Epidemiology of Cercosporella Footrot of Wheat: Disease Spread

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ABSTRACT

Point sources of inoculum were established in November in a field of seedling winter wheat by depositing 30 gm of colonized oat inoculum on the soil. The inoculum was removed from some plots in April. Early-season disease gradients, calculated by plotting the log percent infection against the log distance from the inoculum source, were only slightly flatter (i.e. the spores moved only slightly farther) than gradients calculated from a rain-splash model system. The effective dispersal range of the fungus was only 3-4 ft. Apparent infection rates were low (r = .005 to .011) as compared with cereal rusts (r = .10 to .50). An increase in r was noted in mid-spring in plots with inoculum but not in plots where the inoculum had been removed, emphasizing the importance of a continuing source of primary inoculum for spring infection. The data fit Van der Plank's "simple interest" model, which suggests no role for secondary inoculum in the development of current-season disease epidemics.

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Additional key words: eyespot, sporulation, inoculum dispersal, infection rate.

Footrot of wheat, *Triticum aestivum* L. caused by *Cercosporella herpotrichoides* Fron. occurs in the Pacific Northwest primarily in higher rainfall regions east of the Cascade Mountains. Disease incidence is variable locally and seasonally depending upon weather conditions, inoculum levels, and methods of wheat culture.

Conidia of *C. herpotrichoides* are dispersed primarily by rain splash from sporulating stubble infected in previous seasons (3, 5, 7, 13, 14). Using a wind tunnel, Schrödter & Fehrmann (17) demonstrated that propagules can become airborne and cause infection where there is no rain splash for "take-off." Although

wind is associated with airborne dispersal, most authors agree that under field conditions spores must first be mechanically lifted by the force of impacting raindrops (9, 14).

The relative importance of primary and secondary inoculum in the spread of footrot has not been clearly defined. Since Sprague (18) first described this disease, most authors have assumed that secondary inoculum is produced from developing lesions, and that abundant production of secondary inoculum in the spring leads to footrot epidemics (4, 6, 7, 12, 19). Ponchet (13, 14) attributed most spring infections to secondary inoculum, but felt that in France these infections did not cause serious injury.

Van der Plank (21) classed epidemic plant diseases into two groups. "Simple interest" diseases are those in which the inoculum source is fixed and increase in disease with time is arithmetic. "Compound interest" diseases have secondary sources of inoculum that develop from primary infections, and disease increases exponentially.

The purpose of this study was to monitor the spread of footrot from point sources of inoculum and to identify and evaluate factors affecting its progress.

MATERIALS AND METHODS.—In November, 1969, plots were established in a field of susceptible seedling winter wheat cultivar 'Gaines' near La Grande, Oregon. The climate in this location is semi-arid, with an average annual precipitation of ca. 60 cm falling mainly from October to June. Average mean temperature in the winter is ca. 5 C and in summer ca. 30 C. Forty-eight point sources of inoculum were set out on a 50-ft grid pattern, which was considered adequate to avoid overlapping spread patterns of inoculum. Inoculum sources consisted of approximately 30 g of oats colonized by *C. herpotrichoides* (2) deposited on the soil surface in a circle 10-12 cm in diameter at each point on the grid. Spores formed on the oat inoculum were dispersed naturally from this point.

Plants surrounding four randomly selected point sources were sampled each month beginning in February 1970,and continuing until harvest in August. Twenty plants were taken at random from each of nine concentric circles laid out at 1-ft radial intervals from the inoculum source. Control plants were taken from between the inoculated plots, at least 20 ft from the inoculum sources. The extent of lesion development and the percent infection on a plant and tiller basis was determined by observation of individual tillers.

Inoculum was removed from 20 point sources in early April to determine the effect of primary inoculum sources on continued disease development. Beginning in May, the plants surrounding four of these point sources, selected randomly, were also sampled each month.

Evaluation of the production of primary and secondary inoculum on naturally infected stubble and newly formed lesions, respectively, was accomplished by monthly sampling of sporulation on these tissues during the spring of 1971 in another field of Gaines wheat near La Grande.

RESULTS AND DISCUSSION.—*Disease gradients.*—Calculation of footrot disease gradients confirmed that rain-splash was the primary mechanism of spore dispersal for *C. herpotrichoides* and emphasized the short-range dispersal ability of this pathogen. Disease gradients were calculated by plotting the log of the percent infection against the log of the distance of infected plants from the inoculum source. Our observations and the work of Ponchet (13) indicated that in the field the interval from infection to visible lesion development was about 4-6 weeks under favorable conditions. In small plots, it is assumed that any factor affecting symptom development should affect all plants equally regardless of distance from the inoculum source. Thus, early in the season the slope of footrot disease gradients should equal the slope of corresponding inoculum dispersal gradients 4-6 weeks earlier.

The slope of a theoretical rain-splash dispersal gradient was calculated from data published by Gregory et al. (8). The log of the total number of secondary splash droplets per square centimeter was plotted against the log of the horizontal distance these droplets traveled before impacting. When secondary droplets of all sizes were considered, the slope of the regression line through these points was −1.58 (Fig. 1-D). However, when the size of the impacting droplets was considered, the negative slopes of the resulting regression lines increased as the average droplet size increased (Fig. 1). This is significant in dispersal of inoculum when the relation of droplet size to spore carrying ability is considered. Gregory et al. (8) reported that few droplets smaller than 30 μm carried spores, and that the percentage carrying spores increased with droplet size. When the log of the total number of spore-containing droplets impacting per square centimeter, regardless of size, was plotted against the log of the horizontal distance these droplets traveled before landing, the resulting regression line had a slope of −1.06 (Fig. 1-G). Splash dispersal of fungal spores can then be expected to result in a dispersal gradient no steeper than −1.06 if dispersal is unobstructed.

The negative slope of the footrot disease gradient changed with time, but in all cases remained flatter than the theoretical rain splash gradient of −1.06 (Fig. 2). This is to be expected, since any factor that increases the horizontal travel of splashed spores, such as wind and secondary splashing by rain, tends to flatten the dispersal gradient and thus the disease gradient. The steepening of the negative slope between April 3 (Fig. 2-A) and May 7 (Fig. 2-B) may have been due to conditions less favorable to dispersal that occurred during the corresponding infection period, 4-6 weeks earlier. The corresponding May 7 infection period had much less rainfall (1.14 cm vs. 3.18 cm) and there was snow cover for 5 days (20). The slopes of the disease gradient from May 28 to July 11 (Fig. 2-C, D, E) became progressively flatter as the season advanced because plants close to the inoculum source had progressively less uninfected tissue available for infection. Because of this factor, slopes of late-season disease gradients are much flatter than the slopes of corresponding dispersal gradients.

The effective dispersal range of *C. herpotrichoides* conidia was only 3-4 ft (Fig. 3). This fits the rain-splash patterns of Gregory et al. (8) who concluded that the probability of infection from rain-splashed propagules dispersed in still air was less than 0.1% at distances of more than 1 m from the inoculum source. The restricted spread of this fungus from point sources of inoculum indicates that these sources must occur at no greater than 1-2 ft intervals in the field for uniform and extensive disease development to occur.

The effect of distance from the inoculum source on the time necessary to attain a 50% infection level was greater for plants than tillers (Fig. 3). This occurred because tillers may be infected not only by conidia from primary inoculum sources but by direct contact with other infected tissues on the same plants. In wheat, new tillers are produced from meristems located between the leaf sheaths of existing tillers and easily become infected as they emerge from behind infected tissues.

The short dispersal range observed for *C. herpotrichoides* indicates that footrot epidemics do not result from air-borne inoculum naturally dispersed over long distances, but from short-range dispersal of rain-splashed conidia produced on numerous uniformly distributed primary inoculum sources already present within the field. Long-range spread of inoculum within fields probably occurs only as a result of tillage and between fields as a result of wind blown infected debris. Because of this requirement for a high density of primary inoculum sources, on the soil surface, any cultural practice which significantly reduces the amount of infected stubble carried over from previous years should reduce the subsequent severity of footrot.

Infection rates.—The steep disease gradients from point sources of primary inoculum observed in this study suggest that footrot is a "simple interest" disease (21) and that secondary inoculum is not involved in epidemic development. Calculations of "apparent infection rates" using Van der Plank's (21) equations supported this hypothesis. The "apparent infection rate" (r) is defined by Van der Plank (21) as the increase in the amount of disease over time. When the proportion of disease (x) is plotted against time, an S-shaped curve results. This can be transformed to a straight line by using the logit transformation $\log_e |1/(1 - x)|$ in the case of "simple interest" diseases. The apparent infection rate is then defined as the regression coefficient of $\log_e |1/(1 - x)|$ on time and is herein given as per unit per day.

When percent infection data were converted to logits and plotted against time, an increase in slope was noted at about mid-May at all sample distances (Fig. 4). Considering the 1-2 week lag between an increase in temperature and an increased sporulation rate (16) and the 4-6 weeks necessary for symptom development, this increase in r corresponds to an increase in average temperatures favoring sporulation and infection that began in late February (Fig. 5). Apparent infection rates were computed for all sample distances by calculating regression lines on either side of the natural break that occurred in mid-May (Fig. 4). Since the resulting r-values

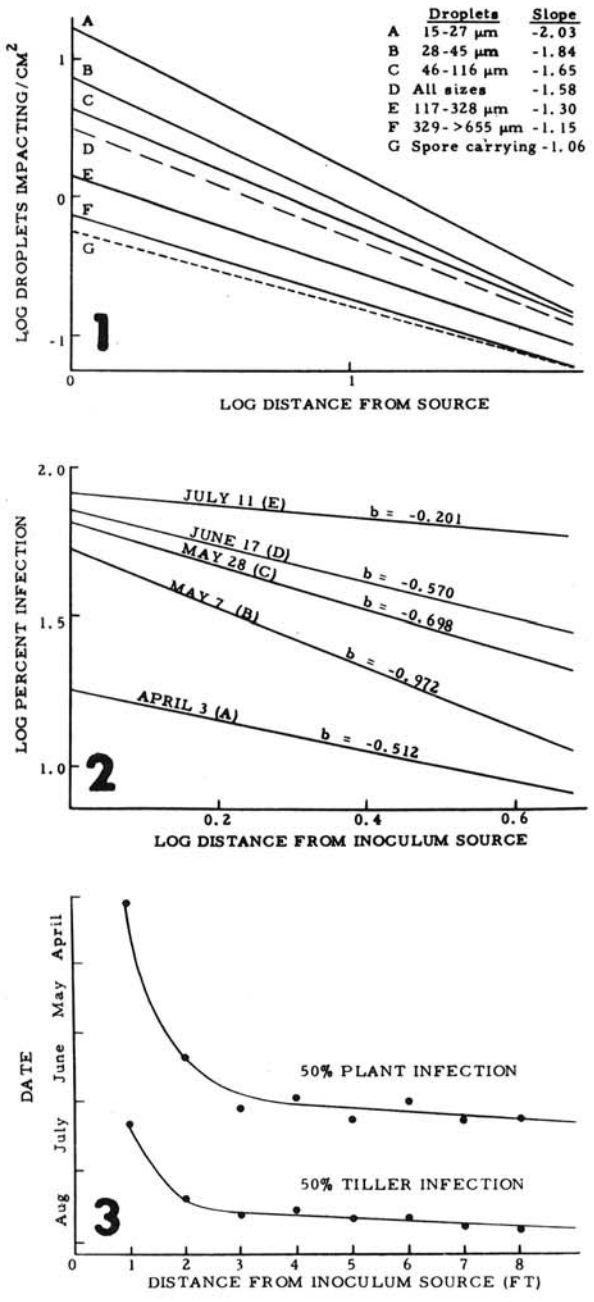

Fig. 1-3. 1) Splash droplet dispersal gradients as a function of droplet size and spore-carrying ability [based on data from Gregory et al. (8)]. **2)** Footrot disease gradients from point sources of inoculum at five points in time. **3)** Effect of distance from the inoculum source on the date when 50% footrot infection was observed on plants and tillers.

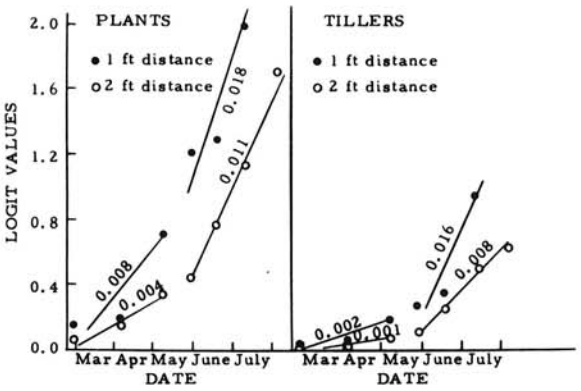

Fig. 4. "Apparent infection rates" (r) for footrot in plants and tillers at two sample distances from the inoculum source.

did not differ significantly ($P = .05$) with distance from the inoculum source, the lines were assumed to be parallel, and the value of r was taken as the pooled regression coefficient of the individual r-values (Table 1).

Analysis of variance of the individual r-values showed that the increase in r observed in late spring, in plots where the inoculum was not removed, was significant ($P = .01$) in all cases. Removal of inoculum in early April effectively reduced or eliminated this increase. Because removal of the primary inoculum source affected r in this manner, the contribution of secondary inoculum to the total inoculum available at the time of removal was of little significance. The apparent inability of *C. herpotrichoides* to produce significant amounts of secondary inoculum is additional evidence that footrot is a "simple interest" disease.

Comparisons between the early spring r-values and those late spring values from plots where the inoculum was removed revealed a significant difference ($P = .01$) only in the case of tiller infection. The slight increase in r from 0.001 to 0.004, even though inoculum had been removed, may have been due to secondary spread of the infection from tiller to tiller by direct contact.

The low "apparent infection rates" calculated for footrot in this study indicate that long periods of time favorable for dispersal and infection are required for epidemic development. The maximum r-value obtained was only 0.011. Beaver (1) calculated r-values ranging from 0.05 to 0.15 for stripe rust caused by *Puccinia* striiformis. Van der Plank (21) reported r-values of 0.08 to 0.55 for leaf rust (*P. recondida*) and 0.46 as a high value for stem rust (*P. graminis*).

Infection periods.—In eastern Oregon, conditions favorable to dispersal and infection occur sporadically during October and November and from late February through mid-May. The dependence of epidemic development on the occurrence of long dispersal-infection periods is substantiated by field observations made during the springs of 1970 and 1971 at La Grande (Fig. 5-A and B). In 1970, temperatures remained favorable for sporulation from late February to early May, and footrot was severe. However, in 1971 temperatures generally remained cold until late March and then rapidly warmed, limiting the amount of sporulation that occurred, and the epidemic was mild. In both cases, peak infection levels developed only late in the season as a result of spring infections.

Early seeding in September increases the incidence of footrot (10, 15). Wheat seeded in late October, however, largely escapes infection because the fall sporulation peak is usually over before the wheat emerges and reaches a susceptible stage. Late seeding aids in the control of footrot by progressively eliminating the fall infection period, leaving disease development in late seeded wheat dependent upon weather patterns that occur from late February through early May.

TABLE 1. The effect of removal of primary inoculum (*Cercosporella herpotrichoides* - infected oat seed) on "apparent infection rates" (r) of footrot of wheat

Sampling time	Pooled r-values	
	Plant infection	Tiller infection
Early spring (prior to inoculum removal)	0.005	0.001
Late spring (inoculum present)	0.011	0.007
Late spring (inoculum removed in early April)	0.003	0.004

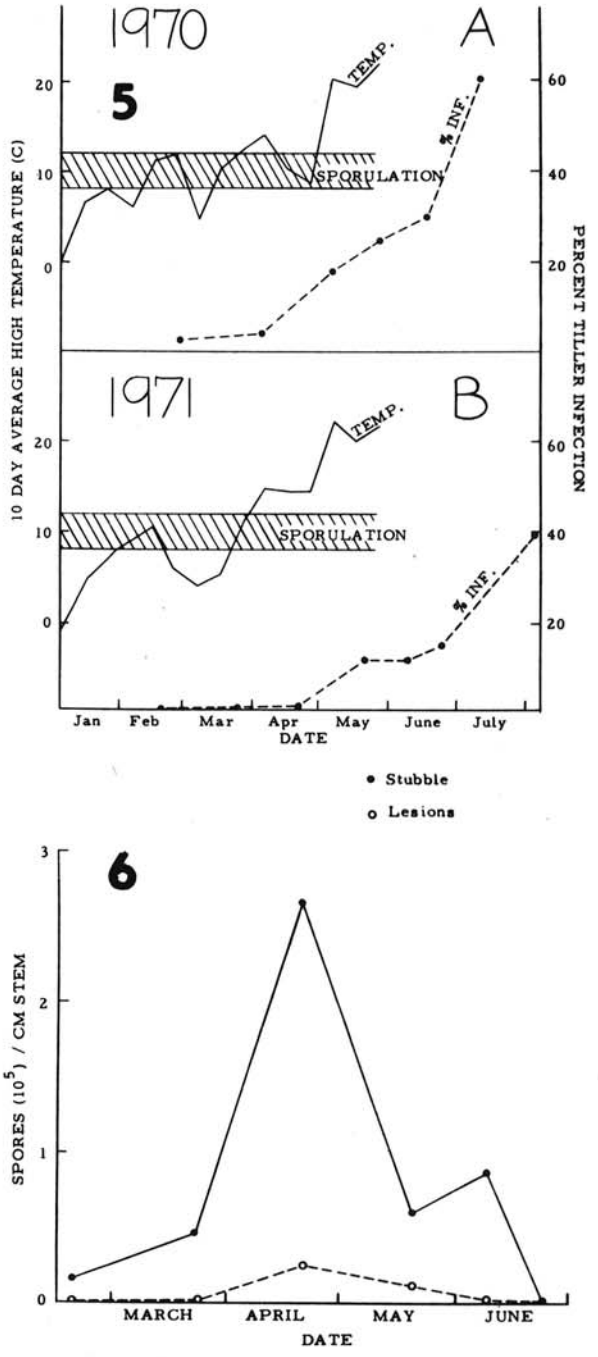

Fig. 5-6. 5) The effect of temperatures on the development of footrot. **6)** Sporulation by *Cercosporella herpotrichoides* on naturally infected stubble and current-season lesions.

Production of primary and secondary inoculum.—The relative production of spores from previously infected stubble (primary inoculum) and current-season lesions (secondary inoculum) was evaluated in the field. Infected stubble was collected from a harvested field and placed in three locations in a winter wheat field that was known to have a high level of natural infestation. Beginning in February 1971, monthly samples were taken from the infected stubble, and from infected plants selected at random throughout the field. Sections 2-cm long were cut from the infected tissue and put into individual vials, to which 0.5 ml of distilled water containing 10% Fabil stain (11) was added to stain and fix the spores. The vials were then agitated for 15 seconds on a Vortex mixer to wash the spores from the plant tissue. The number of spores present in the wash water was determined using a haemocytometer.

No spores were found on developing lesions until late April when a small peak was detected (Fig. 6). Spore production at this time was only 9.2% of that produced on stubble. The few spores produced on current lesions would also be less subject to rain-splash due to their location at the soil line under a protective canopy of leaves.

The development of epidemic levels of footrot is thus dependent upon the production of large amounts of primary inoculum on infected stubble remaining from previous wheat crops. Because of the low "apparent infection rate" of this disease and the restricted air-borne dispersal of conidial inoculum, high levels of Cercosporella footrot will develop only if adequate sources of primary inoculum are available and long periods of favorable weather occur.

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