RESEARCH

Effects of *Verticillium dahliae* Infection on Stem-End Chip Defect Development in Potatoes

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ABSTRACT

Potato chips are the most popular snack food in the United States with annual retail sales of over US$6 billion. Stem-end chip defect, which is characterized by dark color of the vasculature and surrounding tissues at the tuber stem end portion of fried chips, is an important tuber quality concern for U.S. chip production. The cause of stem-end chip defect is not known. Verticillium wilt, caused by a vascular fungal pathogen *Verticillium dahliae* Kleb., is a persistent disease of potato (*Solanum tuberosum* L.) and other vegetables that causes early plant senescence and yield reductions. A 2-yr field trial was conducted to investigate the effects of *V. dahliae* on stem-end chip defect development and the activity of acid invertase at the apical (bud) and basal (stem) ends of tubers. Our results show that potato plants that were more infected with *V. dahliae* had a higher incidence of severe stem-end chip defects than plants with less *V. dahliae* infection. *Verticillium dahliae* infection of plants was correlated with an upregulation of acid invertase activity and an accumulation of reducing sugars on the stem end of tubers. Reducing sugars give rise to dark-colored defects as a result of pigments produced by the Maillard reaction during frying.

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Abbreviations: CFU, colony forming units; VW, Verticillium wilt.

Stem-end chip defect is a serious quality concern for the U.S. potato chip industry. Chips with this defect are unacceptably dark colored along the vasculature and adjacent tissues at a position that corresponds with the tuber stem end (Wang et al., 2012). Stem-end chip defects occur erratically between years and locations, with defects appearing locally in some years and regionally in others. Tubers that produce stem-end defect chips are undesirable to consumers, increase financial risks to chip growers, and can cause supply problems and quality concerns for processors. Defect chips contribute disproportionately to chip acrylamide content, and acrylamide is an important food safety and human health concern (Vinci et al., 2011).

Previous data have shown that stem-end chip defects are associated with high reducing sugar contents and elevated activities of acid invertase on the tuber stem end but not the tuber bud end (Wang et al., 2012). Invertase hydrolyzes sucrose into the reducing sugars glucose and fructose, and these react with free amino acids to produce dark-colored products in the Maillard reaction when tuber slices are fried in hot oil (Shallenberger et al., 1959). Thus, elevated reducing sugars are the immediate cause of dark color in stem end tissues.

The causes of stem-end chip defect have not been identified. It has been suggested that stem-end chip defects result from abiotic

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stresses, biotic stresses, or a combination of the two. The abiotic stresses may include a period of heat stress during plant growth (Wang et al., 2012). Biotic stresses may include pathogens, with pathogens such as *Verticillium dahliae* that infect the vasculature of potato plants being attractive candidates for detailed investigation.

Verticillium dahliae is a soil-borne vascular fungal pathogen that can lead to yield losses of 10 to 50% and decreases in crop quality in potatoes (Powelson and Rowe, 1993) and other crops. The disease caused by *V. dahliae* is variously called Verticillium wilt (VW), potato early dying, or vascular wilt (Fradin et al., 2009). Verticillium wilt is a persistent and serious problem in potato production (Jansky, 2009) that is distributed throughout potato growing regions of the world (Powelson and Rowe, 1993). Two species of *Verticillium* can cause VW, *V. dahliae* as well as *V. albo-atrum* Reinke and Berthier. *Verticillium dahliae* is widespread in the United States, especially in production areas where average daily summer temperatures commonly exceed 25°C (Johnson and Dung, 2010).

The typical visual disease symptoms of VW, acropetal progression of chlorosis and necrosis of leaves followed by premature defoliation, may be indistinguishable from natural senescence although these symptoms sometimes occur on only one side of the plant or of individual leaves (Isaac and Harrison, 1968; Powelson and Rowe, 1993). Vascular browning is often prominent in the bases of affected stems, and necrosis of the vascular ring develops in potato tubers of some cultivars (Powelson and Rowe, 1993). Advanced symptoms, such as loss of plant vigor during mid to late summer followed by senescence and death of the crop a few weeks before normal maturity (Rowe, 1985), usually do not appear until the late tuber-bulking stage (Powelson and Rowe, 1993). The VW disease is typically controlled through the use of fumigants, but concerns about the economic and environmental costs of fumigation bring its sustainability into question (Rowe and Powelson, 2002).

A 2-yr research project was conducted at the Univ. of Wisconsin-Madison Hancock Agricultural Research Station in 2010 and 2011 to test the hypothesis that *V. dahliae* contributes to the formation and development of stem-end chip defect in chipping potato cultivars.

MATERIALS AND METHODS

2010 and 2011 Field Season at Hancock

Research was conducted in nonfumigated fields and in fields fumigated the previous fall. The nonfumigated, *V. dahliae*-infested field was fumigated in 2004 and then inoculated with *V. dahliae* in 2005. It contained approximately 9 and 12 colony forming units (CFU) of *V. dahliae* per gram of soil in 2010 and 2011, respectively. This field is planted with potatoes each year and is used to screen clones from potato breeders for resistance to VW. A second field 50 m away was fumigated with metam sodium (sodium methylaminomethanedithioate) (Vapam HL; AMVAC Chemical Corporation) at a rate of 190 kg ha^{-1} applied by shank injection the previous fall to reduce disease pressure. It contained approximately 0 and 1 CFU of *V. dahliae* per gram of soil in 2010 and 2011, respectively. Three chipping varieties grown commercially in central Wisconsin, FL1879, FL2053, and Snowden, were planted by hand on 4 May 2010 and 2 May 2011 in the two trial fields with three replicates in each field. Each replicate was a single block in 2010 with 10 seed tubers planted 0.6 m apart whereas in 2011 each replicate was two blocks containing five seed tubers planted 0.3 m apart. Blocks in both years were randomized across the field. Potato plots were maintained using best management practices based on University of Wisconsin-Extension recommendations.

Visual Ratings

Field plots were scored for VW based on the percent chlorotic, necrotic, or wilted foliage in each variety with 30 hills on 26 July, 12 Aug., and 20 Aug. 2010 and on 27 July, 16 Aug., and 29 Aug. 2011.

Field Samplings

The first two harvests occurred on 28 July (early season harvest) and 16 Aug. (late season harvest) 2010 and 28 July (early season harvest) and 17 Aug. (late season harvest) 2011. Diquat dibromide (6,7-dihydrodipyrido[1,2-a:2',1'-c]pyrazinediium dibromide) (Reglone; Syngenta) was applied at a rate of 1.8 kg ha^{-1} 10 d before final harvest in 2010 and 14 d before final harvest in 2011 to desiccate vines that remained green. A third harvest (final harvest) was conducted when all plants had completely senesced, on 30 Aug. 2010 and 13 Sept. 2011. During each harvest, tubers from each variety were identified by hill, harvested by hand, and placed into a mesh bag. At the early season and late season harvests, three tubers randomly selected from each mesh bag were processed into chips and scored 1 d later. At the final harvest, three tubers from each harvested hill were sampled and processed as described above, and the other tubers were put into storage at 13°C for 2 mo, after which a fourth sampling occurred. Three tubers from each hill were processed for chips at this time. At the first two harvests, main stems from each hill were collected and bulked for stem sap assay to estimate severity of infection based on the abundance of conidia. At the final harvest, main stems that were wilted and dried were cut and bulked from each hill for dry stem assay to estimate severity of infection based on the abundance of microsclerotia.

Verticillium Culturing Assays

Stem Sap Assay

A 10-cm section of each main stem was cut 8 to 10 cm above the soil surface. The bulked stems were rinsed in tap water, surface disinfested in 1% bleach for 1 min, rinsed in sterile distilled water, and then wrapped in a 20 by 20 cm plastic bag. Each bulked stem sample was squeezed with a press to extract sap and a 100-μL aliquot was plated onto Sorensen's NP-10 medium (Butterfield and DeVay, 1977). Fungal colonies were counted using a stereomicroscope 14 d later after growth at ambient temperature without light.

Dry Stem Assay

For each bulked dry or partially dry stem sample, the apical 10 cm portion was placed in a paper bag and allowed to air

dry for 2 mo. Leaves were removed from the dried stem apices and stems were ground in a Wiley mill with a 40-mesh screen. Per sample, 50 mg was plated on Sorensen's NP-10 medium. Fungal colonies were allowed to grow for 14 d at ambient temperature without light. Plant debris was washed from the plate by gently rubbing the surface of the agar medium with fingers under a stream of tap water. Fungal colonies on each plate were counted using a stereomicroscope.

Soil Assay

Soil samples were taken from each replicate in the two fields on 2 Aug. 2010 and 20 Aug. 2011. Samples were left at room temperature for 4 mo to dry completely and were pounded thoroughly to remove clumps. Three 10-g subsamples were weighed from each soil sample and each was placed into a 250 mL flask along with 100 mL of distilled water. Samples were vigorously stirred with a magnetic stirrer for at least 10 s. One milliliter of the supernatant of each soil solution was pipetted onto a plate with Sorensen's NP-10 medium and spread evenly over the media surface with a glass rod. Fungal colonies were counted using a stereomicroscope 14 d later after growth at ambient temperature without light.

Lab Samplings

Tissue samples for sugar determinations and acid invertase assays were conducted as described in Bethke et al. (2009) with tissue cores (0.7 mm diameter and 0.5–0.8 g fresh weight) removed at each sampling time from positions as close as possible to the vasculature at the apical (bud) and basal (stem) ends of tubers.

Biochemical Analysis of Tuber Tissue Samples

High performance liquid chromatography determination of tuber sugar contents were performed according to published protocols in Bethke et al. (2009). FL1879 was selected for invertase assays because all stem-end chip defect scores were sufficiently well represented for that variety. A total of 30 bud end and 30 stem end tissue samples from cultivar FL1879 were used for invertase activity assay. During the assay, frozen tissue samples of 0.50 to 0.75 g were ground in a freezer mill (SPEX SamplePrep) and stored at −80°C until extracted for proteins. Powdered tissue samples were mixed with 2 mL extraction buffer and 4 to 5 glass beads. The extraction buffer contains 50 mM hydroxyethylpiperazineethanesulfonic acid KOH, pH 7.5, 5 mM $MgC_{12}{\cdot}6H_2O$, 1 mM ethylenediaminetetraacetic acid, 1 mM ethylene glycol tetraacetic acid, 0.1% w/v Triton X-100, 10% w/v glycerol, 5 mM dithiothreitol, and 1 mM phenylmethanesulfonylfluoride. Homogenized extracts were incubated on ice and shaken every few minutes by hand. Homogenates were centrifuged 10 min at 4°C at 16,000 × *g* in a tabletop microfuge to pellet debris. Supernatant of 1 mL were desalted using PD MidiTrap G25 columns following the manufacture's directions for gravity flow (GE Healthcare). About 250 μL of desalted extract were placed into 0.2 mL strip-tubes and shaken using a Vortex Genie-2 (Scientific Industries) at speed 6 for 30 min at 4°C to minimize the activity of invertase inhibitor proteins. Enzyme assays contained 20 μL protein extract and 60 μL of reaction buffer containing 133 mM sucrose and 26.7 mM CH_3COONa pH 4.7 and were incubated at 30°C. After 60 min 8 μL sodium phosphate (1 M, pH 7.4) was added to stop the reaction and tubes were heated at 97°C for 3 min to inactivate the enzyme. Control samples contained all additions but were neutralized and heated immediately. Net glucose formed was quantified using the assay described in Bethke and Busse (2008). Protein content of desalted extracts was measured using the Bio-Rad protein quantification reagent (Bio-Rad Laboratories) following the manufacture's directions for the micro assay. Invertase activity was calculated as nanomoles glucose formed per minute per milligram of proteins.

Chip Frying and Scoring

Longitudinal slices approximately 1 mm thick were cut through the stem end attachment point of each tuber and were fried and rated for stem-end chip defect on a scale of 0 to 5 according to the index in Wang et al. (2012). In this system, no color development on the chip was a 0, dark color of vascular tissue only was a 1, color shadow surrounding the vascular tissue was a 2, color penetrating up to 1 cm into the tissue was a 3, color development up to 1.3 cm into the tuber tissue was a 4, and color development beyond 1.3 cm was a 5. Since chips with sores of 3, 4, and 5 may be scored as defects at processing facilities, those three scores were categorized as severe defects. Chips with scores of 0, 1, and 2 are likely to meet processor requirements for defect free.

Data Analysis

Defect score data were analyzed to test pathogen infection effects at each of the four sampling time points by using the Logit Model procedure of R (R Development Core Team, 2008) based on its binomial distribution. Differences in percentage of severe defects under each treatment for each variety were compared using chi-square test. Pearson's correlation was used to analyze relationships between defect score and sugar contents as well as relationships between defect score and stem CFU. Analysis of variance was performed to test pathogen and storage treatment effects on invertase activities by using the linear model procedure of R.

RESULTS

Plants in the Nonfumigated Field Had Higher Rates of *Verticillium dahliae* Infection than those in the Fumigated Field

At the time of the early or late season harvests in both 2010 and 2011, sap from freshly cut primary stems had higher CFU from the nonfumigated field than from the fumigated field across all varieties (Fig. 1). All three varieties had greater CFU counts in dry stems from the nonfumigated relative to the fumigated field at final harvest (Fig. 1). The CFU in stems from both treatment fields in 2011 at the late season harvest were significantly greater than in 2010 (Fig. 1; $p < 0.05$).

Increased *Verticillium dahliae* Pressure Was Associated with Increased Incidence of Severe Stem-End Chip Defects

In field year 2010, none of the plants in the fumigated field showed symptoms of VW. In comparison, plants of the three varieties in the nonfumigated field showed a similar, progressive development of VW disease symptoms from 10 to 20%

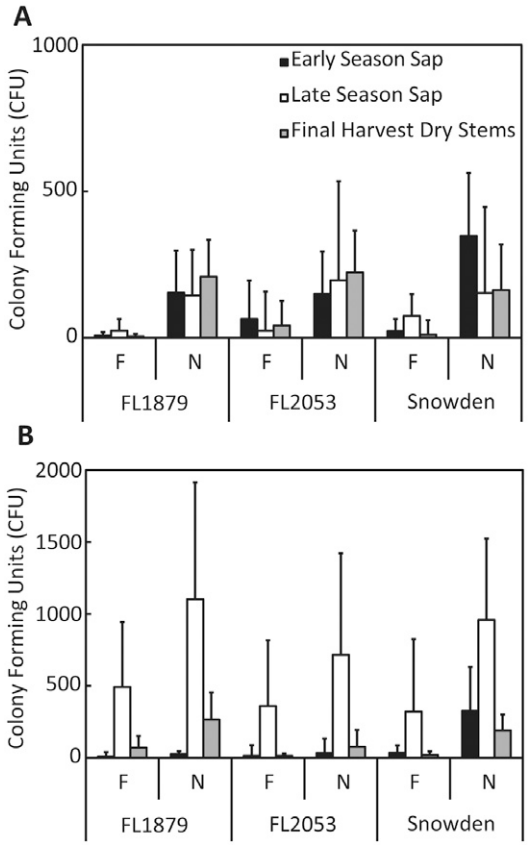

Figure 1. Mean colony forming units (CFU) in stems of potato plants grown in fumigated (F) or nonfumigated (N) soil at early season (black bars), late season (white bars), and final harvest (gray bars) for cultivars FL1879, FL2053, and Snowden in 2010 (A) and 2011 (B). Error bars are standard deviations of 10 plants.

in the early season to 85 to 90% at final harvest (Table 1). In field year 2011, the plants in the fumigated field showed signs of VW disease pressure from late season to final harvest, suggesting that soil fumigation was less effective than in the previous year. Cultivar FL1879 displayed the highest percentage of plants showing VW disease symptoms in the 2011 fumigated field, 30% at the late season harvest and 90% at final harvest, whereas cultivars FL2053 and Snowden had lower percentages of plants with VW symptoms (Table 1). The nonfumigated field had plants with more severe VW symptoms in 2011 compared to 2010 (data not shown), but approximately the same percentage showed VW symptoms at each harvest in 2010 and 2011 in all the three varieties (Table 1).

Few or no severe stem-end chip defects were found at the early season sampling in either the fumigated or nonfumigated field in both years (Table 2). In the late season, however, stem-end chip defects were observed in each of the three varieties examined, with FL1879 having a severe defect percentage of 16% in 2010 and 47% in 2011 when grown under conditions of high infection pressures compared with only 2 and 3% when grown with less infection pressure in 2010 and 2011, respectively ($p < 0.05$). At the late season harvest in both years, FL2053 and Snowden did not display significantly higher severe stem-end chip defect percentages when grown under nonfumigated rather than fumigated conditions.

At final harvest, FL1879 had 36 and 33% more stem-end chip defects in tubers from the nonfumigated field than in those from fumigated field in 2010 and 2011, respectively ($p < 0.05$). Snowden at this time also displayed significantly higher severe stem-end chip defect incidence in the field with higher VW pressures in both years (Table 2). Tubers from Snowden had severe defect incidence as high as 40% in the nonfumigated field in 2011 (Table 2). Cultivar FL2053 had greater incidence of stem-end chip defects in the nonfumigated field compared to the fumigated field in 2011.

After 2 mo of storage at 13°C, tubers from plants exposed to greater VW pressure produced chips with more severe stem-end defects than control tubers, and this difference was statistically significant at $p = 0.05$ for Snowden in 2010 and FL1879 and FL2053 in 2011. The out of storage severe defect incidence from both fumigated and nonfumigated treatments were significantly higher in 2011 than in 2010 (Table 2). Severe stem-end chip defect percentage out of storage increased in 2011 and decreased in 2010 across all cultivars and fumigation treatments (Table 2). No significant relationship was observed between stem-end chip defect scores across the three sampled tubers and overall CFU counts of the bulked primary stems from each hill (data not shown).

More Severe Stem-End Chip Defect Scores Were Associated with Elevated Stem-End Glucose Contents

Under both fumigation treatments at harvest and out of storage, stem-end chip defect scores were correlated with tuber stem-end glucose contents in all three varieties. Correlation

Table 1. Percentage of 10 hills in cultivars FL1879, FL2053, and Snowden that showed Verticillium wilt symptoms in fumigated (F) and nonfumigated (N) fields at each harvest time point during the growing season in 2010 and 2011.

		2010			2011		
Variety	Treatment	Early season	Late season	Final harvest	Early season	Late season	Final harvest
FL1879	F	0	0	0	0	30	90
	N	20	75	90	25	70	80
FL2053	F	0	0	0	0	25	30
	N	15	80	85	5	55	100
Snowden	F	0	0	0	0	10	15
	N	10	70	85	15	40	60

Table 2. Percentage of chips with severe stem-end chip defects in each variety under growth in fumigated (F) or nonfumigated (N) fields at four different sampling time points in 2010 and 2011.

		2010				2011			
Variety	Treatment	Early season	Late season	Final harvest	Out of storage	Early season	Late season	Final harvest	Out of storage
FL1879	F	0a†	2b	25b	17a	0a	3b	20b	53b
	N	6a	16a	61a	20a	2a	47a	53a	70a
FL2053	F	0a	0a	22a	10a	0a	2a	14b	43b
	N	2a	2a	27a	13a	2a	3a	21a	83a
Snowden	F	0a	0a	13a	3b	0a	3a	13b	43a
	N	3a	4a	22b	17a	0a	10a	40a	47a

†At each time point for each variety, values followed by the same letter did not differ significantly between fumigation treatments using chi-square test at $p = 0.05$.

coefficients were 0.37, 0.45, and 0.39 for FL1879, FL2053, and Snowden, respectively (Table 3). Correlation coefficients between severity of stem-end chip defect and stem-end sucrose contents as well as the ratio of stem-end glucose to sucrose were small and ranged from 0.01 to 0.15. Tuber sucrose, glucose, or the ratio of glucose to sucrose on the bud end of tubers also did not correlate with defect score (Table 3). Bud- and stem-end glucose and sucrose data are presented in Supplemental Tables S1 and S2. Statistically supported relationships between these data and stem-end chip defect incidence or severity were not observed.

Table 3. Correlation coefficients from Pearson's correlation analysis of relationship between stem-end chip defect score and sucrose, glucose, and the ratio of glucose to sucrose on the bud end and stem end of tubers in potato varieties FL1879, FL2053, and Snowden. Data are compiled from all harvests and both years.

Variety	Location	Sucrose	Glucose	Ratio of glucose to sucrose
FL1879	Bud end	0.12	0.07	0.06
	Stem end	0.11	0.37**	0.13
FL2053	Bud end	0.16	0.08	0.04
	Stem end	0.05	0.45***	0.15*
Snowden	Bud end	0.11	0.16	0.08
	Stem end	0.07	0.39***	0.01

*Significant at the 0.05 probability level.

**Significant at the 0.01 probability level.

***Significant at the 0.001 probability level.

Tubers with High Stem-End Chip Defect Scores Had High Rates of Stem-End Acid Invertase Activity

Acid invertase activities in the stem end of FL1879 tubers from fumigated fields were higher in tubers that produced chips with defect scores of 4 and 5 than in tubers with defect scores of 0 to 3 (Fig. 2). For tubers from nonfumigated fields, higher acid invertase activities were observed in the stem end of tubers that had defect scores of 3, 4, and 5 relative to tubers with scores of 0, 1, and 2 (Fig. 2). Invertase activities at the tuber stem end were higher out of storage than at harvest under both fumigation treatments for defect scores of 3, 4, and 5 (Fig. 2; $p < 0.05$). Invertase activities in tubers that produced chips with a defect score 0 were lower than those in tubers with other defect scores when averaged across treatments (Fig. 2; $p < 0.05$). For chips with severe stem-end chip defects, average stem-end invertase activities in tubers at harvest and out of storage were greater in tubers from fields with greater VW pressure than in fields with less VW pressure (Fig. 2; $p < 0.05$). A relationship between bud end acid invertase activity and stem-end chip defect score was not observed (data not shown).

DISCUSSION

Severe stem-end chip defects were observed in tubers harvested from fumigated and nonfumigated fields for each of the three varieties examined, but incidence in the fumigated field was lower than that in the nonfumigated field (Table 2). The growing seasons in 2010 and 2011 were both characterized by periods of hot weather that may have contributed to stem-end chip defects in many varieties grown in central Wisconsin. Previous data showed that heat stress might increase stem-end chip defect severity (Wang et al., 2012). Therefore, environmental stress and pathogen stress from *V. dahliae* may have both contributed to the development of stem-end chip defects. Warm temperatures favor growth of *V. dahliae* in the xylem while at the same time increasing the need for transpiration by the plant. It is possible that *V. dahliae* infection exacerbates the influence of high temperature stress by restricting transpiration and reducing evaporative cooling, thereby increasing the severity of the defect in chips (Table 2). Fumigation is relatively indiscriminate with regard to the organisms that it kills, however, and organisms that co-infect plants along with *V. dahliae* remain candidates for making a contribution to stem-end chip defect.

Vascular ring discoloration, where vascular tissues are locally yellow or brown, may be observed in raw potato tubers as a result of VW or Fusarium wilt (Fiers et al., 2012). A general observation was that tubers that showed discoloration before frying were more likely to produce chips with stem-end defects than those that did not show discoloration.

Colony forming units from early season fresh stem sap to final harvest dry stems were always higher in plants grown in the nonfumigated field than in plants from the fumigated field (Fig. 1). Johnson and Dung (2010) stated that

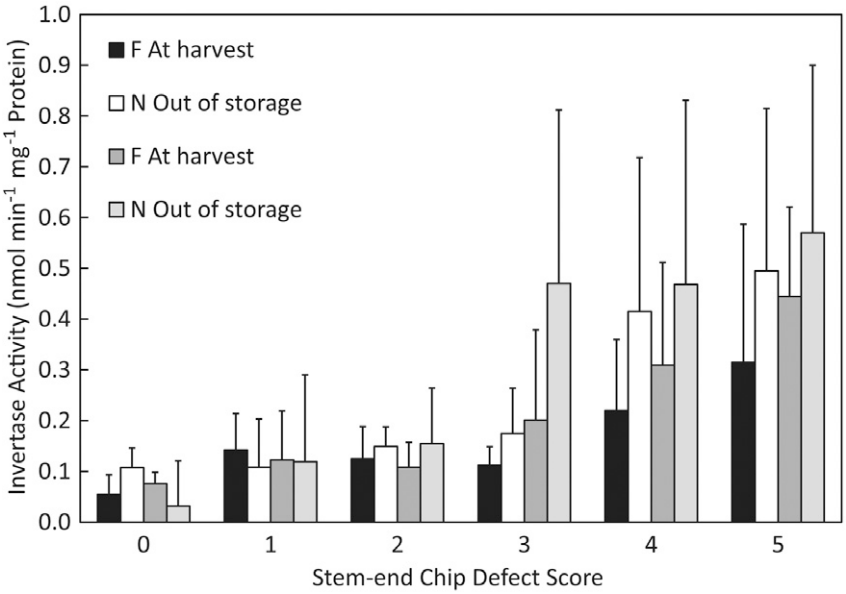

Figure 2. Invertase activities on the stem end of tubers of cultivar FL1879 from fumigated (F) (black and white bars) and nonfumigated (N) fields (dark gray and light gray bars) at harvest (black and dark gray bars) and out of storage (white and light gray bars). Error bars are standard deviations of tubers that produced chips with the respective stem-end chip defect score.

although infection may occur early in the growing season, VW symptoms generally do not develop until the later part of the growing season when rapid tuber bulking occurs and the fungus has spread and colonized the whole plant. This is consistent with the VW data from 2010 but not the data from 2011 when VW disease pressure was greater and VW symptoms were observed at the early season harvest. The dramatic increase in sap CFU at the late season sampling in 2011 suggests that there was more extensive growth of the fungus in plants in 2011 relative to 2010. It is interesting to note that more stem-end chip defects were observed in 2011 than in 2010 and that the percentage of chips with severe defects increased during storage in 2011 and decreased in storage in 2010. These observations may reflect the differences in severity of *V. dahliae* infection between the 2 yr although additional data are needed to confirm this.

High acid invertase activities at the tuber stem end were correlated with high stem-end chip defect scores (Fig. 2). Within the severe defect scores of 3, 4, and 5, stem-end invertase activity was greater in tubers from the nonfumigated field relative to the fumigated field, both at harvest and out of storage. This finding confirms and extends the observation that high rates of acid invertase activity were associated with more severe stem-end chip defects (Wang et al., 2012). Heat or water stress during the growing season (Thompson et al., 2008) and low temperatures during storage (Sowokinos, 2001) have also been shown to cause an increase in acid invertase activity at the tuber stem end. We are not aware of reports demonstrating an upregulation of this enzyme in potato tubers by pathogen stress although an upregulation of cell wall acid invertase activity following pathogen infection of leaves has been reported for several species (Berger et al., 2007; Bolton, 2009). The data presented here, however, suggest that infection of plants by *V. dahliae* or other soil-borne pathogens contributes to an upregulation of acid invertase activity on the stem end of potato tubers.

In summary, it is suggested that high rates of infection of potato plants with *V. dahliae* during the growing season are associated with an upregulation of acid invertase activity on the stem end of potato tubers. Higher invertase activities cause an accumulation of reducing sugars, which are the primary cause of dark pigment formation in stem-end defect chips.

Supplemental Information Available

Supplemental material is included with this manuscript.

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