Interaction of Onion Cultivar and Growth Stages on Incidence of *Pantoea ananatis* Bulb Infection

Spencer Stumpf and **Ron Gitaitis**, Department of Plant Pathology, Coastal Plain Experiment Station, University of Georgia, Tifton, 31793; **Timothy Coolong**, Department of Horticulture, Coastal Plain Experiment Station, University of Georgia, Tifton, 31793; **Cliff Riner**, Area Program Coordinator, Vidalia Onion and Vegetable Research Center, University of Georgia, Lyons, 30463; **Bhabesh Dutta**,[†] Department of Plant Pathology, Coastal Plain Experiment Station, University of Georgia, Tifton, 31793

Abstract

Center rot, caused by *Pantoea ananatis*, has been one of the most important bacterial diseases of onion leading to considerable economic losses. Symptoms can be expressed in the onion foliage and bulb, with the pathogen moving from the infected leaves to bulb scales. However, little is known regarding which growth stage the plant is most susceptible to bulb infection and if there are differences in susceptibility to bulb infection among sweet onion cultivars. In this study, five cultivars of sweet onion (Pirate, Sweet Harvest, 1518, Granex YPRR, and 1407) were inoculated by clipping the tips of onion foliage and depositing 1 ml of 1×10^8 CFU/ml of *P. ananatis* suspension into the central leaf cavity. The inoculations were done at three growth stages (first leaf senescence, bulb initiation, and bulb swelling). Center rot incidence was assessed for precured and cured onion bulbs. In addition, total bulb incidence of center rot for each cultivar inoculated at three growth stages were also calculated. Total bulb center rot incidence was significantly higher for Granex YPRR (84%)

compared with other cultivars. Also, cultivars 1518 (49%) and 1407 (33%) had significantly lower incidence of bulb infection compared with other tested cultivars. Onions were significantly more susceptible to bulb infection when inoculated during first leaf senescence (62%) as compared with bulb initiation (37%) and bulb swelling (31%) stages in precured bulbs ($P = 0.041$). Significantly higher incidence of center rot was observed for bulbs whose foliage were inoculated during first leaf senescence stage (64%) compared with bulb initiation (55%) and bulb swelling (52%) stages ($P = 0.048$). Interactions between onion cultivar and inoculation stage on center rot bulb incidence were not significant ($P \geq 0.218$), when evaluated at different assessment periods. However, different cultivars displayed significant variability in susceptibility to bulb infection. The outcomes of this study may have implications in devising management strategies aimed at protecting most susceptible onion growth stages against *P. ananatis*.

Center rot of onion (*Allium cepa* L.) is caused by the gram-negative bacterium, *Pantoea ananatis* (Serrano) Margaret, formerly known as *Erwinia ananas*. The pathogen was first reported in the United States in Georgia on Vidalia onions (Gitaitis and Gay 1997) and since then it has been observed in other parts of the country including Colorado (Schwartz and Otto 2000), Michigan (Gent and Schwartz 2008), New York (Carr et al. 2010), and Pennsylvania (Pfeuffer et al. 2015). Under favorable conditions, growers can experience yield losses up to 100% (Gitaitis and Gay 1997; Nischwitz et al. 2007; Walcott et al. 2002). Symptoms of center rot in onion include water soaked lesions along the length of the leaf that gradually turn necrotic. Severe foliar infection can result in wilting of entire leaves (Gitaitis et al. 2002). Foliar infection can also lead to symptom expression in the bulb, which may open avenues for secondary microbes, ultimately predisposing bulbs for storage rot. Recently, researchers demonstrated the movement of *P. ananatis* from the point of inoculation in foliage to neck of the bulb and, eventually, into the corresponding scale (Carr et al. 2013).

In Georgia, short-day onion seeds are sown in seedbeds in September and typically transplanted in November to December and harvested during mid-April to early June (Boyhan et al. 2001). As the seeds germinate, radicle and flag leaves emerge within 20 days of sowing. True leaf emergence occurs within 30 to 35 days after sowing. Further, onion plants reach the 4- to 7-true-leaf stage within 70 days of sowing. First leaf senescence can be observed within 90 to 100 days of sowing. Bulb initiation is marked by the development of bladeless scales, beginning shortly after the first leaf senesces (Brewster 2008). After bulb initiation, photosynthate and water accumulate in the individual leaf bases resulting in bulb swelling (Rubatzky and Yamaguchi 1997). Leading up to harvest, the bulb

swells dramatically as more bladeless scales develop and additional leaves senesce (Brewster 2008). Maturity is characterized by the complete lodging of pseudo-stem and foliage (Jones and Mann 1963). The entire vegetative period of onion growth can therefore be divided into the following growth stages: sowing of the seed, 'loop' stage, 'whip' stage, cotyledon senescence, 'leek' stage, first leaf senescence, bulb initiation, bulb swelling, foliage 'fall-down,' and bulb ripening (Brewster 2008).

Identifying infection court and the growth periods when onion plants are susceptible to *P. ananatis* infection are critical in devising management strategies against this pathogen. In the center rot pathosystem, little is known about the growth stages when onions are most susceptible to *P. ananatis*. According to previous reports, only 11% of plants whose foliage were inoculated with *P. ananatis* resulted in onion bulb infection (Carr et al. 2013). These results suggest that bulb infection resulting from foliar inoculation may be inconsistent and perhaps is dependent on susceptibility of different onion growth stages, differential susceptibility of onion varieties to the pathogen, or interaction of both factors. The interaction of cultivar and growth stage on bulb incidence of center rot has never been investigated. Hence, the objectives were to (i) determine the susceptibility of onion growth stages to *P. ananatis*; and (ii) assess the interaction of bacterial inoculation at different growth stages with different onion cultivars on bulb incidence of center rot.

Materials and Methods

Bacterial strain and inoculum preparation. *P. ananatis* strain (PNA 97-1; accession no. DQ777968) was used in this study. This strain was previously isolated from the foliage of center rot-infected onion plants (Gitaitis and Gay 1997). For routine culture, 10 ml of nutrient broth was inoculated with a single colony of 48-h-old culture and shaken overnight on a rotary shaker (Inova; New Brunswick Scientific, Edison, NJ) at 150 rpm. After 12 h of incubation, 10 ml of bacterial suspension was centrifuged at $6,000 \times g$ (Allegra 25R, Beckman Coulter, Fullerton, CA) for 5 min. The supernatant was discarded and the pellet resuspended in 0.1M phosphate-buffered saline (PBS) and inoculum was adjusted using

[†]Corresponding author. E-mail: bhabesh@uga.edu

a spectrophotometer (Spectronic 20; Bausch and Lomb, Rochester, NY) to an optical density of 0.3 at 600 nm ($\sim 1 \times 10^8$ CFU/ml). Bacterial populations were confirmed by plating a 0.1 ml aliquot onto nutrient agar (NA).

Interaction of onion cultivar and growth stages on incidence of *P. ananatis* bulb infection. Two-week-old, healthy appearing seedlings of sweet onion cultivars 1407, 1518, Granex YPRR, Pirate, and Sweet Harvest (Vidalia Onion and Vegetable Research Center, Lyons, GA) were established in sterile pots (10 cm diameter $\times$ 8 cm height) containing a sterile potting mix (Scotts Miracle-Gro Company, Marysville, OH) under greenhouse conditions. These seedlings were grown from seedlots, which were tested for *P. ananatis* presence prior to planting using pathogen-specific PCR assay (Walcott et al. 2002). Seedlings were kept in a greenhouse (University of Georgia Tifton Vegetable Park; 31.4° N, 83.5° W) maintained at 25 to 28°C, 80 to 85% relative humidity, and ambient light conditions from December to May. Potted plants were kept on metal benches 1 m off the ground and watered every other day by hand, directed toward the base of the seedling. Seedlings ($n = 20$ /growth stage/cultivar/experiment) were inoculated at three different onion growth stages: first leaf senescence, bulb initiation, and bulb swelling. For inoculation, the newest fully expanded leaf of each onion seedling was cut using sterile pair of scissors approximately 3 cm from the tip. One milliliter of bacterial inoculum (PNA 97-1) containing 10^8 CFU/ml was pipetted into the interlamina cavity of the clipped leaf. Seedlings treated with 1 ml of 0.1M PBS buffer as above served as controls. Seedlings were arranged in a randomized complete block design in two independently conducted greenhouse experiments.

At harvest maturity, bulbs were harvested, stored temporarily in sterile 4.5-kg mesh onion bags (Harris Seeds, Rochester, NY), and carefully transported to the Natural Products Lab, Coastal Plain Research Station, University of Georgia, Tifton, GA. Onion bulbs were sorted for any visual symptoms prior to curing. Bulbs displaying softening of the neck were cut longitudinally and assessed for center rot incidence. Center rot incidence was calculated for bulbs from different cultivars, which were inoculated at three growth stages as the number of bulbs displaying symptoms at each growth stage divided by 20×100 . Bulbs that appeared healthy and did not possess softened necks were further cured at room temperature (25°C) for 7 days.

After 7 days of curing, bulbs were cut through the center using a sterile knife and assessed for center rot symptoms (discoloration of interior bulb scales). Onions displaying center rot symptoms were recorded as diseased and asymptomatic bulbs were recorded as healthy. Incidence of center rot for cured bulbs was calculated for each cultivar and growth-stage inoculation events as the number of bulbs displaying symptoms divided by total number of stored bulbs $\times 100$. Total disease incidence was calculated based on number of bulbs displaying center rot symptoms (number of bulbs with center rot prior to curing + number of bulbs with center rot after one week of curing) divided by 20×100 .

Bacterial identification. To confirm that symptoms observed were caused by *P. ananatis*, bacteria from symptomatic bulbs were isolated from a region adjoining the necrotic and healthy tissue and streaked onto Tryptic soy broth agar (TSBA) plates and incubated for 48 h at 28°C. After 48 h of incubation, yellow-pigmented colonies were isolated and tested for *P. ananatis* using physiological (Gram reaction, utilization of glucose in oxidative and fermentative reaction, indole reaction, and nitrate reductase reaction) and species-specific TaqMan based PCR assays (Dutta et al. 2014; Walcott et al. 2002). Briefly, presumptive *P. ananatis* colonies were picked using a sterile inoculation loop and suspended separately in 2-ml microcentrifuge tubes containing 25 μ l of sterile deionized water. The bacterial suspension was heated (Modular Dry Block Heaters, Cole Parmer, IL) for 3 min at 95°C. A suspension (5 μ l) was amplified in 20 μ l of PCR master-mix containing 10 mM Tris-HCl (pH 9.0), 50 mM KCl, 0.1% Triton X-100, 1.5 mM $MgCl_2$, and 0.2 mM of each nucleotide (dATP, dCTP, dGTP, and dTTP), 25 μ M each of primer PanITS1 (5'-GTCTGATAGAAAGATAAAGAC-3') and EC5 (5'-CGGTGGATGCCCTGGCA-3'), and 10 μ M of TaqMan probe

6-FAM TAGCGGTTAGGACTCCGCCCTTCA-BHQ (Dutta et al. 2014; Walcott et al. 2002). The PCR was conducted in a Cepheid Smart Cycler (Sunnyvale, CA) using the following thermal profile: denaturation at 95°C for 180 s, 35 cycles each of denaturation at 95°C for 15 s, and annealing at 60°C for 40 s. Samples with cycle threshold (Ct) values less than 35 were considered positive for *P. ananatis*.

Statistical analyses. Mean incidence of center rot for cultivar and growth stage inoculations for precured and cured onion bulbs were calculated using Proc Glimmix with Beta distribution for percentage response variable in SAS 9.4 (SAS Institute) at $P = 0.05$ level. The effects of cultivar and growth stage inoculations on total center rot incidence in onion bulbs were also analyzed as described above. The effect of interaction of onion cultivar and growth-stage inoculations was analyzed using Proc Glimmix in SAS 9.4 at $P = 0.05$ level.

Results

Effect of interaction of onion cultivar and growth stage inoculations on incidence of *P. ananatis* bulb infection. There were no significant interactions among inoculation growth stage and cultivar for precured, cured, and total bulb-center rot incidence (Tables 1-3). However, main effects of inoculation growth stage and cultivar significantly affected precured, cured, and total bulb-incidence of center rot ($P \leq 0.001$) (Tables 1-3). In addition, the effect of experiment was not significant ($P > 0.05$) and hence, the data from two experiments were pooled together and analyzed. In precured onion, significantly higher incidence of bulb rot was observed for Granex YPRR (69%) compared with other cultivars (Fig. 1A) ($P = 0.002$). The cultivar 1407 (13%) displayed significantly lower bulb incidence than Pirate (50%), Sweet Harvest (47%), and 1518 (36%) ($P = 0.032$). In cured onions, bulb incidence was significantly higher for Pirate (30%) and Sweet harvest (33%) compared with other cultivars ($P = 0.014$). Granex YPRR (16%) had numerically lower incidence of center rot; however, it was not significantly different from 1518 (21%) or 1407 (22%) (Fig. 1B) ($P = 0.242$). When total bulb-incidence of center rot was calculated and analyzed, Granex YPRR (84%) had significantly higher center rot incidence than other cultivars ($P = 0.012$). Center rot incidence was significantly lower for cultivars 1407 (33%) and 1518 (49%) and they were not significantly

Table 1. Summary of analysis of variance of incidence of *Pantoea ananatis* bulb infection (precured bulbs) at different onion growth stages and onion cultivar

Growth stages, variety	df ^a	F-value	P > F
Experiment ^b	1	3.67	0.632
Growth stages of onion ^c	2	23.32	0.028
Cultivar ^d	4	21.12	<0.001
Growth stages of onion $\times$ cultivar	8	45.52	0.421

^a Degrees of freedom.

^b Number of experiments.

^c Inoculation was done at first leaf senescence, bulb initiation, and bulb swelling growth stages.

^d Five Vidalia onion cultivars (1518, 1407, Granex YPRR, Pirate, and Sweet Harvest) were used.

Table 2. Summary of analysis of variance of incidence of *Pantoea ananatis* bulb infection (cured bulbs) at different onion growth stages and onion variety

Growth stages, variety	df ^a	F-value	P > F
Experiment ^b	1	2.37	0.421
Growth stages of onion ^c	2	20.2	0.014
Cultivar ^d	4	27.6	<0.001
Growth stages of onion $\times$ cultivar	8	38.2	0.218

^a Degrees of freedom.

^b Number of experiments.

^c Inoculation was done at first leaf senescence, bulb initiation, and bulb swelling growth stages.

^d Five Vidalia onion cultivars (1518, 1407, Granex YPRR, Pirate, and Sweet Harvest) were used.

different from each other (Fig. 1C) ($P = 0.414$). In contrast, Pirate (65%) and Sweet Harvest (64%) had significantly higher incidence of center rot compared with 1407 and 1518 (Fig. 1) ($P = 0.004$).

Since significant interactions between onion cultivar and the growth stage of *P. ananatis* inoculation and between two experiments were not observed, center-rot incidence data from each growth stage from two experiments were pooled together for precured, cured, and total disease incidence and analyzed ($P > 0.05$) (Tables 1-3). In precured onions, significantly higher bulb-incidence of center rot was observed when onion foliage were inoculated at first leaf senescence (62%) than at bulb initiation (37%) and bulb-swelling (31%) stages ($P = 0.041$) (Table 1, Fig. 2A). When remaining bulbs were cured for a week, significantly lower incidence of center rot were observed for bulbs, which were inoculated at the first leaf senescence (6%) stage compared with bulb initiation (29%) or bulb swelling (31%) stages ($P = 0.023$) (Table 2, Fig. 2B). When total bulb incidence was calculated and analyzed, significantly higher incidence of center rot was observed for bulbs whose foliage were inoculated during first leaf senescence stage (64%) compared with bulb initiation (55%) and bulb swelling (52%) stages ($P = 0.048$) (Table 3, Fig. 2C). Onion leaves inoculated with PBS did not display center rot bulb symptoms in two independent experiments.

Confirmation of infection by *P. ananatis*. The bacterial colonies isolated from infected tissue were gram-negative, utilized glucose as facultative anaerobe, positive for indole, and negative for nitrate reductase. Putative colonies were tested with primers specific for *P. ananatis* in a real-time PCR assay. One hundred percent of the colonies tested were positive for *P. ananatis*. No bacterial colonies were isolated from onion samples from negative control bulbs.

Discussion

The interaction among onion cultivars and their susceptibility to *P. ananatis* infection at different growth stages on bulb-incidence has not previously been investigated. In addition, the effect of these parameters on precured and cured (7 days at 25°C) bulb incidence of center rot has not been studied either. In this study, we investigated the effects of the above-mentioned variables on *P. ananatis* bulb incidence (precured and cured) under controlled greenhouse conditions.

The findings of this study suggest considerable variability in susceptibility to center rot incidence among sweet onion cultivars. The cultivar 1407 (13%) displayed significantly lower *P. ananatis* incidence prior to curing compared with all other cultivars, while Granex YPRR (69%) displayed the highest level of bulb incidence. In contrast for cured bulbs, we observed significantly lower levels of incidence with 1518 (21%), Granex YPRR (16%), and 1407 (22%) as compared with Sweet Harvest (33%) and Pirate (30%). A possible explanation for this observation could be reduced growth of *P. ananatis* during storage. Of the cultivars studied, 1407 (33%) and 1518 (49%) had significantly lower total bulb incidence of *P. ananatis*. Granex YPRR (74%) had significantly higher total bulb incidence than other tested cultivars. Similar observations of higher bulb incidence was observed earlier where Granex YPRR displayed highest level of center rot bulb incidence (80%) compared with other onion cultivars ($n = 42$) in a naturally infested field (Boyhan et al. 2014).

Table 3. Summary of analysis of variance of incidence of *Pantoea ananatis* bulb infection (total bulb incidence) at different onion growth stages and onion cultivar

Growth stages, variety	df ^a	F-value	P > F
Experiment ^b	1	3.17	0.248
Growth stages of onion ^c	2	24.2	0.037
Cultivar ^d	4	29.2	<0.001
Growth stages of onion × cultivar	8	48.3	0.923

^a Degrees of freedom.

^b Number of experiments.

^c Inoculation was done at first leaf senescence, bulb initiation, and bulb swelling growth stages.

^d Five Vidalia onion cultivars (1518, 1407, Granex YPRR, Pirate, and Sweet Harvest) were used.

Fig. 1. Percent incidence of center rot in precured and cured bulbs and total bulb incidence in sweet onion cultivars (1407, 1518, Granex YPRR, Pirate, and Sweet Harvest). Seedlings ($n = 20$ /cultivar/experiment) were inoculated with suspension containing *P. ananatis* (1×10^8 CFU/ml) at three different onion growth stages: first leaf senescence, bulb initiation, and bulb swelling, and incubated until harvest maturity under controlled greenhouse conditions. Disease assessments were made for precured and cured onion bulbs. Data points represent the mean bulb incidence of center rot in two independent experiments. Bars indicate the standard error of the mean. Means with similar lower case letters are not significantly different according to least significant difference (LSD) at $P = 0.05$ level.

Fig. 2. Percent incidence of center rot in precured and cured bulbs whose foliage were inoculated at three different onion growth stages (first leaf senescence, bulb initiation, and bulb swelling). Seedlings ($n = 20$ /cultivar/experiment) were inoculated with suspension containing *P. ananatis* (1×10^8 CFU/ml) at three different onion growth stages and incubated until harvest maturity under controlled greenhouse conditions. Disease assessments were made for precured and cured onion bulbs. Data points represent the mean bulb incidence of center rot in two independent experiments. Bars indicate the standard error of the mean. Means with similar lower case letters are not significantly different according to least significant difference (LSD) at $P = 0.05$ level.

We observed that inoculation of *P. ananatis* at different growth stages significantly affected bulb incidence in onion. When center rot incidence in onion bulbs was evaluated prior to curing, plants inoculated at first leaf senescence (62%) had significantly higher rates of center rot symptoms than plants inoculated at bulb initiation (37%) and bulb swelling (31%) stages. When center rot incidence in cured bulbs was evaluated, only 6% of bulbs inoculated at the first leaf senescence stage displayed center rot symptoms, while inoculation at bulb initiation (29%) and bulb swelling (31%) had significantly higher levels of *P. ananatis* incidence. The reason for lower bulb incidence for cured-bulbs inoculated at first leaf senescence stage is yet to be determined. It is possible that host defense is more pronounced during first leaf senescence stage than during later growth stages (bulb initiation and bulb swelling). Future studies could monitor the expression levels of defense genes at these growth stages following inoculation. Interestingly, inoculation at bulb initiation (52%) and bulb swelling (55%) stages did not result in significantly different levels of bulb incidence, whereas inoculation at first leaf senescence (64%) stage had significantly higher bulb infection. It is possible that longer incubation periods associated with early inoculation (first leaf senescence) might have resulted in higher bulb incidence than in later growth stage inoculations (bulb initiation and bulb swelling). In addition, we acknowledge that the data obtained in this study were based on one strain (PNA 97-1); future studies with multiple strains should help us to deduce if these observations are common among other pathogenic *P. ananatis* strains.

Overall, the interactions among onion cultivars and inoculation of *P. ananatis* at different growth stages on precured and cured onion bulbs were not significant. Rather, we observed that onion growth stage can influence incidence of *P. ananatis* in bulbs. Additionally, significant variability among sweet onion cultivars' susceptibility to bulb infection was also observed. These findings may provide information in devising management strategies against *P. ananatis* bulb incidence. Perhaps selecting less susceptible cultivars and concentrating protective chemical management (using bactericides) during the most susceptible onion growth stage/stages may help in reducing center rot incidence in onion bulbs. Further rigorous evaluation of more sweet onion cultivars and their susceptibility to *P. ananatis* at different growth stages should be conducted to develop integrated management strategies against this pathogen.

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