described in 1919 (3), and since then most investigations have been devoted to viability studies of *P. glycinea* in culture, in diseased plant material in the soil, and in association with the seed. Evidence indicates that the pathogen overwinters in or on the seed, and under some conditions, in the diseased plant debris associated with soil (4, 8).

Studies of other bacterial diseases (6, 16) suggested that wind-rain storms are probably important in the spread of *P. glycinea* in the field, and soybean bacterial blight increases in wet weather (12, 17). However, no detailed work has been published on the epidemiology of blight outbreaks. The present paper reports studies of effects of wind-rain storms on the incidence of blight, particularly in reference to the layer pattern. Detailed field observations, made during 1969 when three major storms occurred at Wooster, Ohio, were complemented in studies employing simulated storms. A brief account of some of this work has appeared (5).

MATERIALS AND METHODS.—*Observations concerned with natural storms.*—Detailed

observations of lesion initiation were made in an 80-x 85-m study field of the cultivar Harosoy 63, drill-planted 27 to 36 seeds/m in rows 17 cm apart on 27 May. Individual plants and designated disease areas within the field were observed weekly or more often, especially after each of the three wind-rain storms. The developmental stage of the plants and information concerning storms are noted in Table 1. A record was kept of the times at which lesions were first discernible, especially after storms. Rainfall was measured about 0.7 km away.

Simulated wind-rain storm in the field.—Rows (1 m apart) of a field of 0.5- to 0.7-m-high plants of Harosoy 63 were divided into experimental units 1 m long (30 to 38 plants) by removing 1.5- to 1.8-m portions of rows. These rows were separated by guard rows. A small amount of natural infection was present in the field.

There were 10 blocks with one replicate of four treatments (Table 2) randomized in each block. Inoculum was prepared by soaking 100 naturally infected trifoliolate leaflets for 30 min in 64 liters of water in the mixing tank of a tractor-mounted hydraulic pesticide sprayer. Spray from the spray gun nozzle was directed on the central 10 to 12 plants of a replicate. Adjacent plants were shielded. The amount of disease on inoculated plants 10 days later was 19 to 21 mean per cent, as rated by the Barratt-Horsfall method (10) and converted by the Elanco Conversion Tables (Elanco Products Co., Indianapolis, Ind.). No lesions were noted on shielded plants.

A wind-rain storm was produced with the hydraulic sprayer. About 50 liters of water were directed onto the diseased and adjacent healthy plants of a replicate, using a back-and-forth motion of the spray nozzle in about 3 min. Water congestion of some leaves indicated the force of the application.

TABLE 1. Relationship of three wind-rain storms at Wooster, Ohio, in 1969 to stages of soybean plant growth

	Storm I (30 June-1 July)	Storm II (4-6 July)	Storm III (12 July)
General description of storm winds, gross plant injury	Gusty Young leaves torn	Moderate breezes Young leaves torn	Very strong winds Young leaves torn
Rainfall during storm period	1.7 cm	24.0 cm	2.5 cm
Approximate duration of storm	8-9 hr	48 hr	0.33 hr
Growth stage: number of open trifoliolate leaves	3-4	4-5	6-7

TABLE 2. Disease incidence following a simulated wind-rain storm in the field on soybean plants adjacent to soybean plants infected with *Pseudomonas glycinea*

Treatment		Lesioned leaflets on plants adjacent to central diseased plants		
Diseased plants in the central portion of a replicate	Plants of entire replicate subjected to simulated wind-rain storm	1-15 lesions	16-50 lesions	51 or more lesions
+a	-	21 ^b	0	0
+	+	142	57	35
-	-	7 ^b	2 ^b	0
-	+	9 ^b	7 ^b	2 ^b

^a + = plants treated as indicated by the column heading; - = control plants.

^b An occasional lesion was seen on these plants; lesions were thought to be the result of natural infection.

TABLE 3. The effect of simulated wind on initiation of bacterial blight (*Pseudomonas glycinea*) on field grown soybean plants

Treatment ^a			Estimated leaf area diseased (%)	
No.	Inoculum sprinkled on leaves before wind treatment	Simulated wind	Exp. I	Exp. II
1	+ ^b	-	1.2	1.2
2	+	+	14.0	15.0
3	-	-	0.0	0.5
4	-	+	3.0	1.4

^a Each treatment consisted of 10 replicates.

^b + = plants treated as indicated by the column heading; - = control plants.

The lesioned leaflets and number of lesions on the four youngest leaves of the adjacent, noninoculated, shielded plants were counted 12 days later.

Simulated wind storms in the field.—Block, replication, and kind and size of plants were the same as in the simulated wind-rain storm. Inoculum was prepared by soaking and occasionally squeezing naturally infected leaves in water (50 leaflets/10.5 liters) for 12 min. In the one experiment in which *P. glycinea* was assayed by the plate dilution method (14), 4.5×10^4 cells/ml were in the unused portion of the inoculum 1 hr after inoculation.

To place inoculum on leaves in Treatment 1 (Table 3), 2 liters of suspension were gently sprinkled on all plants in a replicate using a sprinkling can with a rose flare nozzle. Inoculum for Treatment 2 was applied as follows: a sprinkling, as above, of 0.66 liter of inoculum/replicate was followed immediately by application of "wind" for 20 sec from an air-blast back-pack sprayer set at full throttle. The inoculum and wind application was repeated 3 times, with the result that each replicate of Treatment 2 received about 2 liters of inoculum and 1 min of air blast. The sprayer, a Solo Mixer, Type 400010 (SOLO

KLEINMOTOREN GMBH Maichingen, Postfach 20, West Germany) was operated without liquid. The nozzle was held about 30 cm from the foliage and the air was played back and forth, creating a forceful agitation of the plants. Plants in Treatments 3 and 4 were sprinkled with water instead of inoculum. The amount of disease was rated after 8 days by the Barratt-Horsfall method, and converted to mean per cent of diseased tissue.

RESULTS.—*Natural storms.*—*Disease incidence prior to and following Storms I and II.*—Scattered lesions were observed on unifoliolate and first trifoliolate leaves in the study field prior to the first wind-rain storm the night of 30 June (Storm I, Table 1). Cotyledons observed appeared healthy.

On the morning after Storm I, leaflets (about 5 cm long) of most young trifoliolate leaves were torn from the margin to the midrib. Storm II (Table 1) occurred 4 days later. A large amount of rain fell, beginning in late evening of 4 July and continuing through 6 July. Winds were not strong, but foliage was moving most of the time when observations were made.

Blight symptoms were extensive on 8 and 9 July. "Heavy-disease areas" and "light-disease areas" (areas in which plants had scarcely any blight) were apparent in the study field. These areas were of irregular form and 15 to 20 m in greatest dimension. The reason for these irregularities is not known, but they had no apparent relationship to moisture or soil factors. Lesion development was similar to that reported by Coerper (3) and Wolf (17). Plants varied in size (0.3 to 0.5 m high), the smallest having four open trifoliolate leaves, and the largest, five (Table 1). Leaf development on the young, upper parts of plants was similar, irrespective of plant size. No lesions were on leaves 3.5 to 4 cm in length (these leaves were in the bud at the time of Storm II), or on "birdclaw" leaves, i.e., those partly opened, or on the leaf that had just opened (this leaf was a birdclaw at the time of Storm II). However, lesions were abundant on the two next-lower or older leaves. Virtually every lesioned leaf was torn, and leaves that were not torn bore few lesions, indicating that infection took place during the storms. Of the two diseased leaves, the younger bore the least developed lesions. Since other field observations indicated that symptoms appeared 2 to 4 days after inoculation, this leaf probably was infected in the more recent storm, Storm II. The older of the two leaves apparently was infected in the earlier storm (Storm I), for it had the older lesions. The leaf below this leaf was not observed in detail, but it was noted that few lesions were present.

Lesions often were grouped in linear patterns between major veins and along the leaf margins following these storms. Lesions were located at obviously worn areas or at actual perforations; i.e., lesion patterns coincided with injuries. We call this symptom pattern an "injury-lesion line".

Source of inoculum for disease initiated in Storm II.—Observations on 8 and 9 July showed, almost without exception, that if the older of two consecutive leaves on a plant was diseased, the younger was

also diseased. This suggested that lesions on the older leaf were the source of inoculum which had initiated lesions on the younger leaves.

To extend this observation, disease incidence was evaluated on 60 plants from each of two areas within the study field, a light-disease area and a heavy-disease area. A disease rating of the torn older leaf (infected in Storm I) was given to each plant. This leaf was recognized by its torn condition, and additionally, if lesions were present, by the age of lesions. The next youngest torn leaf, recognized by younger lesion age to have been infected in Storm II, was similarly rated.

Results showed that plants from the light-disease area which had few or no lesions on the older leaf also had few or no lesions on the younger leaf. Thus, 94% of older leaves bore < 15 lesions/leaf; 79% of younger leaves had < 15 lesions; and 13% had 15 to 30 lesions. Plants from the heavy-disease area which had many lesions on the older leaf also had many lesions on the younger leaf. Thus, 38% of older leaves bore 31 to 100 lesions/leaf, and 53% bore 101 to 300 lesions; 90% of younger leaves had > 101 lesions. These observations support the view that lesions initiated in Storm I were indeed the source of the inoculum which incited lesions in Storm II.

Storm III.—After Storm II, weather was mild until a heavy wind and rain storm on 12 July, during which 2.5 cm of rain fell in 20 min (Table 1). Plants drooped downwind. Leaflets of some birdclaws were split from margin to midrib, and much tearing of youngest open leaves occurred. Lower surfaces of many leaflets had small areas that were bruised or appeared to be water congested, perhaps because rain had been driven into intercellular spaces. These deep-green areas disappeared in about 30 min. Most of the leaves heavily lesioned and torn in Storm I and in Storm II were apparently weakened by necrosis, and many were torn further. Lesions were visible on young open leaves 2 to 4 days after the storm.

Stage of leaf development as related to lesion initiation in Storm III.—To extend further the observations that lesions were initiated only on young open leaves in storms, immediately following Storm III the youngest open leaf of each of 21 plants in a diseased area of the study field was identified by the attaching of a tag to the stem just below the petiole (Fig. 1). Plants were isolated by removing nearby plants. All bore lesioned leaves as a result of Storms I and II. Leaves infected in these storms were in positions (in respect to elevation above the soil) as high as or higher than the identified leaf (Fig. 1). The identified leaf and younger structures were healthy, although some had been torn in Storm III.

Identification of a given leaf permitted a later identification of leaves which at the time of the storm were (i) the youngest open leaf; (ii) the birdclaw; or (iii) the leaves folded in the bud. Eleven days after the storm, the youngest open leaf had increased in size, the birdclaw had opened fully, and the three leaves in the bud had opened. Lesions were counted on these five leaves. Results in Table 4 show that few or no lesions were initiated on leaves still within the

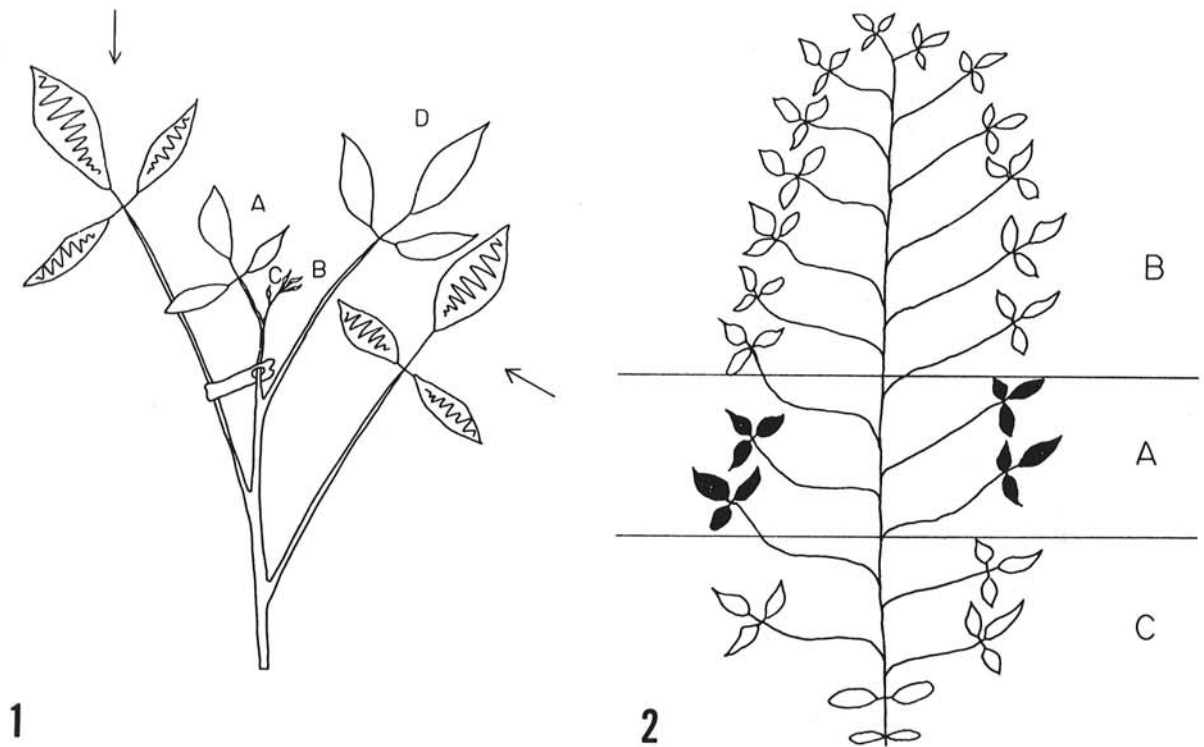


Fig. 1-2. 1) Terminal portion of a typical field soybean plant at time of Storm III. Note the possibility of transfer of *Pseudomonas glycinea* in raindrops and by contact between wet foliage parts during a wind-rain storm from leaves lesioned in Storm I and II (arrows) to open leaves (A) and (D), the birdclaw (B), and the bud (C). Tag for subsequent identification of leaves was placed on stem below youngest open leaf (A) immediately following Storm III. Following Storm III, most lesions were on leaves (A) and (D); a few were on leaf (B). 2) A representative late-season soybean plant of the study field showing distribution of lesions incited by *P. glycinea*. Black leaves were severely diseased. (A) = section of plant on which many lesions were initiated on young open leaves in Storms I, II, and III; leaves were torn in storms; (B) = section of plant on which few or no lesions were initiated in the stormless period following the period of storms; no leaves were torn; (C) = section of plant on which few or no lesions were initiated prior to the storm period; leaves were not torn.

TABLE 4. Stage of leaf development on soybean plants in relation to leaf injury and amount of blight caused by *Pseudomonas glycinea* following Storm III

Stage of leaf development at time of storm	Lesions on 21 leaves	Storm-torn leaves ^a
Youngest open leaf	1,772 (range:10-260/leaf)	21/21
Partly open leaf (birdclaw)	97 (range:0-23/leaf)	4/21
In bud (3 leaves)	3 on 63 leaves ^b	0/63

^a Number of leaves observed to be torn at times lesions were counted per number of leaves.

^b One lesion on each of three leaves (lesions on tip or edge of leaflet).

bud at the time of the storm. Lesions were initiated on open leaves and on birdclaws in numbers suggesting a positive relationship between injury and lesion initiation. No new lesions were on leaves infected in Storms I and II. Also, these old lesions had not increased in size.

Thus, at the time of the storm, lesions were initiated in large numbers on the two youngest open leaves, a few were initiated on partly opened leaves, and virtually none was initiated on leaves in the bud. Leaves that were torn had the highest incidence of disease. Lesions were distributed along the margins of

the obvious (tear) injuries, as well as on parts of the leaf on which gross injury was not discernible.

Pseudomonas glycinea in suspension on leaf surfaces following Storm III.—An assay of plant surface water for the presence of *P. glycinea* in suspension on two lesioned plants (leaves lesioned as a result of Storms I and II) was made immediately following Storm III. Water taken from eight healthy-appearing leaves, two birdclaws, and two buds caused typical blight symptoms when applied to greenhouse plants by the Q-stick method of inoculation (13). The

results indicated dissemination of the pathogen from lesions of the same and neighboring plants, presumably by dripping, blowing, splashing, and foliar contacts. Results of other similar assays indicated that *P. glycinea* was in dew on healthy leaf areas near lesions, which suggests that the pathogen also may move in dew water.

Distribution of lesions toward the end of the growing season.—To study the distribution of blight on individual plants, on 12 July (7 days after Storm II and immediately after Storm III), nine representative plants were isolated by removing nearby plants. Lesions resulting from Storms I and II were counted on each plant. A few days later, lesions resulting from Storm III were counted. As new leaves expanded, new counts were made. The last count was on 22 August, when the last terminal leaves formed during the season were about half their final size.

The distribution of lesions on 22 August is represented in Fig. 2. Note that the youngest leaves (B), those emerging from the bud during the stormless period following Storm III, were essentially free of disease (12 lesions on 351 leaflets). Leaves (C) that had emerged from the bud prior to the first storm (except the leaf on which lesions were initiated in this storm) also were essentially free of disease. The development of disease on these plants was found to be the same on nearby plants which had not been isolated, clearly indicating that the initiation of most disease took place during the three storms.

Rain without damaging winds occurred during the latter part of the season, and during windy rainless periods, foliage agitation caused leaves to be in contact. We concluded that water and wind by themselves were not sufficient to transfer inoculum and to predispose the leaf to infection.

Simulated storms.—Lesion initiation in a simulated wind-rain storm in the field.—Lesions developed on large numbers of leaflets of plants adjacent to diseased plants following the simulated wind-rain storm (Table 2). Very few leaflets were lesioned in the absence of the wind-rain storm treatment.

Distribution of lesions on individual plants after the simulated storm was the same as observed following natural storms. Thus, 10 days after the storm, nearly all lesions were found on the one-to-two leaves that were newly opened at the time of the storm.

Lesion initiation in simulated wind storms in the field.—In the foregoing, the importance of wind-rain storms has been stressed. Results from two simulated wind storm tests in the field indicated that wind was an essential factor in lesion initiation, because inoculum without wind produced little infection (Table 3).

Wind-caused foliage abrasion injury.—Injury-lesion lines on young open leaves strongly suggested that injury was an important predisposing factor in blight initiation under storm conditions. The following year, 1970, the importance of foliage abrasion was investigated.

The intermixed foliage of a group of 18 field plants about 0.5 m high was agitated in a manner

similar to wind-caused movements by the moving of the lower stems by hand for 10 min. Foliage then was inoculated (10^7 cells/ml) with a No. 15 DeVilbiss atomizer held not less than 20 cm from foliage, so that inoculum mist fell gently on the plants. Many lesions (75 to 200/leaflet) developed on young, open leaves of agitated plants. Very few (0 to 10/leaflet) developed on leaves of control plants. Lesions did not develop on old leaves. In a similar experiment, one of two transplanted groups of field plants was subjected to 4 hr of simulated wind (wind speed ca. 24 km/hr [15 mph]) from an electric fan in the greenhouse. Both groups were subjected to mist inoculum. Six times as many lesions developed on wind-treated plants as on control plants not subjected to wind.

In another test, a cluster of pots containing healthy greenhouse seedlings with only the unifoliate leaves open was placed in a moderate natural wind (16 km/hr [10 mph]) for 1 hr. Plants shifted and rubbed against each other. Upon return to the calm greenhouse air they were mist-inoculated, as were control plants that had been left in the greenhouse. Only wind-treated plants became diseased. Injury-lesion lines were common. Results were similar on trifoliate leaves of older plants (two trifoliate leaves open) placed in the wind, and when groups of seedlings were placed in a gentle natural wind (3.2 to 6.4 km/hr [2 to 4 mph]) for 2 hr, then inoculated.

In these experiments, lesions did not develop on leaves in the bud or at birdclaw stage at the time plants were placed in the wind, thus confirming observations made following natural storms. Also, lesions were absent on older leaves, as was observed following natural storms. In work reported elsewhere (4), lesions developed on field or greenhouse plants when a 1- to 3-hr interval elapsed following injury and prior to misting leaves with inoculum. The response after longer intervals was not examined.

Only preliminary examination of water congestion of leaves as a predisposing factor was made. In general we observed that, with field plants, leaves that are subject to disease in storms are not those most often water congested. Water congestion, especially on dewy mornings, is most common on the lower, older leaves. However, some lesions were produced when inoculum was misted on water congested young open leaves of greenhouse plants and on old leaves of field plants (4).

DISCUSSION.—This investigation indicates that a succession of wind-rain storms will result in soybean blight of epidemic proportions if inoculum is present early in the growing season. It was shown that, during storm periods, rain and wind spread the inoculum, as has been known with a number of bacterial plant pathogens. The wind component of storms proved to be of great importance in producing injuries that were predisposing to blight. Other workers also have suggested that wind injury promotes bacterial diseases (1, 2, 7). It is worth noting that in the year following the season of storms, no major wind-rain storms and no blight outbreaks occurred at Wooster. Inoculum was known to be present.

Wind alone appears to be of little importance in the epidemiology of soybean blight unless moisture is present. Wind-rain storms are most likely to provide these essential components. The movement of the pathogen by motility, gravity, or capillarity in dew or gentle rain appears to be of little consequence, probably because movement is limited and plants are not injured.

Lesions on soybean cotyledons early in the growing season have been reported (17), and the transfer of inoculum in simulated wind-rain storms from cotyledons and lower leaves to leaves higher on the plant has been shown (4). Storms may thus distribute inoculum from cotyledons and cause foliage injuries resulting in the initiation of a few lesions on the seedling foliage. Under some conditions, the pathogen may be a bud resident and be carried up the plant, although this has been shown only under conditions of high humidity in the laboratory (14). Also, the pathogen has been suggested as systemic (11); however, a similar study (4) indicated that movement of the pathogen within the plant was limited and of questionable epidemiological importance. The part these mechanisms play in nature is uncertain. However, the present work demonstrates that a major wind-rain storm may cause general distribution of inoculum from scattered lesions and extensive predisposing abrasion of young, open leaves. Since older leaves do not become diseased, the consequence of the storm would be a horizontal layer of diseased, young, open leaves in areas of a field where sufficient inoculum is present and when subsequent weather conditions are favorable for disease development.

Under our conditions, one such layer seems to be of small consequence; on the other hand, a series of storms about 1 week apart would be expected to reduce productive leaf area considerably. Also, plant growth may be reduced by systemic toxemia (9, 15). Evidence suggests that, given the currently prevalent lines of *P. glycinea* and the currently used soybean cultivars, blight will not cause general serious soybean crop losses in Ohio. This statement is based on the observation that a series of wind-rain storms at intervals of 1 to 2 weeks throughout the season are not common in local areas, and would be expected to be even less common in large regions.

It is noteworthy that only a small part of the plant, the youngest, one-to-two open leaves, becomes lesioned following a wind-rain storm. Leaves that are partly opened or those in the bud appear to be protected from abrasion. It is not certain why old leaves do not become diseased. Perhaps they are less

subject to injury: they are torn less by a storm than newly opened leaves. Old leaves of field plants were susceptible when inoculum was applied with an abrasive (Daft & Leben, *unpublished data*).

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