Colonization of Potato Plants after Aerial Infection by *Colletotrichum coccodes*, Causal Agent of Potato Black Dot

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ABSTRACT

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Colonization of potato plants by *Colletotrichum coccodes* after artificial inoculation of stems was modeled in relation to the severity of chlorosis and necrosis on foliage using logistic regression. *C. coccodes* internally colonized the stems up to 24 cm above the inoculation court (mean of 11.3 ± 7.7 cm and 14.1 ± 9.3 cm in 2003 and 2004, respectively), and completely colonized the stem below the inoculation court including the roots (mean of 13.2 ± 3.1 cm and 12 ± 2.3 cm in 2003 and 2004, respectively). *C. coccodes* grew significantly faster from the inoculation court toward the roots, stolons, and tubers than toward the plant apex. Internal stem colonization was restricted before senescence but rapidly increased as the plants started to senescence, suggesting that physiological events associated with plant senescence activated fungal growth and plant colonization. The results showed no relationship between *C. coccodes* growth and plant colonization and the development of chlorosis and necrosis severity of leaves. The severity of chlorosis and necrosis on foliage in this study was the outcome of natural plant senescence, and was not caused by the fungal colonization. Therefore, chlorosis and necrosis severity is an inaccurate tool to diagnose infection in potato by *C. coccodes* and does not indicate the presence or amount of *C. coccodes* in potato plants. Disease assessment for *C. coccodes* in potato plants should not rely on severity of chlorosis and necrosis. Black dot assessment should be carried out by isolation and quantification of the fungus from stem and root tissues or by polymerase chain reaction techniques before plant senescence.

Potato black dot, caused by *Colletotrichum coccodes* (Wallr.) S.J. Hughes (syn. *C. atramentarium* (Berk. & Br. Taub)), is characterized by small black sclerotia on the roots, stems, stolons, and progeny tubers of infected plants (4,8,29). Black dot is common in most potato growing areas in the world (1,5,6,17,20) and may cause up to 30% yield reduction on susceptible cultivars (13,21,26,29).

In the field, infection of potato plants by *C. coccodes* may be associated with soil-borne, tuber-borne, and airborne inoculum, either solely or jointly in different combinations (12,13,18,29). In the Columbia Basin of central Washington, *C. coccodes* has been isolated relatively early in the growing season from below- and above-ground potato stems and from a high proportion of plants by midseason (13,14). Disease symptoms often do not become evident until late in the growing season, and numerous sclerotia develop on plant parts as plants senesce and die (13,18). Latent infections by *C. coccodes* in potato

stems were suggested in an earlier study to explain the early infections and later expression of sclerotia (13).

The consensus from the literature is that soil-borne and tuber-borne inocula are the main inoculum sources for black dot development in potato plants. However, airborne infection may be an important contributor to the initiation and development of the disease. The frequency of black dot development from aerial infection in potato plants under field conditions is not documented. In the Pacific Northwest and other regions with similar semiarid conditions, sandstorms and sprinkler irrigation systems are common. Therefore, a high potential for foliar infection of potato plants is present, as sandstorms are capable of wounding potato foliage, while sprinkler irrigation disseminates conidia and sclerotia via splashing and contributes the requirement of high relative humidity for inoculum germination and infection (2,13).

The effect of aerial infection by *C. coccodes* on black dot development was tested in field and greenhouse trials in Idaho and Washington State (12,13,18). The foliage of Russet Burbank potato plants was sand-blasted and immediately spray-inoculated with a conidial suspension of *C. coccodes*. The inoculated plants developed foliar chlorosis, necrosis, and dark brown to black lesions on leaves, petioles, and stems (12,13,18). The results of the field trials in

Idaho indicated a significant yield reduction of 6.5 t/ha (about 10% in relation to contemporary average yield) (18). Results of the greenhouse trials in Washington State recorded a significant reduction in total yield (19 to 32%) and a reduction of mean tuber weight (29 to 43%) (12). Results of the field trials in Washington State recorded a significant reduction in total tuber weight by 7, 12, and 11% and a significant reduction of yearly U.S. No. 1 potatoes weight by 12, 18, and 16% over 3 years (12).

Little information is available on the colonization process of potato stems after aerial infection by *C. coccodes* and the relationship between colonization and development of the foliar symptoms of chlorosis and necrosis. Such knowledge would be helpful in understanding the development of *C. coccodes* in the field, and in developing disease management strategies for black dot. The present study tested the hypothesis that *C. coccodes* is capable of infecting potato plants via stems and spread internally up and down the stem. The objectives of the study were: (i) to determine the relationship between chlorosis and necrosis severity on potato foliage and *C. coccodes* colonization of the plants; and (ii) to determine the colonization pattern of *C. coccodes* originating from stem inoculation and construct a colonization and disease progress model.

MATERIALS AND METHODS

***C. coccodes* isolates.** The monoconidial isolates 1018 (origin: potato, Washington State, 1990) and 2044 (origin: potato, Washington State, 1997) were used. The isolates were previously assigned to the North American vegetative compatibility groups NA-VCG1 and NA-VCG2, respectively (19), indicating that they were from different genetic backgrounds. The isolates were subcultured onto 1% potato dextrose agar (PDA: 10 g Difco PDA, 15 g Bacto agar, 0.1 g streptomycin sulfate, and 975 ml distilled water), incubated in the dark at 25°C for 7 days, and maintained at 4°C until use.

Plant material. Disease free, nuclear potato tubers of the *C. coccodes*-susceptible cultivar Russet Burbank were used. Tuber pieces (24 g average) were cut from surface-sterilized tubers (1% NaClO for 10 min) and were stored at 10°C for 48 h to allow partial wound healing. A total of

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90 plants were prepared (30 per isolate including noninoculated control plants) by planting one tuber piece per pot in 3-liter pots (16 cm diameter by 18 cm height) in the middle of the pot, about 1 cm below the soil surface. Sunshine LC1 potting mix (Sun-Gro Horticulture Distributor Inc., Bellevue, WA) was used. Twelve grams of Osmocote fertilizer (16-16-16 NPK) were added to the soil at planting. The pots were placed in the greenhouse (20°C, 16 h light), and the plants were allowed to develop until they reached an average height of 20 cm (about 6 weeks).

Preparation of inoculum. The isolates were cultured on 1% PDA in 9-cm petri plates for 7 days at 25°C in the dark. Immediately after incubation, the conidia were harvested with 3 ml of sterile distilled water by scraping the conidia from the agar surface and filtering through four layers of autoclaved cheesecloth. The conidia suspensions were adjusted to a final concentration of 1.3×10^5 conidia per ml using a hemacytometer.

Inoculation of plants. Forty-five day old potato plants that were grown in the greenhouse were injured in the middle of the stem, halfway between the soil surface and the plant apex, with a sterile scalpel. The epidermis tissue was carefully peeled off to form a 5×5 mm wound, which served as the inoculation court. A sterile filter paper (1 × 1 cm) amended with 50 µl of conidial suspension was placed on the wound. The conidial suspension was absorbed by the filter paper to retain the inoculum at the inoculation court. The filter paper was left attached to the plant. Immediately after inoculation, the plants were transferred to a mist chamber for 24 h, and were water-sprayed for 1 min every 10 min (6 sprays per hour) with two atomizers (Herrmidifier Co. Inc., Model 707 TW; Lancaster, PA). After the 24-h wetting period, the sterile filter paper amended with the conidial suspension was removed, and the plants were transferred to the greenhouse and arranged in a completely randomized design structure (CRD). The entire inoculation process, from wounding until plants were transferred into the mist chambers, ranged between 30 to 60 s per plant. The temperature during the wetting period was 20°C. Sterile distilled water was used for noninoculated control plants. The *C. coccodes* isolates were used separately, and coinoculations were not performed.

In 2003, inoculations with both isolates were carried out on 2 September. In 2004, inoculation with isolate 1018 was conducted on 15 June, and with isolate 2044 on 19 June. Temperatures during the trials were $19 \pm 5.1^\circ\text{C}$ and $23 \pm 3.4^\circ\text{C}$ in 2003 and 2004, respectively.

Severity of chlorosis and necrosis. The amount of chlorosis and necrosis, due to either disease development or natural plant senescence, was assessed using a visual

scale from 0 to 6. Plant stems were divided equally into three sections (lower, middle, and apex) to facilitate the assessment. The scale was as follows: 0 = green, healthy appearing plant, absence of chlorosis and necrosis; 1 = moderate chlorosis and flaccid, yellow lower leaves, the middle leaves turgid and mildly chlorotic, and the apex with green leaves; 2 = lower leaves flaccid with severe chlorosis and orange/brownish discoloration, and necrosis on the tips of the leaflets, moderate chlorosis on the middle leaves, and the plant apex with mild chlorosis; 3 = lower leaves with necrosis, all middle leaves with moderate chlorosis and 10 to 30% of them with necrosis, the plant apex with moderate chlorosis; 4 = lower and middle leaves with necrosis or fallen off the plant, and the plant apex with severe chlorosis; 5 = lower and middle leaves with necrosis or fallen off the plant, the plant apex flaccid with severe chlorosis, and the stem soft with dark orange discoloration; 6 = dried plants or plants left in the pots to dry on the greenhouse bench for 10 to 14 days until sclerotia were formed. The beginning of plant senescence was considered when symptoms reached category 3, in general, 100 days after planting for long-season potatoes such as Russet Burbank.

Plant sampling and evaluation of infection. Ninety plants were grown for research purposes in 2003 and again in 2004. The plants were randomly assigned to three groups with 30 plants per group: 1) plants inoculated with isolate 1018; 2) plants inoculated with isolate 2044; and 3) noninoculated plants as the control. Approximately five plants were randomly selected from each treatment group according to the symptom categories of chlorosis and necrosis development, and the time of their expression. In 2003, chlorosis and necrosis developed rapidly. Therefore, plants with symptom categories 1, 2, and 3 were sampled 49 days after inoculation (DAI), or 94 days after planting; and plants with symptom categories 4, 5, and 6 were sampled 64, 77, and 90 DAI, respectively, or 109, 122, and 135 days after planting, respectively. In 2004, plants inoculated with isolate 1018 and control plants were sampled 28, 42, 57, 65, 80, and 87 DAI, or 73, 87, 102, 110, 125, and 132 days after planting; and plants inoculated with isolate 2044 were sampled 24, 38, 53, 61, 76, and 83 DAI, or 69, 83, 98, 106, 121, and 128 days after planting for symptom categories 1, 2, 3, 4, 5, and 6, respectively.

Following harvest, leaves were excised and stems were disinfested (1% NaClO for 10 min). The stems were divided into three sections. The first stem section was the inoculation court (the 5×5 mm segment where inoculation was conducted) that was used as a positive control for infection. The second and third stem sections were the upper section (above the inoculation court) and the lower section (below the inocula-

tion court), respectively. The upper and lower stem sections were dissected entirely into 1-cm segments, progressing by 1 cm from the inoculation court. The roots were not sampled. To prevent potential contamination of healthy stem sections, the scalpel was sterilized between cuttings. The stem segments were placed onto modified PDA (1 g Difco PDA, 20 g Bacto agar, 0.2 g streptomycin sulfate, 5 ml ethanol, and 975 ml distilled water) and were incubated for 10 days at 25°C in the dark. The inoculation court was also placed on modified PDA to confirm infection. On the last five plants, which were allowed to die and dry (symptom category 6), sclerotia were visible without magnification, and the distance from inoculation court that sclerotia were formed was directly measured with a ruler.

Statistical analysis. The fungal colonization of stems was recorded as a binary outcome with 1 = colonized stem segment, and 0 = noncolonized stem segment. Colonization was the response of interest, and since it was recorded as a binary outcome, a multiple linear logistic regression analysis was used to assess its relationship to the predictor variables (11). Proc Logistic in SAS (SAS 9.1; SAS Institute, Cary, NC) was used to compute the coefficients of the logistic regression model, which included the following linear predictors: isolate type (1018 or 2044), chlorosis and necrosis severity (a scale of 1 to 6), distance (cm) from the inoculation court, and direction of fungal colonization (above or below the inoculation court). Fungal colonization was calculated as infection probability (P_i), which indicated the odds of a stem segment to be colonized by the fungus at a certain distance above or below the inoculation court. P_i was calculated as the logarithm of infected segments (P) divided by noninfected segments ($1 - P$), or $P_i = \log(P/1 - P)$. Because the type of isolate and the direction of fungal colonization in the stem were two-level quantitative predictors, these variables were dummy coded: 0 if isolate = 2044 and 1 if isolate = 1018; and 0 = direction down from inoculation court, and 1 = direction up from inoculation court. All inferences were based on a level of significance at $P = 0.05$. Differences in the severity of chlorosis and necrosis and the time of their expression between inoculated and noninoculated control plants were tested using student's t test ($\alpha = 0.05$).

RESULTS

C. coccodes was isolated from 47 of 60 (78%), and 52 of 60 (87%) potato plants that were stem-inoculated in 2003 and 2004, respectively. The fungus did not infect control plants and was not isolated from the controls. *C. coccodes* colonized the stems of infected plants to a maximum height of 24 cm above the inoculation court (mean $\pm$ standard deviation: 11.3 ± 7.7 cm and 14.1 ± 9.3 cm in 2003 and

2004, respectively). The lower stem sections were completely colonized both years (mean $\pm$ standard deviation: 13.2 ± 3.1 cm and 12.0 ± 2.3 cm in 2003 and 2004, respectively). On the dry plants (chlorosis and necrosis severity category 6), sclerotia were observed on both stems and roots of inoculated plants.

C. coccodes was only isolated from inoculated stems; therefore, the logistic regression analysis was only conducted using the data of the inoculated plants. The logistic regression analysis indicated that isolate 2044 colonized the stems significantly ($P = 0.0016$) faster than isolate 1018 in 2003, but not in 2004 ($P = 0.7765$). Isolates 1018 and 2044 had similar patterns of colonization above and below the

inoculation court, and their results are presented jointly (Figs. 1 to 4). In both years, fungal colonization significantly ($P < 0.0001$) decreased as the distance away from the inoculation court increased. Fungal colonization was also significantly ($P < 0.0001$) greater below than above the inoculation court both years (Figs. 1 to 4).

The severity of chlorosis and necrosis and the time of their expression did not vary ($P > 0.05$) between inoculated and noninoculated control plants (Fig. 5). However, fungal colonization had a curvilinear relationship with the severity of chlorosis and necrosis both years. In 2003, the curvilinear relationship had significant ($P < 0.0001$) linear and quadratic regression coefficients. Therefore, the fungal

colonization of the upper stem sections declined from 0.8 to 0.4 as the severity of chlorosis and necrosis progressed from category 1 to 3 (Fig. 1). The colonization of the upper stem sections in 2003 increased to 0.9 as the severity of chlorosis and necrosis exceeded category 3 (plant senescence). The increased colonization is indicated by the upward curvature of the regression surface (Fig. 1). In 2004, the fungal colonization again possessed a curvilinear relationship with the severity of chlorosis and necrosis. However, in 2004, only the linear regression coefficient was significant ($P < 0.0001$), whereas the quadratic coefficient was not significant, but notable ($P = 0.0513$). In 2004, the fungal colonization of the upper stem sections

Fig. 1. Relationship among fungal colonization of the upper stem section, chlorosis and necrosis severity, and distance from the inoculation court in potato plants infected with *Colletotrichum coccodes* isolates 1018 and 2044 in 2003.

Fig. 3. Relationship among fungal colonization of the lower stem section, chlorosis and necrosis severity, and distance from the inoculation court in potato plants infected with *Colletotrichum coccodes* isolates 1018 and 2044 in 2003.

Fig. 2. Relationship among fungal colonization of the upper stem section, chlorosis and necrosis severity, and distance from the inoculation court in potato plants infected with *Colletotrichum coccodes* isolates 1018 and 2044 in 2004.

Fig. 4. Relationship among fungal colonization of the lower stem section, chlorosis and necrosis severity, and distance from the inoculation court in potato plants infected with *Colletotrichum coccodes* isolates 1018 and 2044 in 2004.

(Fig. 2) was restricted between 0.0 and 0.2 as chlorosis and necrosis severity developed from category 1 to 3. Similar to the results of 2003, fungal colonization in 2004 increased from 0.2 to 0.9 (Fig. 2) as the severity of chlorosis and necrosis exceeded category 3.

On the lower stem sections, fungal colonization was similar to that of the upper stem sections. Fungal colonization was restricted until the severity of chlorosis and necrosis exceeded category 3 (plant senescence), after which it greatly increased (Figs. 3 and 4). In contrast to the upper stem sections, colonization of the lower stem sections did not decrease as quickly as distance from the inoculation court increased (Figs. 1 to 4).

DISCUSSION

The similar development of chlorosis and necrosis severity on infected and non-infected control plants (Fig. 5) pointed to the probable hypothesis that natural senescence was the cause of chlorosis and necrosis and not the infection and colonization of the plants by *C. coccodes*. The curvilinear relationship between fungal colonization and chlorosis and necrosis severity pointed to another probable hypothesis that natural senescence significantly affected *C. coccodes* growth and colonization of potato plants. The restricted colonization of plant stems by *C. coccodes* indicated a latency period that was overcome as the potato plants started to senesce. *C. coccodes* colonization was then activated, probably by physiological

events associated with plant senescence that took place in the Russet Burbank potato plants between 95 and 105 days after planting. Comparisons between growth stages of potato plants grown in the greenhouse and in the field should only be made for relative purposes. With this in mind, at 100 days after planting, long-season potatoes, such as Russet Burbank, in the Columbia Basin of Washington State are generally starting the last half of the linear bulking in growth stage 4 (31). During this period, which generally ends at 120 to 130 days after planting, the vegetative growth stops, sugars are translocated to the maturing tubers, and plants start to senesce.

The relationship between *C. coccodes* and plants' senescence has been previously reported in potato and tomato plants (7,18). Mohan et al. (18) noted the lack of foliar symptoms on inoculated potato plants in the field until senescence. Dillard and Cobb (7) demonstrated that isolation of *C. coccodes* from infected plants increased as tomato fruit matured, and Last and Ebben (16) reported a rapid increase in black dot symptoms in tomato roots toward the end of the season. In addition, latent infections in various hosts have been observed for *Colletotrichum* spp. Infection of *C. coccodes* in tomato fruit and of *C. piperatum* and *C. gloeosporioides* in pepper, mango, and avocado demonstrated latency prior to fruit ripening and a break of latency when fruit ripened (3,7,16,22–24,30).

Plant hormones such as ethylene and jasmonic acid are reported to be involved

in the regulation of many diverse aspects of plant development, including organ senescence (10,15,25) and the development of potato tubers (27). Ethylene has also been reported to induce germination and appressorium formation of *Colletotrichum* species (9). The role of plant hormones in black dot development has never been studied. However, it is possible that the increased endogenous levels of ethylene and/or jasmonic acid as plants senesce and/or potato tubers mature and enlarge is the cue that activates *C. coccodes* growth and colonization.

The insignificant quadratic coefficient in 2004 caused the regression surface to have a more linear pattern than in 2003. The difference was most evident as chlorosis and necrosis severity progressed from category 1 to 3. This difference may be either the result of a variation in temperatures ($19 \pm 5.1^\circ\text{C}$ and $23 \pm 3.4^\circ\text{C}$ in 2003 and 2004, respectively) or due to photoperiod. The 2003 trial was conducted during the fall (short days), whereas the 2004 trial was conducted during the spring (long days). *C. coccodes* and consequentially black dot development has been reported to be affected by day length (28,29), which may explain the observed differences in the regression surface between the two years.

Based upon the findings of the present study, we hypothesize the following model of stem colonization after aerial infection by *C. coccodes* in potato. *C. coccodes* may be distributed on leaves and stems of potato and with moisture, spores, and/or scler-

Fig. 5. Development of chlorosis and necrosis severity on potato plants that were either infected or noninfected with *Colletotrichum coccodes* isolates 1018 and 2044 in 2003 and 2004 in the greenhouse.

rotia germinate and the pathogen infects the host. Sandstorms and water from irrigation promote this process (12,13). Infection becomes latent until plants enter growth stage 4, in which tubers bulk and plants start to senesce. Increased levels of endogenous ethylene and/or jasmonic acid may be the cue that activates fungal growth and colonization (10,15,25,27). Latent infection and activation of fungal growth in association with plant senescence may also occur in the presence of soil-borne and/or tuber-borne inocula.

The study suggests that *C. coccodes* infection of potato plants is latent, and that physiological processes in the plants, which are associated with plant senescence, activate *C. coccodes* growth. The results of the present study show no relationship between plant colonization by *C. coccodes* and the severity of chlorosis and necrosis of leaves (Fig. 5). Chlorosis and necrosis severity was found to be an inaccurate tool to diagnose infection in potato by *C. coccodes*, and therefore did not indicate the presence or amount of *C. coccodes* in potato plants. Disease symptoms of black dot in previous field studies have been noted not to appear or to be confused with those caused by *Verticillium dahliae* or natural senescence (14,18,28). The lack of specific disease symptoms on potato foliage due to *C. coccodes* has made detection of the disease and the development of control practices for black dot difficult. Because black dot symptoms are not readily evident on foliage, other methods of disease and pathogen detection are needed, such as examination of roots for cortical rot, and isolation and quantification of the fungus from stems and roots using CFU/g of fresh weight or polymerize chain reaction techniques. Assessment for *C. coccodes* in potato should begin before plant senescence and likely much earlier. Furthermore, contact fungicides applied after initial infections are not likely to effectively reduce disease severity because of latent infection and internal expansion of *C. coccodes* in potato stems. Therefore, systemic fungicides, host resistance, crop rotations, and other disease management practices may be needed to achieve economic control of potato black dot.

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