Transmission of *Phytophthora infestans* From Infected Potato Seed Tubers to Emerged Shoots

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**ABSTRACT**


Transmission of *Phytophthora infestans* from infected seed tubers to emerged potato sprouts, infectivity of sporangia deposited on whole tubers before burial in soil, and infectivity of sporangia in a loamy fine sand to leaflets were investigated in the greenhouse under simulated spring planting conditions of the Columbia Basin. Incidence of late-blight-infected shoots from infected seed tubers was significantly greater when foliage was exposed to wet periods in mist chambers (mist for 45 s every 15 min) for either 24 or 48 h than when not exposed to a wet period. Proportion of infected shoots from infected tubers was 0.210 to 0.261 in a moist environment versus 0.013 to 0.052 in a nonmist environment. Development of chlorosis, necrosis, and sporangia occurred on shoots that emerged from infected, symptomatic tubers buried in soil. However, approximately 20% of the infected shoots produced sporangia before stems had visible discoloration of late-blight symptoms. Sporulation was sparse and formed near the soil line on some of the shoots after 24 h in the moist environment. The latent period or time from inoculation to sporulation on young stems of Russet Burbank was 5 to 6 days, which is too long to account for an infection from either sporangia or zoospores at the soil level of shoots during the wet period in this study. Sporangia were infective when placed directly on eyes of whole tubers before planting. Leaflets touching a loamy fine sand infested with sporangia developed typical late-blight lesions beginning at the leaflet tip within 7 days after a 24-h wet period and the infested loamy fine sand was infective when splashed on leaflets.

*Phytophthora infestans* (Mont.) de Bary, the cause of potato late blight, survives in tubers which may act as an inoculum source the following season (26,38). In the Pacific Northwest of North America, oospores of *P. infestans* are not currently known to be a factor in overwintering, and a continuum of viable host tissue is essential for overwintering and transmission of *P. infestans* to new plant tissues (26,38,39). Infected tubers are considered to be the main means of overwintering for the pathogen. Infected tubers serving as overwintering and primary inoculum sources may be (i) potato seed tubers (9,19), (ii) potato tubers left in the field after harvest that produce volunteer plants (12,39), and (iii) cull potato tubers (6,21,39). Infected potato seed tubers, cull piles, and volunteer potato plants have all been important primary source of inoculum in the Columbia Basin of Washington and Oregon (17; unpublished). Latently infected tubers have been implied from an epidemiological study in the Ecuadorian highlands (23) and demonstrated by detecting the pathogen in asymptomatic tubers with the polymerase chain reaction (PCR; 1,2,15) and by inducing symptom development and sporulation on potato seed tubers after long-term cold storage (18). The relative importance of the three types of late-blight-infected tubers likely varies and depends, in part, on microclimates, local conditions, and the extent of infection the previous fall (9,12,39).

Transmission of *P. infestans* from infected tubers to plant tissues of the next season’s crop may occur during seed-tuber handling, cutting, and planting (25) or in the field (14,26). The potential for one or more cycles of infection has been demonstrated when seed tubers are handled and cut (25). For secondary infection to occur during cutting and handling, the pathogen must survive in intact tubers during the winter, sporulate, be dispersed, and infect additional tubers or plants. *P. infestans* produces sporangia on tubers (11,12,25,29), and sporulation has occurred within 19 h after seed tubers with late-blight symptoms were removed from cold storage to a warmer environment at high humidity (32). Sporangia were readily transmitted by direct contact from infected tubers or seed pieces to noninfected seed pieces (11). Infection during seed-tuber cutting and handling likely increases the threat of late-blight outbreaks on foliage in the field (25). Fungicide seed piece treatments potentially reduce transmission on seed tubers (16,34).

The exact pathway by which *P. infestans* progresses from planted, infected seed tubers to the new plant is not clear and is disputed (3,7,26). *P. infestans* is thought to spread by mycelial growth within infected seed tubers, advancing contiguously or following growing shoots to produce sporangia capable of generating new infection foci (10,26,38). This pathway was recently confirmed when *P. infestans* was detected in asymptomatic shoots emerging from infected tubers with the aid of the PCR (1,2,4,15). Latent infections in tubers and emerging shoots pose additional challenges in managing late blight.

Emergence of infected shoots from infected seed pieces is often low, and infected seed tubers frequently result in a reduced stand due to tuber rot and pre-emergence blighting of shoots (7,14,26). For example, over five consecutive years, only 21 of 3,260 (0.64%) infected seed tubers planted produced infected above-ground shoots capable of sporulating (14). In experiments in Oregon and Washington (27), transmission from artificially infected seed pieces to emerging shoots was 1.9 to 3.8% of the inoculated seed piece, depending on the cultivar. In a recent study, tuber-to-sprout transmission was as high as 25% on plants held at 60 to 90% relative humidity in the greenhouse. Transmission was greater with a US-8 than US-11 isolate (12). Temperature and moisture have not been directly associated with transmission of *P. infestans* from tubers to shoots (7,14); however, rainy and cloudy weather may have favored the extension of *P. infestans* from infected seed-tubers to aboveground shoots during 2 years in a field study (14). In preliminary experiments in my laboratory, infected shoots arising from infected seed tubers were more numerous when incubated in a mist chamber than when kept outside of a moist environment. Environmental conditions that may promote development of infected shoots from the infected seed tubers have not been identified. Understanding factors that promote pathogen transmission from seed tubers to shoots is a major goal in developing management strategies for the disease.

The transmission rate from infected seed tuber to foliage does not need to be very high for a late-blight epidemic to develop, given the explosive polycyclic capabilities
of *P. infestans* and the large amount of potato seed planted in a major production region like the Columbia Basin (14,26, 37,38). For example, the number of seed pieces planted in the Columbia Basin in 2007 was over $2.625 \times 10^9$, with the total weight of seed pieces being 156,300 metric tons. The number of seed potato pieces cut was over $8.7 \times 10^9$, assuming a mean seed piece weight of 60 g, a mean seed tuber size of 170 g, and 5% wastage. Calculations were based on 65,650 ha harvested in the Columbia Basin of Washington and Oregon; approximately 39,970 potato seed pieces planted per hectare when using standard within-row and between-row spacings of 29 and 86 cm, respectively (35); and accounting for planting inefficiencies such as skips and doubles (28). Because of the extremely large ratio of seed tubers planted to those capable of inducing an outbreak, a greater understanding is needed of the transmission of *P. infestans* from potato seed tubers to the succeeding crop.

Sporangia produced on potato seed tubers during cutting, handling, and planting could potentially be dispersed and deposited on noninfected tubers and field soil (23). Transmission of *P. infestans* from infested soil to leaves was demonstrated by Boyd (7); however, infectivity of soils from the Columbia Basin to foliage is not known. In addition, infectivity of sporangia deposited on tuber eyes before planting is not known.

The primary purpose of this research was to investigate the effect of a mist period on the expression of late-blight symptoms on shoots emerging from infected seed tubers. The hypothesis is that air movement following asymptomatic shoot emergence promotes symptom expression and transmission of the pathogen from the tuber. The study was done in a greenhouse under simulated potato-growing conditions representing spring soil temperatures and moisture of the semiarid environment of the Columbia Basin. Secondary objectives were to investigate the infectivity of sporangia deposited directly on tubers before planting, and the infectivity of sporangia in a loamy fine sand to leaflets.

**MATERIALS AND METHODS**

A US-8 isolate of the A2 mating type (BF05) of *P. infestans* was used in these studies. The isolate was obtained from potato foliage collected in northern Idaho in 2005, maintained at 4.2°C in potato tubers of cv. Russet Burbank during winter months of October through March, and transferred to excised leaflets of cv. Russet Burbank during the remainder of the year as previously described (31). Inoculum was increased on inoculated leaflets placed on a nylon mesh screen over moistened paper towels in a glass tray (humidity chamber), sealed in a polyethylene bag, and incubated at 15°C for 7 days (31).

Certified seed tubers of cv. Russet Burbank used in this study were produced near Ronan, Montana, where late blight was not known to be present, and were assumed to be free of the pathogen. Tubers were stored at approximately 4°C and warmed to 15°C before inoculation.

Tubers were inoculated with *P. infestans* using one of two methods. With the first inoculation method, the concentration of sporangia in a water suspension was adjusted to 10,000 sporangia/ml of distilled water using a hemacytometer. Sporangia were then chilled for 2 h at 4°C to induce zoospore formation. Inoculation was done by applying 0.05 ml of inoculum with a micropipette to a Whatman no. 2 filter paper cut into 10-by-10-mm squares and then placing the saturated filter paper square onto a single eye on a tuber or onto an excised leaflet. Inoculated tubers were then placed in a mist chamber for 20 h at 16 to 20°C and allowed to air dry for 1 h. A filter paper square saturated in distilled water was placed over a single eye of tubers used for the noninoculated control.

The second inoculation method was done to determine the infectivity of sporangia placed directly on tubers before planting. Inoculum was produced on excised leaflets of Russet Burbank as described previously. Inoculations were done by applying sporangia directly onto tuber tissue by gently tapping the leaflet containing sporangia on a certified seed tuber of Russet Burbank. The leaflets used for inoculation did not have visible, condensed moisture. Tubers that did not receive sporangia from an excised leaflet were used as noninoculated controls. Inoculated and control tubers were placed in a greenhouse potting mix (LCL1 Soil Mix; Sun Gro Horticulture, Canada Ltd.).

Experiments in the greenhouse were done in the spring and summer months and artificial lights were not used. Temperatures in the greenhouse were 20 to 24°C during the day and 10 to 19°C during the night. Relative humidity ranged from 20 to 45%.

**Transmission from infected seed pieces to shoots.** Emerged shoots from infected tubers were used to investigate the role of a mist period on the expression of late-blight symptoms on shoots. Infected tubers were produced by inoculating certified seed tubers. Seed tubers were inoculated at a single eye near the stem end of tubers using the filter paper method and placed in a mist chamber as previously described. Tubers were then planted in a potting mix in 3-liter pots, covered with approximately 4 cm of the potting mix, and placed on a greenhouse bench. When shoots had emerged and grown to approximately 3 to 14 cm in height, randomly selected pots with shoots were placed in a mist chamber for 24 h at 17 to 20°C. Shoots were examined for sporangia and blighting after each 24-h wet period.

In a set of seven trials in 2007, 4 to 17 pots with emerged shoots were either placed in the mist chamber (mist for 45 s every 15 min) for 24 h or left in the greenhouse (Table 1). In a second set of six trials in 2007, 12 to 17 pots with emerged shoots were placed either in the mist chamber for 48 h or left in the greenhouse (Table 2). In a set of three trials in 2008, 3 to 5 pots with emerged shoots were either placed in a mist chamber for 24 h or left in the greenhouse (Table 1). Shoots were individually examined for sporangia with a x10 power hand lens and late-blight sympotms immediately after each wet period. Each of the 16 trials was done on a separate day.

Pots with shoots were then returned to the greenhouse bench and again assessed for late-blight symptoms 24 h after the termination of the 24- or 48-h wet periods. Control shoots that had not been subjected to a mist period on the expression of late-blight symptoms on shoots...
to the wet period were also assessed for late-blight symptoms and sporangia. In four trials in 2007, some shoots did not have symptoms immediately after the wet periods but had late-blight symptoms 24 h after the termination of the wet period (Table 3). These four trials were combined for a fourth set of trials for data analysis.

The potting mix around the below-ground segment of eight symptomatic shoots from three trials was gently extracted and the shoot was removed from the seed tuber. The stem tissue from the seed tuber upward was then examined for external discoloration.

**Infectivity of sporangia deposited on tubers before planting.** Tubers were inoculated before planting to determine infectivity of sporangia deposited directly on tubers before planting. Sporangia were applied to 20 whole tubers by tapping a leaflet containing sporangia on tubers. Five tubers were not inoculated and were used as controls. Tubers were planted (1 tuber/pot) in the potting mix in 3-liter pots (15 cm in diameter) and placed on a greenhouse bench for 3 weeks. Depth of potting mix over the top of the tuber was 4 cm. Moisture content of the potting mix was 50%, as determined by weighing samples before and after drying in an oven. Distilled water was first applied to each of five pots at planting, 7 days after planting, 14 days after planting, or not applied. Distilled water was applied at planting to the noninoculated controls. Tubers were assessed for infection 6 weeks after inoculation. The trial was repeated.

**Infectivity of infested soil.** A Quincy loamy fine sand from sagebrush land adjacent to a potato field east of Paterson, WA was used to determine infectivity of infested soil because it was assumed to be free of *P. infestans*. Sporangia were produced on excised leaflets, washed from leaflets, adjusted to 10,000 sporangia/ml of distilled water and then chilled for 2 h at 4°C. Approximately 50 ml of the sporangium suspension was added to 150 cm³ of the soil. The infested soil was placed on top of noninfested fine loamy sand contained in two 15-cm-diameter, 3-liter pots that were nearly filled to capacity. The depth of the infected soil was approximately 4 mm. The pots with infested soil were placed in a mist chamber with a leaflet tip from an adjacent, noninfected, potted Russet Burbank plant touching the infested soil. One leaflet tip was touching infested soil in each of the two pots. A small aggregate of infested loamy fine sand about 0.5 mm³ in volume was placed on the center of a leaflet on a separate leaf to simulate sand that had splashed on to the leaflet. The plants were left in the mist chamber for 24 h at 18 to 21°C, then placed on a greenhouse bench at 13 to 23°C for 9 days. Plants that developed lesions were placed again in the mist chamber for 24 h at 18 to 21°C to induce sporulation from developed lesions. This experiment was conducted three times.

**Data analysis.** The incidence of infected tubers with symptomatic shoots and the proportion of symptomatic shoots per total number of infected tubers were used to determine the development of symptomatic shoots from infected tubers. Data for development of symptomatic shoots immediately after termination of the wet period were used for the first three sets of trials and data for development of symptomatic shoots 24 h after termination of the wet periods were used for the fourth set of trials. Data for each of the discrete trials were calculated separately within the four sets of trials. The individual trials were considered replications within each of the four set of trials or experiments. Data from the four sets of trials were analyzed separately because they were done at different times and not intended for comparison (Tables 1, 2, and 3). Each of the four sets of trials was analyzed as a two-way analysis of variance (ANOVA), where independent variables were the contiguous trials (replications) and the treatments were mist and nonmist periods. Data for incidence of infected tubers with symptomatic shoots and the proportion of symptomatic shoots per total infected tubers were analyzed using an arcsine square root transformation to best satisfy variance assumptions. PROC MIXED procedure (SAS Institute, Carey NC) was used to test for significance among trials and treatment effects for each of the four experiments. Comparison of treatment means was based on the least square means from the ANOVA model using PROC GLM in SAS (version 9.1; SAS Institute).

Data collected to investigate the infectivity of sporangia deposited on tubers before planting were analyzed with regression analysis using REG. Incidence of infected tubers (dependent variable) was considered to change over time of first watering (independent variable) if the regression coefficient (slope) was significantly greater than 0 at P < 0.05. The treatment not receiving water was not included in the analysis because zero was not defined by number of days for first watering. The trials to investigate the infectivity of a loamy fine sand infested with sporangia on potato leaflets was observational and statistical tests were not done to test hypotheses. Koch’s postulates were used to demonstrate infection.

### RESULTS

**Transmission from infected seed pieces to shoots.** The incidence of infected tubers with symptomatic shoots was greater when foliage was exposed to a 24-h wet period than when not exposed in 2007 (P = 0.002) and 2008 (P = 0.001) (Table 1). The proportion of symptomatic shoots per infected tubers was greater when foliage was exposed to a 24-h wet period than

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**Table 2. Incidence of infected and symptomatic tuber and shoots when healthy-appearing shoots from Russet Burbank seed tubers inoculated with *Phytophthora infestans* were placed or not placed in a mist period (45 s of mist every 15 min) for 48 h in six trials in 2007.

<table>
<thead>
<tr>
<th>Trial</th>
<th>Mist</th>
<th>No mist</th>
<th>Proportion of symptomatic shoots</th>
<th>Total no. of infected tubers</th>
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<td>0</td>
<td>0.07</td>
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<tr>
<td>5</td>
<td>8</td>
<td>0</td>
<td>0.17</td>
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<tr>
<td>6</td>
<td>17</td>
<td>0</td>
<td>0.17</td>
<td>0</td>
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<tr>
<td>Mean</td>
<td>14.7*</td>
<td>3.8</td>
<td>0.19**</td>
<td>0.052</td>
</tr>
</tbody>
</table>

* Number of shoots with symptoms per total infected tubers.
* For difference between mist and no mist, * and ** indicate P = 0.042 and 0.020, respectively.

**Table 3. Incidence of infected tubers and symptomatic tubers and shoots when healthy-appearing shoots from Russet Burbank seed tubers inoculated with *Phytophthora infestans* were placed or not placed in a mist chamber (45 s of mist every 15 min) for 48 h and then placed on a greenhouse bench for 24 h in four trials.**

<table>
<thead>
<tr>
<th>Trial</th>
<th>Mist</th>
<th>No mist</th>
<th>Proportion of symptomatic shoots</th>
<th>Total no. of infected tubers</th>
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<tr>
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<td>2</td>
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<td>0.33</td>
<td>0</td>
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<tr>
<td>3</td>
<td>9</td>
<td>0</td>
<td>0.18</td>
<td>0</td>
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<tr>
<td>4</td>
<td>10</td>
<td>0</td>
<td>0.30</td>
<td>0</td>
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<tr>
<td>Mean</td>
<td>22.8*</td>
<td>0</td>
<td>0.43**</td>
<td>0.029</td>
</tr>
</tbody>
</table>

* Number of shoots with symptoms per total infected tubers.
* For difference between mist and no mist, * and ** indicate P = 0.032 and 0.029, respectively.
when not exposed in 2007 ($P = 0.002$) and 2008 ($P = 0.008$) (Table 1). The incidence of infected tubers with symptomatic shoots was greater when foliage was exposed to a 48-h wet period than when not exposed ($P = 0.042$) (Table 2). The proportion of symptomatic shoots per infected tubers was greater when foliage was exposed to a 48-h wet period than when not exposed ($P = 0.020$) (Table 2). All of the symptomatic shoots from the mist chamber supported sporangia of *P. infestans*. In all, 9 of 40 (22.5%) infected shoots in the mist environment produced sporangia before stems had visible discoloration. Sporulation was light and formed near the soil line on some infected shoots at 24 h (Fig. 1). Infected shoots were not produced from noninoculated control seed tubers.

Additional symptomatic shoots became evident within 24 h of removing plants from the mist chamber in four trials in 2007. The incidence of infected tubers with symptomatic shoots was greater when foliage was previously exposed to a wet period than when not exposed ($P = 0.032$; Table 3). The proportion of symptomatic shoots per infected tubers was greater when foliage was previously exposed to a wet period than when not exposed ($P = 0.029$; Table 3). The control plants not placed in the mist chamber did not exhibit symptomatic shoots in any of the trials.

Discolored stem tissue began about 1 cm or less below the soil line and continued up into the aboveground stem in the eight symptomatic shoots examined for belowground tissue discoloration. The remaining belowground tissue down to the seed piece remained asymptomatic in seven of the examined shoots. Reddish-brown discoloration appeared on the belowground stem of the eighth shoot.

**Infectivity of sporangia deposited on tubers before planting.** Whole tubers became infected when sporangia were placed directly on eyes before planting. The percentage of infected tubers was 100, 80, 100, and 60 when pots were first watered at time of planting, 7 days after planting, 14 days after planting, and not watered, respectively. When the trial was repeated, the percentage of infected tubers was 40, 60, 20, and 0 when pots were first watered at time of planting, 7 days after planting, 14 days after planting, and not watered, respectively. Percentage of infection did not significantly change with time of first watering in first ($P = 1.00$) and second trials ($P = 0.089$). Typical late-blight symptoms developed in infected tubers, and sporangia of *P. infestans* later developed on tubers tissues placed in a humid chamber at 15°C. The noninoculated control tubers did not become infected in either trial.

**Infectivity of infested soil.** All six leaflets touching infested soil during the three experiments developed typical late-blight lesions beginning at the leaflet tip within 7 days after the wet period. In addition, late-blight lesions developed in the leaflets where a small aggregate of infested loamy fine sand was placed within 7 days after the wet period. Initial lesions were usually observed 6 days after inoculation. Sporangia of *P. infestans* developed in and around the lesions when potted plants with the lesions were placed in a mist chamber for 24 h after the lesions had developed. Late-blight lesions did not develop on six leaflets used as noninoculated controls.

**DISCUSSION**

**Mist periods promoted transmission of *P. infestans* from infected seed tubers to emerged shoots.** Healthy-appearing shoots developed late-blight symptoms during a 24- to 48-h mist period or within a day after shoots were exposed to a wet period. Mean proportion of shoots expressing symptoms after emergence ranged from 21 to 42 per 100 infected tubers (Tables 2 and 3). Expression of symptomatic shoots has not been consistent in previous studies (7,14,26), likely because the effect of moisture on blighted shoot expression was not fully recognized. This result may also explain why transmission rates from infected tubers to foliage are generally low under arid conditions (14).

De Bary was the first to summarize that “the vegetation” of *P. infestans* was “largely hastened and assisted by damp, and on the other hand, retarded by drought” (10). The validity of De Bary’s work on vegetation of the pathogen was then questioned when not duplicated by other researchers, as described by Melhus (26). In addition to promoting expression of infected shoots, moisture favored production of sporangia on the emerged shoot. Sporangia were also sometimes produced on shoots before lesions appeared. Wet periods at mild temperatures not only promote expression of blighted shoots from infected seed tubers but also favor repeated cycles of sporulation and infection on foliage in the field. Late blight is highly dependent on moisture for sporulation and infection, and results from this study imply that moisture also promotes initial epidemic development.

Determining the initial source of late-blight infection after overwintering is difficult once potato plants in a commercial field are infected (14). The dilemma has been previously summarized in that, by the time a group of infected plants is noticed in a field, it is usually too late to decide how the infection originated (8). This observation is more understandable since the present research determined that sporulation may precede late-blight symptoms of chlorosis and necrosis on shoots arising from infected seed pieces and also that initial sporulation may be sparse, making early detection extremely difficult. As a result, one or more secondary cycles of infection often occur before symptomatic plants are observed in commercial fields (37,38). An advantage of evaluating transmission of *P. infestans* from seed tubers in a greenhouse over trials in the field is that moisture was regulated and that plants could be readily observed several times daily.

Sporulation and late-blight symptoms on shoots arising from infected tubers became evident within 24 to 48 h of the start of mist conditions in this study. The rapidity with which sporulation occurred suggests that mycelium of *P. infestans* was likely latently present in the aboveground stem tissue and produced sporangia and late-blight lesions upon exposure to the
wet conditions. The latent period or time from inoculation to sporulation on young stems of Russet Burbank is generally 5 to 6 days for isolate BF05 (unpublished data), which is too long to congruously account for an infection being initiated from either sporangia or zoospores at the soil level of shoots during the wet period in this study. Latently infected shoots arising from infected tubers have been detected using PCR (4). Melhus reported that mycelium of P. infestans spreads most rapidly in the cortical tissues of the stem, where it travels more rapidly up than down, and that the pathogen progressed more rapidly in young than in old tissues (26).

Necrotic tissue was not observed on the belowground stems from the infected seed piece to the symptomatic aboveground stem tissues of seven examined stems in this study. Lesions on belowground stems arising from infected seed tubers have been previously observed and were expected in this study (14,37,38). However, basal necrosis and necrosis beginning “some distance up the stem” (20) and plants with asymptomatic belowground stems and symptomatic aboveground stems have been previously observed on stems growing from planted infected seed pieces (7). Healthy-appearing belowground stem tissue between an infected seed piece and a lesion on an aboveground stem is shown in a figure in a recent article on late-blight management (33). Some researchers have not excavated belowground stems from soil of stems with aboveground symptoms to observe whether there was continuity of symptoms between the aboveground stems lesion and the belowground stem. Boyd (7) suggested that stems were infected near the soil surface by zoospores in the soil. Symptoms in this study appeared too rapidly after the beginning of the wet period to have originated from spores in the soil, given a 5- to 6-day incubation period. A possible explanation is that mycelium of P. infestans was latently present in the below- and aboveground stem tissue but the belowground stem tissue was less susceptible to the development of necrosis. Development of necrotic tissue on belowground stem tissues would increase the likelihood of girdling and shoot death before sporulation on the aboveground stems, where sporangia could be dispersed in air currents and infect additional plants. Strains of the pathogen that do not rapidly girdle belowground stems from a latent infection would have a survival advantage over those that rapidly killed belowground stems. Additional work is needed to conclusively demonstrate the pathway of transmission from planted, infected seed tubers and to support a hypothesis that necrosis may be retarded on belowground, latently infected stems.

Sporangia produced on tubers during cutting and handling can potentially become airborne and be carried by air currents to cut tuber surfaces or tuber eyes within a handling and cutting facility or to plants and soil in a neighboring field, resulting in additional infections (25). Sporangia were also placed directly on tuber eyes were infected when tubers were planted. Cut tuber surfaces contain visible free water and are readily infected by sporangia directly deposited on them (unpublished data). Sporangia on tuber eyes may survive several days to weeks and later cause infection when the tubers are planted. Potato handling and cutting facilities are generally enclosed, blocking incoming solar radiation, which could also increase longevity of sporangia (36).

Infested loamy fine sand was infective when in contact with leaves during a wet period. Infection of foliage may also occur from splashing soil during wet periods. Efficiency of infection from contaminated soil appeared high, in that leaflets of all inoculated leaflets become infected in this study. Sporangia remained viable for 23 to 30 days in soil in the Columbia Basin in a previous study (31) and contaminated soil in the spring could serve as a source of initial infection for late-blight epidemics. This may explain field observations of initial lesions of late blight developing at the leaf tip touching the soil surface without other known infected plants nearby (7). Soil may become infected from sporangia produced on tubers or foliage in cull piles or on seed tubers during handling and cutting. Sporangia could then become airborne and be carried on air currents and deposited in a field (5). Sporangia can then survive in the soil and surface water in the field for 3 weeks (24,30,31). Recent research in the Andean highlands where potato plants are cropped at various growth stages throughout the year demonstrated that sporangia of P. infestans can be deposited on field soil from infected foliage in adjacent fields and preemergently infect potato sprouts (23).

Inoculum originating from infected seed tubers in commercial fields can be especially devastating because of the potential earliness of the initial inoculum, the rapidity with which it can be produced, and the proximity of inoculum to the crop. A large number of seed tubers are planted in major potato production regions and only a few infected tubers are needed to initiate an epidemic (14,26,37,38). A few infected shoots arising from infected seed pieces in a commercial field is below the perception threshold and will not likely be noticed during the early stages of epidemics. Common isolates of P. infestans, particularly the US-8 strain, are highly aggressive and rapidly rot tubers, limiting the availability of viable host tissue (19,22,28).

Tubers infected during handling and cutting just prior to planting may be more likely to produce viable shoots than those infected in the field near harvest because...