Localized Accumulation of Condensed Tannins Associated with Poinsettia Bract Necrosis

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ABSTRACT. The early onset of bract necrosis in poinsettia (*Euphorbia pulcherrima* Willd. ex. Klotzch) is characterized by small dark-stained spots that precede the development of enlarged necrotic lesions. Electron micrographs of adaxial epidermal and subepidermal tissues with early symptoms of necrosis revealed large, electron-dense deposits in cell vacuoles. These spherical bodies resembled condensed tannins observed in the epidermal tissues of peach and apple fruit. Chemical analysis of bract tissues confirmed the presence of condensed tannins. Furthermore, there were higher concentrations of condensed tannin in bract samples with 2-mm-diameter lesions than in samples with lesions <0.5 mm (equivalent to catechin concentrations of 59 and 13 mg·g⁻¹ fresh mass, respectively). No tannin bodies were observed in parallel samples of healthy-appearing bracts in which only trace concentrations of condensed tannins were measured (0.2 mg·g⁻¹ fresh mass). The evidence suggests an association between condensed tannin accumulation in localized areas of the bract and the early appearance of bract necrosis symptoms.

Bract necrosis can cause extensive damage on the poinsettia crop at the end of the production cycle. Necrosis begins as small dark-stained areas near the looped ends of lateral veins in the margins of the mature bracts on susceptible cultivars like 'V-17 Angelika White', and 'Supjibi Red' (McAvoy and Bible, 1996a). As plants reach initial anthesis, symptoms begin as a few isolated lesions near the vascular tissues of individual bracts. As the disorder progresses in the weeks following anthesis, lesions begin to appear on previously healthy bracts and become larger and more numerous on necrotic bracts. While necrotic lesions may ultimately exceed 5 mm in diameter, they first become visible to the eye when they exceed 0.5 mm in diameter and under magnification can be observed at an even earlier stage of development.

Susceptibility to bract necrosis has been related to genotype (McAvoy and Bible, 1997; Wissemeier, 1993) and nutritional factors (Nell and Barrett, 1985, 1986; McAvoy and Bible, 1996a). Woltz and Harbaugh (1986) reported that topical sprays of calcium (Ca) reduced the incidence of bract necrosis; and McAvoy and Bible, (1996a, 1996b) reported a similar effect with both silica and strontium sprays. However, susceptibility to bract necrosis is not related to bract margin Ca concentration (McAvoy and Bible, 1997; Wissemeier, 1993), nor is there evidence of potentially phytotoxic levels of mineral nutrients in bract margins (McAvoy and Bible, 1996a). Complicating the situation further is the observation that a single spray of benzyladenine (McAvoy and Bible, 1998) effectively suppresses the development of bract necrosis.

Struckmeyer (1960) conducted histological examinations of poinsettia leaves from plants grown under nutritional deficiencies, but did not examine bracts. Nell and Barrett (1986) conducted

studies on the necrosis-susceptible cultivar 'V-14 Glory', but only examined the gross anatomical structure of transverse sections of healthy leaves and transition bracts. Thus, while the general symptoms of bract necrosis have been described, the detailed anatomical aspects have not been reported. This report attempts to describe the cellular events associated with the early onset of bract necrosis based on histological examination.

Materials and Methods

PLANT PRODUCTION. Rooted cuttings of poinsettias (*Euphorbia pulcherrima* Willd. ex. Klotzch) 'V-17 Angelika White' in 1995, and 'Supjibi Red' and 'Red Success' in 1996 were transplanted into 0.5-L pots containing Metro Mix 510 medium (O.M. Scott, Marysville, Ohio), at the University of Connecticut greenhouses (Storrs, Conn.). They were initially irrigated with 14.3 mol·m⁻³ N (200 mg·L⁻¹) from a 5N-6.9P-14.1K soluble fertilizer, and thereafter at each irrigation with 14.3 mol·m⁻³ N from a 15N-2.2P-20.8K soluble fertilizer. In 1995, plants were pinched to five nodes on 15 Sept. and photoperiodic lighting (2 μmol·m⁻²·s⁻¹) was provided from 2200 to 0200 HR nightly between 15 Sept. and 12 Oct. In 1996 plants were pinched on 23 Sept. leaving four to five nodes per plant. Photoperiodic lighting (2 μmol·m⁻²·s⁻¹) was provided from 2200 to 0200 HR nightly between 30 Aug. and 25 Oct.

TISSUE SAMPLING AND FIXATION FOR EXAMINATION WITH LIGHT MICROSCOPY, 1995. Bracts on 20 'V-17 Angelika White' plants were visually examined at weekly intervals between 12 Dec. 1995 and 23 Jan. 1996. Once symptoms of bract necrosis began to appear, 1-cm² tissue samples were excised from the looped end of the lateral veins near the bract margins—the localized portion of the bract where symptoms typically begin (McAvoy and Bible, 1996a). At each sampling date, five samples with necrotic lesions and five healthy-appearing samples were randomly collected. Samples were immediately fixed in formalin propionic acid solution (FPA) and shipped to Iowa State University.

The FPA was triple rinsed from the tissue using 70% ethanol and the tissue was dehydrated using a tertiary butyl alcohol (TBA) sequence progressing from 20% to absolute TBA at 24-h intervals (Jensen, 1962). After dehydration, samples were embedded in

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Fig. 1. Light micrographs of the transverse cross-section of 'V-17 Angelika White' bracts. (A) Sample obtained from healthy-appearing portion of a bract near the lateral vein where bract necrosis symptoms typically occur. (B) Section of a bract near a developing necrotic spot. Note dark occlusions (arrow) beginning to form in the adaxial epidermal cells. (C) Dark occlusions are seen filling the entire epidermal cell as bract necrosis lesions become larger. (D) Dark occlusions fill the both the adaxial and abaxial epidermal cells and some of the subepidermal cells of a bract sample displaying advanced symptoms of bract necrosis. Scale bar is equal to a length of 50 μ m in A, B, and C and 125 μ m in D.

paraffin, mounted and allowed to cool at room temperature. Five micrometer transverse sections were cut through the area surrounding the curvature of the vascular tissue at the bract margin using a rotary microtome (Model 820, American Optical, Buffalo, N.Y.). Sections were mounted on slides, stained with safranin and fast green and examined using light microscopy.

TISSUE SAMPLING AND FIXATION FOR EXAMINATION WITH ELECTRON MICROSCOPY, 1996. Bract tissues for examination by electron microscopy were obtained from susceptible 'Supjibi Red' poinsettias sprayed with 4 mM Na_2SiO_3 (sodium silicate), or 4 mM SrCl_2 (strontium chloride) or deionized water. Sprays were applied to eight plants in each treatment group at weekly intervals beginning 27 Nov. 1996 (appearance of fully colored transition bracts) and ending 24 Dec., 1 week before initial anthesis.

On 2 Feb. 1997, a 40 $\times$ dissecting microscope was used to examine bract samples for early symptoms of necrotic lesions and 1-mm² bract margin samples were excised from the looped end of the lateral veins. Lesions on bract samples appeared as small areas of dark tissue about 0.1 mm in diameter. Samples of healthy-appearing tissue were collected from the same bracts as the necrotic samples but dark lesions were not evident on these

samples when viewed under the dissecting microscope. From deionized water sprayed plants both healthy-appearing samples and samples with dark stained lesions were collected, while only healthy appearing samples were collected from SrCl_2 and Na_2SiO_3 sprayed plants.

Excised tissues were placed immediately in a solution containing 1.5% paraformaldehyde and 1.5% glutaraldehyde in 0.1 M sodium cacodylate buffer (pH 7.2) and fixed at 20 $^\circ\text{C}$ over night with gentle agitation using a 45 $^\circ$ rotating platform (Glauert, 1975). The tissues were then washed in the buffer three times for 1 h with each change. Tissues were postfixed in 1% osmium tetroxide in 0.1 M sodium cacodylate buffer for 2 h with further gentle agitation and washed in double distilled water three times, for 30 min each change.

The fixed tissues were dehydrated in a graded ethanol series in 30-min steps. There were single changes with ethanol percentages of 12%, 25%, 50%, 70%, 95%, and two changes with 100%. Tissues were passed through two 30-min changes in 100% propylene oxide.

The dehydrated tissues were infiltrated stepwise as follows: a mixture of 2 propylene oxide : 1 resin (Epon 812-Araldite 6005, Electron Microscopy Sciences, Fort Washington, Pa.) for 6 h at 20 $^\circ\text{C}$; a mixture of 1 propylene oxide : 1 resin with gentle

agitation overnight at 20 $^\circ\text{C}$; a mixture of 1 propylene oxide : 2 resin with the addition of 1.5% 2,4,6-tri(dimethylaminomethyl) phenol (DMP-30) overnight at 20 $^\circ\text{C}$; and finally a 100% resin plus 1.5% DMP-30 at 20 $^\circ\text{C}$ for 6 h with gentle agitation. Tissues were embedded in fresh resin plus DMP-30 using rubber embedding molds and polymerized for 24 h at 60 $^\circ\text{C}$.

TRANSMISSION ELECTRON MICROSCOPY. Thin sections were cut from the portion of the embedded bract samples where necrotic symptoms typically occur using an ultramicrotome equipped with a diamond knife. Sections were mounted on copper grids (200 mesh), stained with lead citrate, and examined under a transmission electron microscope (Philips EM 300, Eindhoven, The Netherlands) at the University of Connecticut Electron Microscope Laboratory.

SCANNING ELECTRON MICROSCOPY. Tissue sample preparation for scanning electron microscopic examination were as previously described through to the graded ethanol dehydration step. Tissues were then critical-point dried with CO_2 and sputter coated with gold/palladium for 20 s. Tissues were examined under a field emission scanning electron microscope (DSM 982 Gemini, LEO Electron Microscopy Co, Oberkochen, Germany) at the University of Connecticut Electron Microscope Laboratory at a 2-kV acceleration voltage.

CONDENSED TANNIN ANALYSIS. A 6 mm diameter circular punch was used to excise tissue disks from randomly selected bracts of untreated poinsettia plants. Disk samples from the necrosis susceptible 'Supjibi Red' and the resistant 'Red Success' were immediately weighed and stored at -80 $^\circ\text{C}$ until analysis. Bract samples from 'Supjibi Red' included 1) five sets of five disks that contained $\approx$ 2 mm diameter dark-stained spots that were visible without

magnification, 2) five sets of five disks that contained <0.5 mm diameter dark-stained spots that were visible under a 20× magnifying loop, or 3) five sets of five disks were excised from the healthy-appearing looped-ends of lateral veins that were adjacent to necrotic lesions. An additional five sets of five disks were collected from the mature bracts of 'Red Success' plants that were free of bract necrosis.

The extraction and spectrophotometric (modified vanillin-HCl microassay) measurements of condensed tannins were from Price et al. (1978) as modified by (Lees et al., 1995) for cells containing anthocyanins. The condensed tannin in the five bract disks in each sample were extracted with 1 mL 1% HCl-methanol in a screw cap vial for 30 min at 25 °C with agitation. Following condensed tannin extraction, 0.25 ml aliquots of the supernatant were transferred to three separate vials. The volume was brought up to 0.5 mL with 0.25 mL 1% HCl-methanol added to each sample, and then two of the three vials each received 2.5 mL of vanillin reagent. Vanillin reagent was prepared fresh by mixing equal volumes of 1% vanillin in methanol and 8% concentrated HCl in methanol. The third sample vial served as a blank control (without vanillin) and 2.5 mL of 4% HCl-methanol was added to this vial. After 30 min at 25 °C, each of the sample duplicates was read at A₅₀₀ using a Shimadzu UV160U spectrophotometer (Shimadzu Corp, Kyoto, Japan) and the absorbency of the blank was subtracted from the samples containing vanillin. Duplicate standard curves were constructed simultaneously to the sample analysis using catechin concentrations up to 0.5 mg·mL⁻¹. Thus, the condensed tannin concentrations were estimated as catechin equivalents. Data were analyzed using analysis of variance procedures as a completely random design with five replications and duplicate samples.

Results

Light microscopy revealed that small occlusions form in the bract epidermal cells (both adaxial and abaxial) at the same time that visible symptoms of bract necrosis appear (Fig. 1). No occlusions were found in epidermal cells from healthy-appearing regions of bracts (Fig. 1A). As lesions began to form, small occlusions were observed in the epidermal cells (Fig. 1b). As bract necrosis progressed and lesions enlarged, the cellular occlusions also enlarged, eventually filling the cell (Fig. 1C). Where bract necrosis was advanced, occlusions filled adaxial and abaxial epidermal cells as well as portions of the subepidermal structure (Fig. 1D).

Transmission electron micrographs of bracts that were just starting to develop the earliest signs of necrosis revealed numerous dense osmiophilic bodies lining the inner wall of the tonoplast (Fig. 2A). These spherical bodies, which appeared to be condensed tannins, were not observed in thin sections from healthy appearing portions of bracts sprayed with either deionized water (Fig. 2B) or Si (Fig. 2D) or Sr (not shown). In necrotic portions of bracts, condensed tannin bodies were also observed in the subepidermal cells and the cells surrounding the vascular tissue (Fig. 2C). In cells

next to the collapsed cells, tannin bodies filled a greater portion of the cells (Fig. 2G), and eventually filled the entire vacuole as symptoms progressed (not shown).

The initial development of tannin bodies did not result in the disintegration of membranous cell structures. In subepidermal cells with condensed tannin bodies (Fig. 2F), chloroplasts with starch grains, osmiophilic lipid globules, and granal stacks were observed along side the plasmalemma and the tonoplast membranes. Except for the tannin bodies lining the margins of the vacuole, the chloroplasts, cellular membranes and cell walls appeared similar to those observed in sections from healthy-appearing bract tissues (Fig. 2E).

Small samples (6 mm diameter) of 'Supjibi Red' bracts with visible necrotic lesions of ≈2 mm in diameter contained higher concentrations of condensed tannins, 59 mg·g⁻¹ fresh mass ($P \leq$

Fig. 2. Transmission electron micrographs of thin sections from 'Supjibi Red' poinsettia bracts. (A) Epidermal cells from the vicinity of a developing necrotic lesion. Numerous condensed tannin bodies are evident in the vacuoles (arrow). (B) Epidermal cells from a healthy appearing portion of a deionized water sprayed bract. No condensed tannin bodies are evident. (C) Close up of a section of subepidermal cell from a necrotic portion of the bract. Condensed tannin bodies (arrows) of various sizes can be seen lining the vacuole membrane in the neighboring cells. (D) Epidermal cell from a healthy-appearing silica sprayed bract. No condensed tannin bodies are evident. (E and F) Single chloroplasts from healthy appearing and necrotic sections of the bract, respectively. The chloroplasts lie between the plasmalemma, and tonoplast membranes and the chloroplast in each sample appears intact with osmiophilic lipid globules, starch grains, and thylakoid membrane (grana) stacks evident. Condensed tannin bodies are evident in the vacuole of the cells excised from a necrotic portion of the bract (F). (G) Condensed tannin bodies in the vacuoles of epidermal cells excised from a necrotic portion of the bract. A collapsed cell is adjacent to an intact cell. Scale bars are equal to a length of 4 μm in A, B, C, D, and G, and 1 μm in E and F.

Fig. 3. Scanning electron micrographs of the adaxial surface of 'Supjibi Red' bracts. (A) A developing necrotic lesion. (Inset A) shows a collapsed cell next to an intact cell. (B) Portion of a deionized water sprayed bract that did not exhibit symptoms of bract necrosis. (C) Portion of a strontium sprayed poinsettia bract that appeared healthy. (D) Portion of a silica sprayed poinsettia bract that appeared healthy. Inset D shows magnified view of silica sprayed epidermal cells. The scale bar in A is equal to a length of 200 μm in A, B, C and D. The scale bar in (inset A) is equal to a length of 20 μm in Inset A and Inset D.

0.05), than 'Supjibi Red' bracts samples with lesions of <0.5 mm in diameter ($13 \text{ mg}\cdot\text{g}^{-1}$ fresh mass). By comparison, condensed tannin concentrations averaged $0.2 \text{ mg}\cdot\text{g}^{-1}$ fresh mass in samples from healthy-appearing portions of 'Supjibi Red' bracts, and $1.0 \text{ mg}\cdot\text{g}^{-1}$ fresh mass in samples from the necrosis resistant 'Red Success'.

Examination of the adaxial bract surface under the scanning electron microscope revealed areas of collapsed epidermal cells on bracts that had displayed the earliest signs of bract necrosis (Fig. 3A). These damaged areas of 15 to 20 contiguous cells covered an area of $\approx 0.01 \text{ mm}^2$ and appeared as small dark-stains when the samples were collected under $40\times$ magnification. Apparently, when the earliest symptoms of necrosis were first observed some cells were already weakened. Similar scans of tissue samples from healthy-appearing regions of bracts had few collapsed cells (Fig. 3B). In contrast, scanning electron micrographs of Sr (Fig. 3C) or Si sprayed bracts (Fig. 3D) revealed healthy adaxial epidermal cells. Collapsed cells may have been artifacts resulting from the dehydration and tissue preparation process, since a similar collapse was not observed under light microscopy and the collapse of weakened plant cells often results from the preparation process used in this study (M. Cantino, personal communication). Regardless, it appears that cell weakening was associated with either the

accumulation of condensed tannins or the process associated with the accumulation of condensed tannins.

Discussion

The presence of condensed tannin bodies in the vacuoles of the epidermal and subepidermal cells is the earliest observed cellular event associated with the onset of poinsettia bract necrosis. Based on micrographic evidence and/or chemical analysis, condensed tannins appear to be virtually absent in healthy-appearing portions of bracts of 'Supjibi Red', in the necrosis resistant 'Red Success', or in healthy-appearing bract tissue from plants sprayed with Sr or Si. Both Sr and Si sprays are known to suppress the development of bract necrosis (McAvoy and Bible, 1996a, 1996b). Therefore, tannin accumulation is positively correlated with the stage of development of bract necrosis, and is associated with the localized area immediately surrounding a necrotic lesion.

Although accumulation of polyphenols, and the derived tannins, in cells appears to be commonly associated with plant disease (Walker, 1957), our micrographic observations revealed no evidence of fungi, bacteria or virus particles associated with bract necrosis. This is consistent with the lack of transmission of bract necrosis from a susceptible to a resistant culti-

var by grafting (McAvoy and Bible, 1998).

It is not clear what causes the localized alteration of phenolic metabolism leading to condensed tannin accumulation, why cells with high concentrations of condensed tannins ultimately die, or how treatments such as Ca, Si, Sr, and benzyladenine sprays suppress the development of bract necrosis symptoms. In necrotic poinsettia bracts subepidermal cells with condensed tannin bodies showed the same level of intracellular integrity, in the chloroplast for instance, as cells without condensed tannins. Struckmeyer (1960) using the cultivar Ruth Ecke observed leaf tissue from plants supplied with inadequate levels of Ca and found a dark staining substance between palisade cells; but, we did not observe extracellular occlusions in bracts. However, she did observe occlusions in the adaxial and abaxial epidermal cells of poinsettia leaves from plants supplied with inadequate levels of zinc or boron.

Inking disorder of peach (*Prunus persica*) fruit epidermal tissue results from the formation of metallo complexes between ferric iron and polyphenols or tannins (Arakawa and Ogawa, 1994; Cheng and Crisosto, 1997). Inking disorder is preceded by the abrading of epidermal cells which allows iron and copper, in wash water or from fungicide sprays, to bind with tannins and form dark-colored metallo complexes (Arakawa and Ogawa, 1994; Cheng and Crisosto, 1997). However, there is no physical damage to poinsettia bracts associated with the development of bract necrosis nor is there any relationship between the incidence of necrosis and bract margin iron concentrations (McAvoy and Bible, 1996a).

In russet spotting of iceberg lettuce (*Lactuca sativa* L.), accumulation of soluble phenols that readily oxidize to form the brown discoloration precedes the symptoms of this disorder (Ke and Saltveit, 1988). Russet spotting is ethylene-induced in the presence of high phenylalanine ammonia-lyase (PAL) activity (Ke and

Saltveit, 1988). Both calcium and auxin can inhibit russet spotting by inhibiting PAL activity (Ke and Saltveit, 1986). However, unlike poinsettia bract necrosis which can be suppressed by Sr or benzyladenine sprays, russet spotting was not suppressed by Sr or by hormones other than auxins.

In summary, our observations suggest an association between the localized accumulation of condensed tannins in poinsettia bracts and the appearance of the dark-stained spots that precede the development of necrotic lesions. Although subcellular structures appear intact until well after tannins accumulate, tannin accumulation is the first visible cellular event associated with bract necrosis. However, the mechanism that triggers the onset of bract necrosis is still unknown.

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