J. Amer. Soc. Hort. Sci. 139(5):603–615. 2014.

Characterization and Comparison of Lesions on Ornamental Sweetpotato 'Blackie', Tomato 'Maxifort', Interspecific Geranium 'Caliente Coral', and Bat-faced Cuphea 'Tiny Mice'

Joshua K. Craver[1], Chad T. Miller[2], and Kimberly A. Williams[3,4]
Department of Horticulture, Forestry, and Recreation Resources, Kansas State University, 2021 Throckmorton Hall, Manhattan, KS 66506

Daniel L. Boyle[2]
Division of Biology, Kansas State University, Manhattan, KS 66506

Additional index words. *Cuphea llavea*, *Ipomoea batatas*, oedema, intumescence, *Pelargonium*, *Solanum lycopersicum*

Abstract. **Many plant species are prone to physiological disorders in which lesions develop on the leaf tissue. Nomenclature for such lesions has included intumescences, excrescences, neoplasms, galls, genetic tumors, enations, and oedemata. Interchangeably using these terms causes confusion as to whether these names refer to the same or different disorders. Two of the most commonly used names are oedema and intumescence. The objective of this research was to characterize the development of lesions on ornamental sweetpotato (*Ipomoea batatas* 'Blackie'), tomato (*Solanum lycopersicum* 'Maxifort'), interspecific hybrid geranium (*Pelargonium* × 'Caliente Coral'), and bat-faced cuphea (*Cuphea llavea* 'Tiny Mice') to determine similarities and differences in morphology and nomenclature among these physiological disorders. Light microscopy was used to characterize differences in cross-sectional height, width, and area of lesions on each species. Additionally, leaf tissue samples were embedded in paraffin, and 10-μm cross-sections were stained with Toluidine blue O and observed using light microscopy to identify specific cell layers involved with lesion development. Field emission scanning electron microscopy (SEM) and digital photography were used to observe the microscopic and macroscopic stages of lesion development, respectively, on each species. The lesions observed on ornamental sweetpotato were significantly greater in height and area than on the other three species, whereas tomato lesions were significantly greater in width. Lesions on ornamental sweetpotato and bat-faced cuphea occurred predominantly on the adaxial surface of the leaf, whereas lesions on geranium and tomato occurred predominantly on the abaxial surface. With lesions on tomato, ornamental sweetpotato, and bat-faced cuphea, the epidermis was often subjected to the same hypertrophy apparent in the underlying parenchyma cells, ultimately allowing for greater cell expansion. However, in geranium, the epidermis resisted the expansion of the underlying cells, resulting in the eventual tearing of this tissue layer. Previous research indicates that lesion development on geranium is closely related to water status within the plant and may result in a wound response or provide a means of facilitated gas exchange. On the contrary, development of lesions on ornamental sweetpotato and tomato is believed to involve light quality. Based on these results and observations, two disorders occur across these species. The term "intumescence" should be used when referring to abnormal lesions on ornamental sweetpotato and tomato, and the term "oedema" should be used when referring to lesions on geranium. The term "intumescence" should also be used when referring to bat-faced cuphea lesions resulting from the morphological and anatomical aspects of these lesions closely resembling development on ornamental sweetpotato and tomato. Future research should investigate the role of light quality regarding development on this species.**

Intumescence is a physiological disorder that develops sporadically on the leaf tissue of many plant species, including tomato (Rud, 2009), sweetpotato (Wetzstein and Frett, 1984), and cuphea [*Cuphea* sp. (Jaworski et al., 1988)]. This disorder is often described as abnormal, translucent outgrowths on the leaf surface with a gall or wart-like appearance (Morrow and Tibbitts, 1988; Wetzstein and Frett, 1984). Although the term intumescence is commonly used to describe this disorder, other common and interchangeably used nomenclature in the published literature includes excrescences, neoplasms, galls, genetic tumors, leaf lesions, enations, and oedemata (Pinkard et al., 2006).

Interchangeably using these terms causes confusion as to whether these names refer to the same or different disorders. White (1951) reviewed neoplastic growths on plant tissue and concluded that there was a wide variety of such growths that existed and that these growths may have originated by many different means. Two of the most commonly used names when referring to these growths are intumescence, as described previously, and oedema. Oedema is most commonly found on geranium, whereas the term intumescence is

Received for publication 28 May 2014. Accepted for publication 17 July 2014. This research was funded by The Fred C. Gloeckner Foundation and K-State Research and Extension.
This manuscript has been assigned Contribution no. 14-384-J from the Kansas Agricultural Experiment Station (KAES).
We thank the following individuals, all from Kansas State University, for their assistance: Lea Westervelt, Myrna Gabriela Cruz, Shelly Christenson, Megan Kennelly, and Judy O'Mara.
Mention of a trademark, proprietary product, or vendor does not constitute a guarantee, or warranty of the product by the U.S. Dept. of Agriculture and does not imply its approval to the exclusion of other products or vendors that also may be suitable.
[1]Graduate Research Assistant.
[2]Assistant Professor.
[3]Professor.
[4]Corresponding author. E-mail: kwilliam@ksu.edu.

more often associated with growths on sweetpotato and tomato.

Dale (1905) suggested that these disorders were equivalent by stating that these growths were first called ''intumescences,'' but the term ''oedemata'' was later adopted by American authors. For decades, including today, many plant scientists have adopted (or accepted) that the two names were synonymous. However, Lang and Tibbitts (1983) suggested that the names oedema and intumescence refer to completely different disorders. According to these authors, oedema can best be defined as ''a 'watery swelling of plant organs or parts,' resulting from water congestion in plant tissue'' (Lang and Tibbitts, 1983). On the other hand, they suggested the disorder observed on tomato in their research should be termed intumescence, because the plants exhibited symptoms when relative humidity levels were low and water status within the tissue was normal (Lang and Tibbitts, 1983). Morrow and Tibbitts (1988) added to this differentiation by stating that ''edema'' has traditionally been found to develop under conditions where excess water and high humidity prevent sufficient transpiration by the plant.

Rangarajan and Tibbitts (1994) further described and suggested differences between oedema and intumescence. Based on the results obtained in their research involving the failure of far-red light to inhibit oedema injury on ivy geranium (*Pelargonium peltatum*), they suggested that the causative factors and physiological systems that regulate oedema on geranium and intumescence on solanaceous species were different. They supported this statement through their observation that ultraviolet radiation aids in the prevention of intumescence on solanaceous species (Morrow and Tibbitts, 1988) but has little to no effect regarding the development of oedema on geranium (Rangarajan and Tibbitts, 1994).

There are multiple instances in which microscopy methods have been used to assist in better defining and understanding the development of these disorders. Light microcopy has been used to evaluate intumescences on tomato (Lang et al., 1983), sweetpotato (Wetzstein and Frett, 1984), and eucalyptus [*Eucalyptus nitens* and *E. globulus* (Pinkard et al., 2006)] and oedema on geranium [*Pelargonium ×hortorum* (Balge at al., 1969; Metwally et al., 1970)] and eggplant [*Solanum melongena* (Eisa and Dobrenz, 1971)]. Additionally, electron microscopy has been used to further analyze intumescences on sweetpotato (Wetzstein and Frett, 1984) and tomato (Rud, 2009) leaves.

Although lesions have been previously observed using microscopy on a single species, no extensive comparison of these lesions among different species has been conducted. To remove bias related to accurately characterizing this physiological disorder on each species, the term ''lesion'' is used when referring to these abnormal growths. The objective of this research was to further characterize lesion nomenclature and development on four plant species: ornamental sweetpotato 'Blackie', tomato 'Maxifort', interspecific hybrid geranium 'Caliente Coral', and bat-faced cuphea 'Tiny Mice'. Specific goals were to 1) use digital imaging, including field emission SEM (FESEM) and photography, to evaluate stages of development for the disorders on each species; and 2) characterize and determine cellular layers involved in lesion development to evaluate differences and similarities in abnormal cellular growth among the species.

Materials and Methods

Plant materials. Rooted cuttings of ornamental sweetpotato were obtained from a commercial supplier (Four Star Greenhouses, Carlton, MI) and were potted on 20 Jan. 2014 in 11.4-cm-diameter (465 mL volume) pots. Tomato seeds were sown on 20 Nov. 2013 and were kept under mist until transplanting into 15.2-cm-diameter (940 mL volume) pots on 9 Dec. 2013. Plants were transplanted a second time on 5 Feb. 2014 into 11-L-volume pots. Interspecific hybrid geranium tip cuttings were taken from stock plants maintained in greenhouses at Kansas State University's Throckmorton Plant Sciences Center (Manhattan) and stuck in foam medium (Oasis® Wedge® Growing Medium; Smithers-Oasis North America, Kent, OH) on 17 Dec. 2013; cuttings were transplanted on 2 Jan. 2014 into 12.7-cm-diameter (625 mL volume) pots. Rooted cuttings of bat-faced cuphea were obtained from a commercial supplier (Kaw Valley Greenhouses, Manhattan, KS) and were potted on 6 Mar. 2014 in 11.4-cm-diameter (465-mL-volume) pots. A peat-based root medium (Fafard #2; Conrad Fafard, Agawam, MA) was used at each instance of transplant. Plants were grown at a greenhouse temperature set point of 22/20 °C (day/night). Data loggers (Onset Computer Corp., Bourne, MA) were placed in the greenhouse to monitor temperature and relative humidity. Plants were fertigated with a 200 $mg \cdot L^{-1}$ nitrogen constant liquid feed using 20N–4.4P–16.6K (Peters Professional Peat-Lite Special; Everris NA, Dublin, OH) when greater than half of the plants reached ≈45% container capacity as a result of water loss.

Digital photography and FESEM. Lesions on each species were photographed at three separate stages of development (Canon 7D; Canon U.S.A., Melville, NY). These stages included 1) initial lesion development and early expansion; 2) full expansion of the lesions and beginning stages of senescence; and 3) complete senescence of the lesion and surrounding tissue. These stages of development were also imaged using FESEM. For FESEM imaging, tissue displaying the specific stage of each disorder was excised into ≈0.5-cm-diameter sections and fixed using a 2% paraformaldehyde/2% glutaraldehyde solution in a 0.2 M phosphate-buffered saline (PBS) solution, pH 7.2. The tissue was fixed for 2 h and then transferred to a 0.2 M PBS solution, pH 7.2, until imaging was conducted. Tissue sections were then mounted on carbon tape and imaged using FESEM (Nova NanoSEM 430; FEI Co., Hillsboro, OR). Images were collected using a low-vacuum detector at a chamber pressure of ≈0.98 torr with water vapor.

Light microscopy cross-sectional measurements. Lesions on each species were characterized by measuring the cross-sectional height, width, and area of three lesions on six separate plants per species at the same stage of development. The first stage of development, described previously, was targeted where lesions would be fully expanded. On 20 Feb. 2014, six ornamental sweetpotato plants displaying signs of lesion development were sampled. A single leaf from each plant that was near full expansion but had not fully developed the typical purple pigmentation was selected. The lesions targeted for sampling were translucent protrusions on the adaxial surface that had not yet begun to senesce. On 21 Feb. 2014, six tomato plants displaying signs of lesion development were sampled. A single leaf from each plant, five to seven nodes from the apical meristem, was selected and on that leaf, the terminal three leaflets were used for sampling. The lesions

sampled were green protrusions or bumps on the abaxial surface that had not yet begun to senesce. On 26 Feb. 2014, six geranium plants displaying signs of lesion development were sampled. From each plant, two to three leaves (excluding the youngest leaves approximately two nodes from the apical meristem) were selected for sampling. The sampled lesions were green protrusions or bumps on the abaxial surface that had not yet begun to senesce. On 12 May 2014, six bat-faced cuphea plants displaying signs of lesion development were sampled. From each plant, two to three leaves (excluding the youngest leaves approximately three nodes from the apical meristem) were selected for sampling. The sampled lesions were green protrusions or bumps on the adaxial surface that had not yet begun to senesce.

Leaf sections of ≈1 cm in diameter with developing lesions were excised for each species. These were further sectioned to 200-µm-thick cross-sections using a tissue chopper (TC-2; Sorvall Instruments, Newtown, CT). Sections were carefully selected so that the optimal center of each lesion was achieved. This selection of the optimal center involved selecting cross-sections closest to the exact center of the lesion, producing the most accurate measurements of maximum lesion cross-sectional height, width, and area. Three sections of different representative lesions from each of the six plants for each species were mounted with deionized water and observed using a light microscope (Eclipse E600; Nikon, Melville, NY). Cross-sectional lesion measurements were obtained using ImageJ Processing and Analysis in Java (National Institutes of Health, Bethesda, MD). Lesion height (micrometers) was measured from the apex of the lesion to the leaf lamina surface (Fig. 1A). Lesion width (micrometers) was measured along the epidermis with start and end measurement points where the lesion began to protrude (Fig. 1A). Lesion area (square millimeters) was measured above the leaf lamina surface (Fig. 1B). Homogeneity of variance for each response variable was evaluated using the Browne-Forsythe test ($\alpha = 0.05$). For the height response variable, a log transformation was necessary to achieve homogeneous variance and meet model assumptions. Additionally, for the area response variable, a reciprocal square root transformation was used. These data were analyzed using a general linear mixed model that fit a completely randomized design. Data were analyzed using the GLIMMIX procedure of SAS (Version 9.2; SAS Institute, Cary, NC). Multiple comparisons were made using the transformed data for both height and area measurements. Estimated means are reported before transformation.

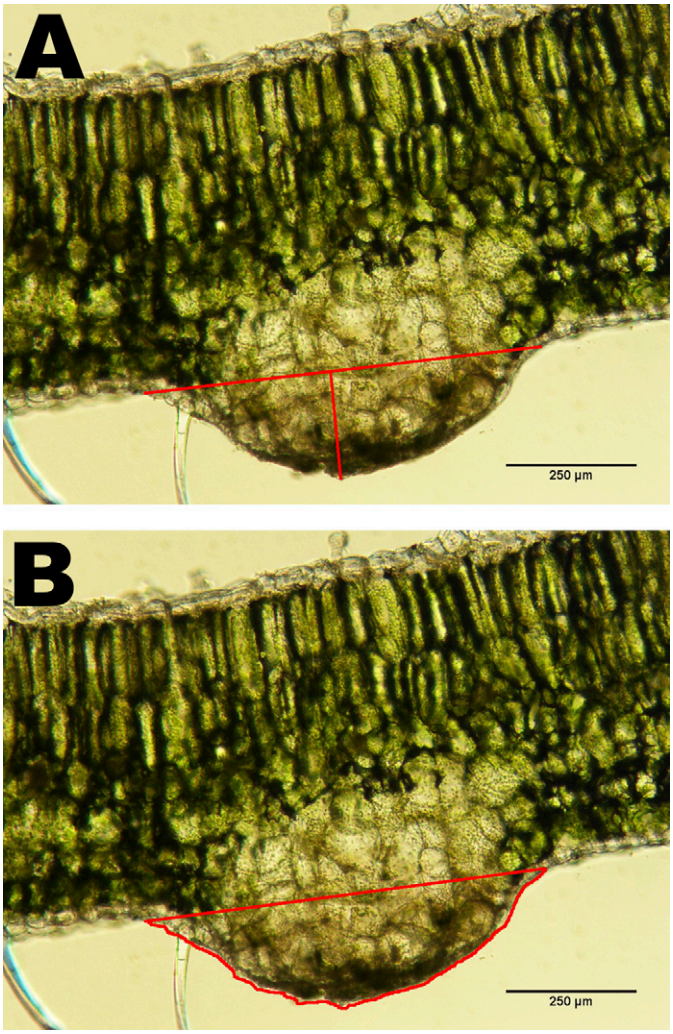

Fig. 1. Method of measuring lesion cross-sectional height, width, and area on interspecific hybrid geranium using ImageJ Processing and Analysis in Java (National Institutes of Health, Bethesda, MD). (**A**) Height (micrometers) was measured from the apex of the lesion to the leaf lamina surface and width (micrometers) along the epidermis. (**B**) Area (square millimeters) of the lesion was measured above the leaf lamina surface.

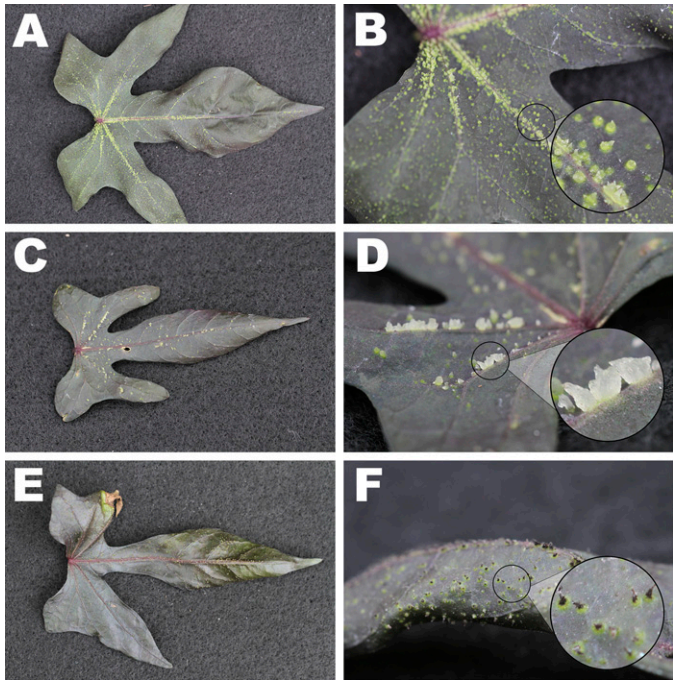

Fig. 2. Digital images of three stages of lesion development on the adaxial leaf surface of ornamental sweetpotato. (**A**) Leaf displaying the early stages of lesion development as small green bumps began to form along the leaf veins as well as interveinally. (**B**) Leaf area close-up characterizing the early stages of lesion development as rounded green lesions formed and began to elongate. (**C**) Leaf displaying further elongated and increasingly translucent lesions, which characterized the intermediate stages of development. (**D**) Leaf area close-up characterizing the significant hypertrophy of cells, which gave rise to the increasingly translucent appearance of the lesions. (**E**) Leaf displaying the latter stages of lesion development, typical on more mature leaves, with the senescence of lesions apparent. (**F**) Leaf area close-up characterizing the senescence and blackening of the lesion apex.

Light microscopy of samples stained with Toluidine blue O. The same sampling criteria described for the light microscopy measurements was used for Toluidine blue O (TBO) staining. Leaf sections, ≈0.5 cm in diameter, were excised from plants of each species displaying lesion development. The leaf sections were fixed in a 10% formalin solution for 2 h, removed from the fixative, and subjected to an ethanol dehydration series before being mounted in paraffin. Cross-sections (10 µm thick) were obtained using a microtome, then mounted and stained with 0.5% TBO. Images were collected using the same light microscope listed in the previous section.

Results and Discussion

Ornamental sweetpotato

Morphology. Lesions on ornamental sweetpotato initiated predominantly on the adaxial surface of leaves approaching full expansion, similar to results reported by Wetzstein and Frett (1984). In the earliest stages of development, lesions appeared as small green bumps that formed along leaf veins as well as interveinally (Fig. 2A–B). Both individual and small groupings of lesions were observed. Lesion formation appeared to initiate around the stomata because guard cells displayed the first signs of cellular hypertrophy (Fig. 3A). After the initiation of guard cell hypertrophy, the surrounding epidermal cells also appeared to hypertrophy (Fig. 3B). Wetzstein and Frett (1984) reported similar observations as they observed extensive hypertrophy that included the enlargement of stomata.

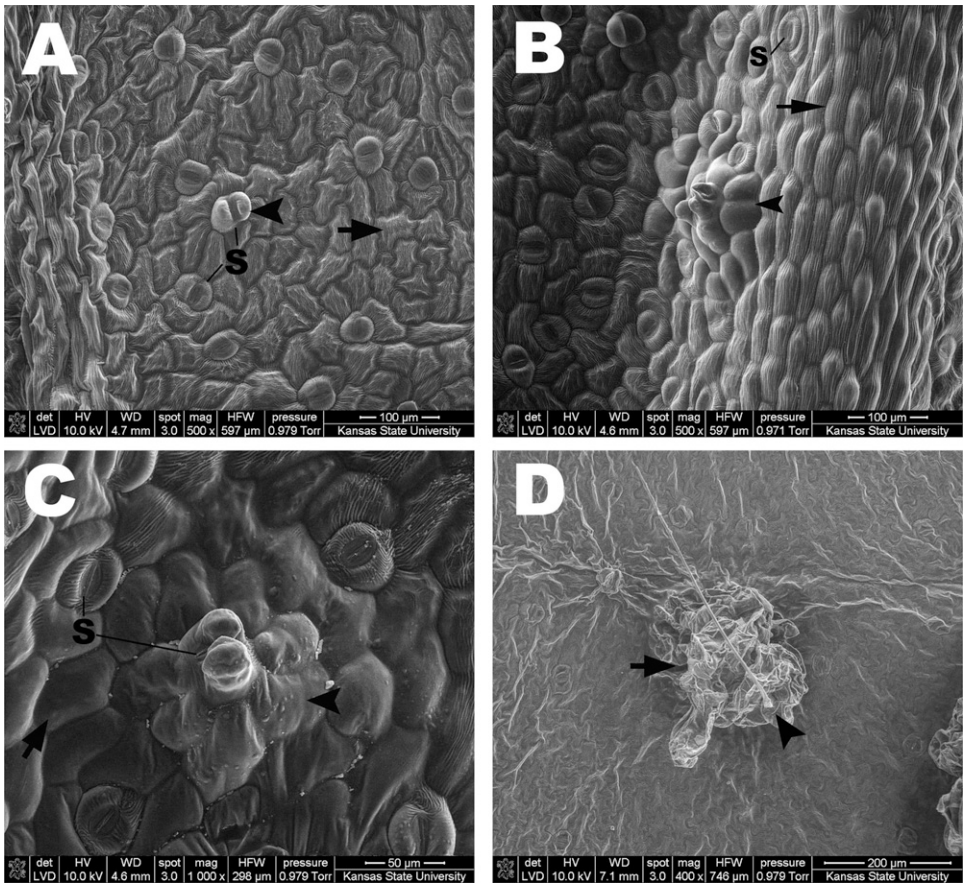

Fig. 3. Field emission scanning electron microscopy of the adaxial leaf surface of ornamental sweetpotato. (**A**) Hypertrophy of guard cells (point) occurred during the initial stages of lesion development; hypertrophy did not appear to affect normal epidermal cells (arrow) during these initial stages. Additional labeling includes stoma (S). (**B**) Hypertrophy of multiple cells (point) during the early stages of lesion development that occurred on the side of a leaf vein (arrow). (**C**) Lesion elongation involved the surrounding epidermal cells (point) with stomata often located at the lesion apex. Normal cells (arrow) around the lesion are unaffected by hypertrophy. (**D**) Lesion in the intermediate stages of development (arrow) with a trichome (point) located near the lesion apex.

In the intermediate stages of development, the lesions became more elongated and translucent (Fig. 2C–D). The more translucent coloration observed was the result of extensive cell hypertrophy. As the cells continued to expand, anthocyanin and chlorophyll pigments were not observed. Our observations are similar to the findings of Wallace (1928), who described the fate of chloroplasts in hypertrophied cells of *Malus* ×*domestica* 'Transparent', reporting that the chlorophyll granules gradually lost their green color, eventually disappearing completely. Moreover, La Rue (1932) reported that hypertrophic poplar (*Populus tremuloides* and *P. grandidentata*) leaf cells did not display a change in the number of chloroplasts contained within the cell; rather, the chloroplasts were spaced more widely apart and the cells appeared translucent rather than green.

Lesion senescence was observed in the latter stages of development (Fig. 2E). These stages occurred mostly on mature foliage, as the leaf continued to age after the initial onset of lesion development. The senescence began as a blackening of the lesion apex and ultimately led to the senescence of the entire lesion (Fig. 2F). These senesced lesions remained on the leaf surface or abscised over time.

Anatomy. Ornamental sweetpotato leaves had a single-layered upper and lower epidermis, a single-layered palisade parenchyma, and multiple layers of loosely packed spongy parenchyma (Fig. 4A). Lesions were mostly groups of hypertrophic cells that elongated above the leaf lamina surface (Fig. 4B). Lesions on ornamental sweetpotato displayed a much greater height than width (Table 1). These data support the tendency of these cells to elongate vertically as hypertrophy continued. The extensive amount of elongation present in these cells led to a high overall area (Table 1) of lesion growth above the leaf lamina.

Based on the light microscopy images with TBO staining, the cell layer that was most directly involved in lesion development was the palisade parenchyma (Fig. 4B). Hypertrophy of these cells resulted in the significant elongation of the lesion, which was also apparent in the height measurements observed. Additionally, it appeared that these hypertrophic palisade parenchyma cells pushed many of the unaffected upper epidermal cells to the side as continued elongation occurred (Fig. 4B). However, findings from the FESEM images suggested

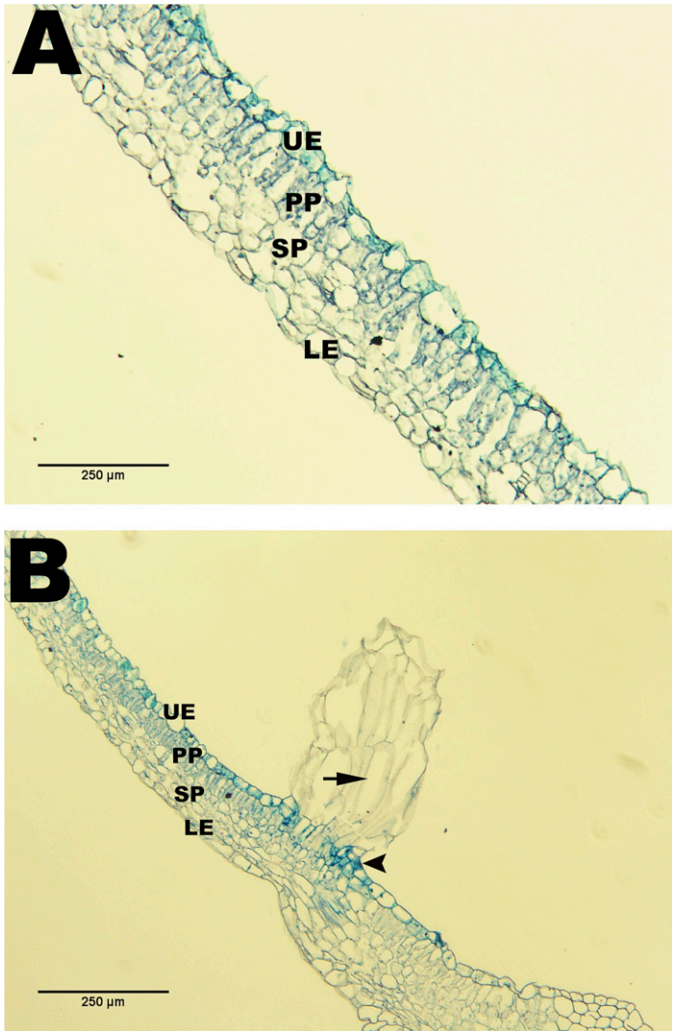

Fig. 4. Light microscopy cross-sections stained with Toluidine blue O of lesions on the adaxial leaf surface of ornamental sweetpotato. (**A**) Asymptomatic leaf; tissue with no lesion development. Additional labeling includes the lower epidermis (LE), palisade parenchyma (PP), spongy parenchyma (SP), and upper epidermis (UE). (**B**) Lesion displaying significant hypertrophy of palisade parenchyma cells above the leaf lamina surface (arrow). Epidermal cells (point) are pushed aside as palisade parenchyma cells expand.

Table 1. Estimated mean lesion height, width, and area on bat-faced cuphea, interspecific hybrid geranium, ornamental sweetpotato, and tomato taken on cross-sections (n = 6).

Species	Lesion ht (µm)[z]	Lesion width (µm)	Lesion area (mm^2)
Bat-faced cuphea	240.4 b[y]	775.5 b	0.13 b
Geranium	186.6 c	807.5 b	0.10 b
Sweetpotato	613.2 a	375.6 c	0.23 a
Tomato	155.9 c	1140.6 a	0.12 b

[z]Estimated means before transformation are reported for height and area.
[y]Estimated means with a common letter within each column are not significantly different based on multiple comparisons at $\alpha = 0.05$.

the participation of the upper epidermis in the onset of these hypertrophic lesions resulting from the incorporation of stomata and the surrounding epidermal cells in lesion expansion (Fig. 3C). This involvement of the upper epidermis is further suggested by the occurrence of trichomes near the lesion apex (Fig. 3D). Thus, it is proposed that epidermal cells are mostly incorporated in the initiation and early stages of lesion development, whereas continued lesion expansion predominantly involves the hypertrophy palisade parenchyma cells. This finding is similar to that of Wetzstein and Frett (1984), who stated that intumescences developing on sweetpotato leaves involved the hypertrophy and hyperplasia of both the epidermis and palisade parenchyma.

We should point out that the FESEM results should be evaluated with caution. The FESEM techniques used to observe stages of lesion development could cause tissue desiccation, especially during intermediate and latter stages. Precautions such as the use of water vapor in the microscope chamber to maintain tissue turgidity and minimizing the duration of beam time on a single focused point were taken to avoid cell collapse; however, artifacts of the imaging process are plausible.

Although hypertrophy was the most evident cellular response, hyperplasia may have also occurred. The palisade parenchyma cells underwent hypertrophic elongation and expansion predominantly, but hyperplasia was also evidenced as a result of what appeared to be an increase in the number of cellular layers within the palisade parenchyma (Fig. 4B). Wetzstein and Frett (1984) reported that intumescences on sweetpotato formed as a result of the occurrence of both hypertrophy and hyperplasia. On the other hand, Trotter (1904) indicated that intumescence development on sweetpotato was solely the result of cellular hypertrophy. In our study, we observed that although hyperplasia may have been present, the most evident response was the extensive hypertrophy of these cells.

Tomato

Morphology. Lesion development on tomato occurred predominantly on the abaxial surface of the leaf, typically beginning five to seven leaves below the apical meristem. The early stages of development appeared as large white–green bumps (Fig. 5A–B). Lesions initiated as groups of single hypertrophic cells scattered across the abaxial surface (Fig. 6A), which ultimately spread horizontally to encompass many of the surrounding cells to form a mounded shape. Lesion development was not concentrated in a specific area of the leaf such as near the veins, but occurred sporadically.

The intermediate stages of lesion development exhibited senescence as the surface of the lesions turned brown and began to collapse (Fig. 5C–D). Large senescent areas on the leaf surface where the lesions once formed characterized the latter stages of lesion development (Fig. 5E). Although the lesions initially developed in a solitary fashion, on collapse the affected areas coalesced to form large senesced regions (Fig. 5F). This cellular collapse (Fig. 6B) occurred through all leaf tissue layers, causing these senescent regions to be observed on both the abaxial and adaxial surfaces of the leaf (Fig. 7). This finding shows that the extent of the damage is not solely restricted to the cell layers immediately affected by the abnormal cell growth. In severe occurrences of lesion development, the entire leaflet would begin to senesce on lesion collapse (Fig. 7). Rud (2009) speculated that the cells involved with these lesions would ultimately rupture as a result of extensive hypertrophy. Regardless of whether rupture occurred, the result was always cellular collapse.

Anatomy. Tomato leaf tissue had a single-layered upper and lower epidermis, a single-layered palisade parenchyma, and

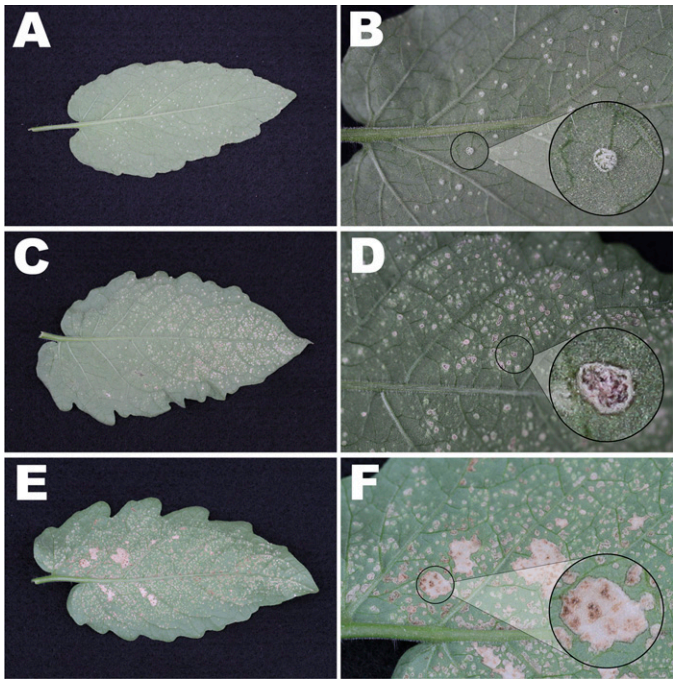

Fig. 5. Digital images of three stages of lesion development on the abaxial leaf surface of tomato. (**A**) Leaf displaying the early stages of lesion development as rounded bumps formed sporadically on the surface. (**B**) Leaf area close-up characterizing solitary lesions that appeared as white–green bumps. (**C**) Leaf displaying the intermediate stages of development as lesions began to senesce and collapse. (**D**) Leaf area close-up characterizing lesions that became brown as a result of senescence and began to collapse. (**E**) Leaf displaying the latter stages of lesion development as large senescent regions began to appear where lesions previously collapsed. (**F**) Leaf area close-up characterizing a large senesced region where multiple lesions previously collapsed.

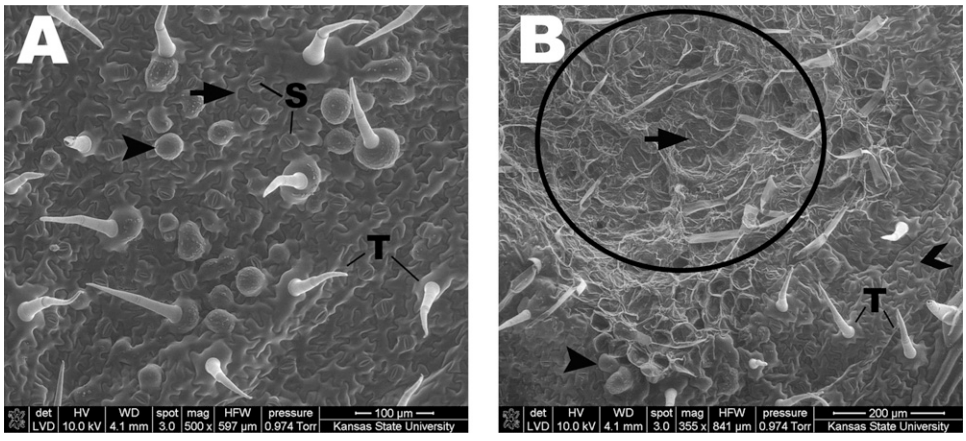

Fig. 6. Field emission scanning electron microscopy of the abaxial leaf surface of tomato. (**A**) The initiation of lesion development occurred as epidermal cells sporadically underwent hypertrophy (point) on the leaf surface. Surrounding cells were often unaffected at this stage (arrow). Additional labeling includes stoma (S) and trichome (T). (**B**) A large collapsed lesion (circle) that appeared to be multiple cell layers deep (arrow). Hypertrophic cells (point) were still apparent around the lesion perimeter. The collapsed lesion did not result in the senescence of surrounding cells at this stage (caret).

a loosely arranged spongy parenchyma (Fig. 8A). These anatomical observations regarding tomato leaf tissue are similar to those of Lang et al. (1983). However, our study differed in the location of lesion development. Lang et al. (1983) reported that lesions formed primarily on the adaxial surface of the leaf, whereas we found lesions predominantly on the abaxial surface.

The lesions appeared to expand both horizontally and vertically (Fig. 8B). Both the spongy parenchyma and lower epidermis were involved in lesion development with the potential for rupture of hypertrophied epidermal cells also apparent as a result of cells swelling many times greater than their normal size (Fig. 8B). These findings are similar to those of Lang et al. (1983) in which they observed hypertrophy of the palisade parenchyma and upper epidermis that led to the occasional rupture of epidermal cells. As such, extensive hypertrophy was the most evident response with hyperplasia in the spongy parenchyma also proposed as a result of an increase in spongy parenchyma cell layers (Fig. 8B). Additionally, lesions on tomato were greater in width than height (Table 1). This may be the result of these cells undergoing more horizontal expansion than vertical. The horizontal expansion, resulting in greater lesion width, is logical because of the loosely packed spongy parenchyma layer, which would have allowed for more expansion within this tissue before cells were forced above the leaf lamina surface. It is likely that this expansion occurring within the confines of the mesophyll led to the lower area of lesion growth above the epidermis when compared with lesions on ornamental sweetpotato (Table 1). Although our study suggests that hyperplasia may play a role in lesion development on tomato, this finding was not observed by Lang et al. (1983).

Geranium

Morphology. Lesion development on geranium occurred solely on the abaxial leaf surface. For the early stages of development, small green bumps formed sporadically (Fig. 9A–B). Leaves that were nearing full expansion or mature were most susceptible to this disorder. Younger, underdeveloped leaves had no signs of lesion development, which is in accordance with previously reported literature (Balge et al., 1969; Metwally et al., 1970). Similar to lesion development on ornamental sweetpotato, lesions on geranium often formed along veins and near the petiole, although development was not limited to these areas of the leaf. The lesions formed as both solitary growths and groupings and were noticeably smooth and rounded (Fig. 9B). This smoothness was likely the result of the epidermal layer not specifically being affected by cellular hypertrophy. Rather, pressure appears to have been placed on the lower epidermis from the expansion of underlying cells, which ultimately caused a reduction in the definition between epidermal cells (Fig. 10A). Thus, it appears that the epidermis was stretched as a result of the expansion of the mesophyll underneath.

In the intermediate stages of development, the lesions appear to have senesced (Fig. 9C), because a brown coloration formed at the lesion apex (Fig. 9D). We propose this browning was the result of cellular senescence, initiated from the tearing of the lower epidermis resulting from mesophyll hypertrophy (Fig. 10B–C).

Fig. 7. Tomato leaflet undergoing senescence and epinasty resulting from the collapse of lesions on the abaxial surface. Lesion collapse resulted in senescent regions on the adaxial surface as well.

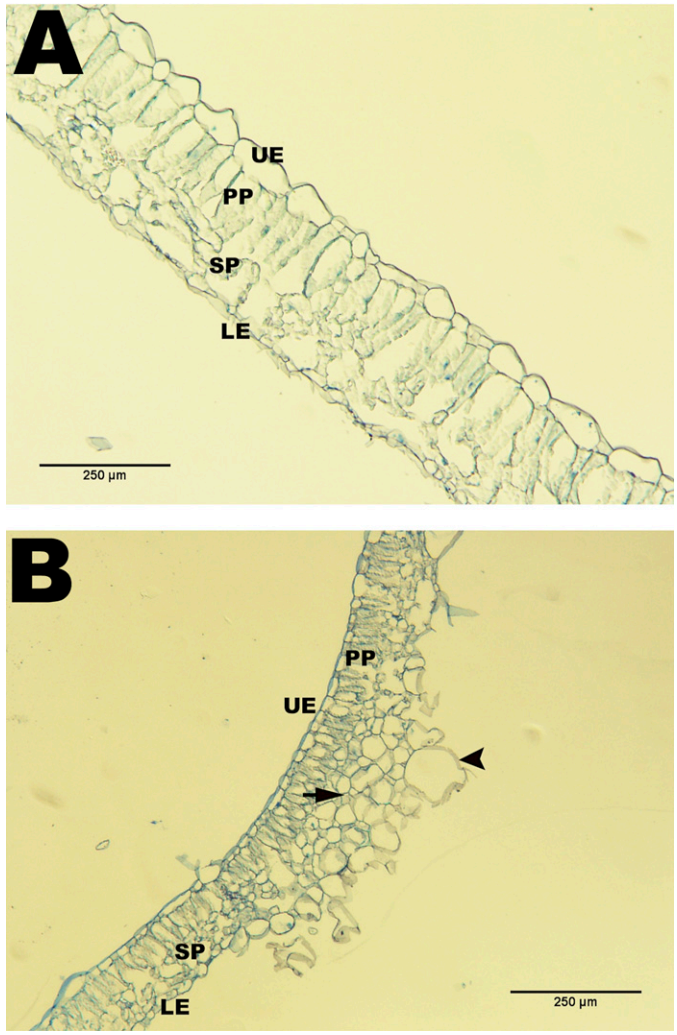

Fig. 8. Light microscopy cross-sections stained with Toluidine blue O of lesions on the abaxial leaf surface of tomato. (**A**) Asymptomatic leaf; tissue with no lesion development. Additional labeling includes the lower epidermis (LE), palisade parenchyma (PP), spongy parenchyma (SP), and upper epidermis (UE). (**B**) Lesion displaying lower epidermis and spongy parenchyma cells undergoing significant hypertrophy (point) and hyperplasia (arrow) both horizontally and vertically.

On significant expansion of the underlying cell layer, the epidermis would have been torn from the increasing pressure. Continued browning and senescence on the lesion surface were observed in the latter stages of development (Fig. 9E–F), similar to the lesion senescence observed on tomato. However, in contrast to later stages of tomato development, the senesced lesions did not always appear to collapse. When lesion collapse was present (Fig. 9F), the adaxial surface of the leaf was occasionally affected in a response similar to that seen on tomato. Under these circumstances, a small senescent circle directly underneath the collapsed lesion would appear on the adaxial surface (Fig. 11). Regardless, lesions did not spread to the surrounding tissues on the abaxial leaf surface, as observed in tomato. On closer examination, we propose that these areas affected by lesion development may develop layers of suberized cells as a wound response to the tearing of the lower epidermis. This wound response is discussed further in the following section.

Anatomy. Geranium leaf tissue was comprised of a single-layered upper and lower epidermis, a single-layered palisade parenchyma, and multiple layers of spongy parenchyma (Fig. 12A). The lesions developed solely on the abaxial surface and involved both the hypertrophy and hyperplasia of spongy parenchyma cells (Fig. 12B). The lower epidermal cells were not involved in the development of these lesions. Rather, the lower epidermis was subjected to tension as the spongy parenchyma cells beneath expanded and multiplied. This ultimately resulted in tearing of the lower epidermis (Fig. 12B). This evidence was further supported as lesions on geranium showed a greater width than height (Table 1). This may have been the result of increased periclinal division in the spongy parenchyma. Balge et al. (1969) reported similar observations, because they found that spongy parenchyma cells would undergo periclinal division. This further supports the finding in our study that hyperplasia was apparent within this cell layer. Additionally, until this epidermal layer was torn, the cells could only expand outward as far as the lower epidermis would allow. Thus, as the cells underwent hypertrophy and hyperplasia, the lesion was forced to grow horizontally. This restriction of lesion growth by the epidermis was also the reason for a lower total area affected when compared with lesion development on ornamental sweetpotato (Table 1).

Our observations of spongy parenchyma cells undergoing hypertrophy are in agreement with Balge et al. (1969) and Metwally et al. (1970); however, in their studies with lesion development on geranium, they reported hypertrophy of epidermal cells as well. Metwally et al. (1970) concluded that the swelling of both parenchyma and epidermal cells resulted in

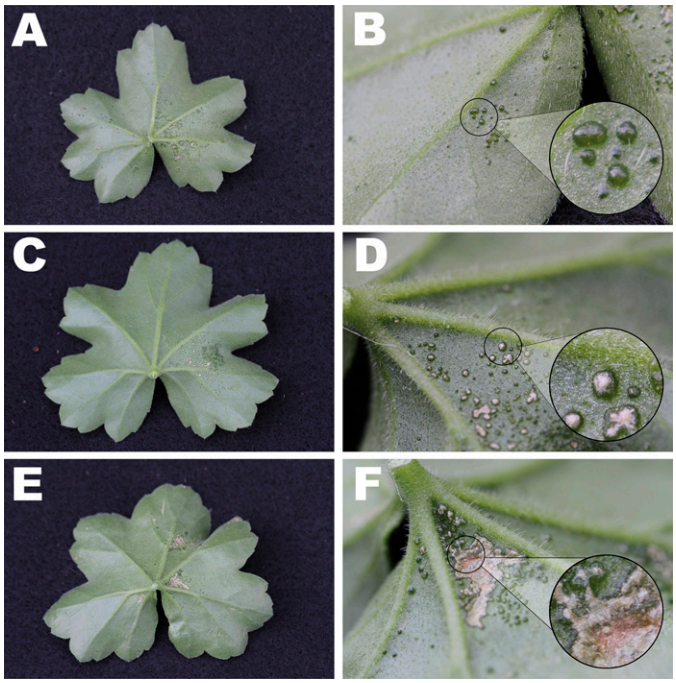

Fig. 9. Digital images of three stages of lesion development on the abaxial leaf surface of interspecific hybrid geranium. (**A**) Leaf displaying the early stages of lesion development as small green bumps formed sporadically on the surface. (**B**) Leaf area close-up characterizing the smooth and rounded appearance of lesions that often appeared in small groupings. (**C**) Leaf displaying the intermediate stages of lesion development as senescence was initiated. (**D**) Leaf area close-up characterizing lesion senescence, which originated around the lesion apex. (**E**) Leaf displaying the latter stages of development as lesions senesced and formed large regions of senescent cells. (**F**) Leaf area close-up characterizing a large region of senesced cells and lesion collapse.

pressure on the guard cells, which caused them to close, and ultimately resulted in stomatal collapse. Their findings are in contrast to ours, because epidermal cells did not undergo hypertrophy. Rather, we observed that the lower epidermis might have been subjected to tearing as a result of excessive pressure from the hypertrophic spongy parenchyma cells. These results are similar to the findings of La Rue (1932). While observing lesion development on poplar leaves, he found that the mesophyll cells closest to the epidermis underwent the most expansion, whereas the epidermis was then forced to stretch as these cells swelled outward. This outward growth was the result of very limited space for lateral expansion among the palisade and spongy parenchyma layers. Additionally, Schrenk (1905) described a similar phenomenon in cauliflower (*Brassica oleracea* var. *botrytis*) leaves where mesophyll cells, both spongy and palisade parenchyma, would enlarge until eventually breaking through the epidermis.

Balge et al. (1969) further stated that the senesced regions in the latter stages of lesion development were spongy parenchyma cells that had redifferentiated into a cork cambium layer. As these cork cambium cells elongated, the authors proposed that a raised periderm was formed. Although leaves usually do not produce periderm (Esau, 1977; Fahn, 1982), they have been found to produce cork cells as a result of wounding, where living plant tissue is exposed to the ambient air (Fahn, 1982). Under these conditions, the dead plant tissue will become separated from the living by a layer of suberized cells. Thus, phellogen may develop and give rise to both phellum and a phelloderm. The layer of cork that is formed would then allow for protection against pathogens as well as prevent water loss through the newly developed wound (Fahn, 1982). However, for the lesion development on geranium observed in the present study, this explanation does not fully characterize the phenomenon. The hypertrophy of spongy parenchyma cells appeared to occur before the exposure of the interior tissues to the ambient air. Thus, although the development of suberized cells may act as a wound response to epidermal tearing, it does not explain why the spongy parenchyma cells would initially undergo hypertrophy and hyperplasia to cause this tearing. Additionally, although a protective layer of suberized cells may have been formed, these lesions on geranium would still often collapse (Fig. 9F). We propose that this observation may have been the result of the extensive hypertrophy of the spongy parenchyma cells, which, regardless of the protective layer of cells formed above, ultimately collapsed.

The wound response proposition in geranium is further supported when observing western flower thrips (*Frankliniella occidentalis*) feeding damage on the abaxial surface of the leaf. Damage by this pest on ivy geranium has been described as pale yellow to dark brown spots on the abaxial leaf surface (Chen and Williams, 2006). Thus, thrips feeding damage may appear very similar to the late stages of lesion development on geranium (Fig. 13). Thrips will feed on both epidermal and mesophyll cells by damaging the cell tissue and then imbibing the cellular fluids (Cloyd, 2010). In the case of both thrips feeding damage and lesion development on geranium, the epidermal layer is compromised. Thus, although the mechanism of tissue wounding may differ, the wound response by geranium resulting from this epidermal damage may be identical. As a result, the proposed idea of a wound response would assist in explaining the similarity between thrips feeding damage and the development of lesions on geranium leaves.

Pinkard et al. (2006) suggested a similar wound response theory as they observed intumescence development on the leaves of eucalyptus seedlings. The authors proposed that these intumescences were actually environmentally induced lenticel-like structures (ERLS). They believed that these ERLS formed on leaves under the conditions of high relative humidity as a means to facilitate increased aeration of the interior tissues. Lenticels will often develop underneath stomata (Esau, 1977; Fahn, 1982). The underlying mesophyll cells beneath these stomata undergo a series of divisions until a phellogen is formed. The phellogen will then push the overlying cells outward until the epidermis ruptures. A response similar to the function of lenticel development may have been observed in the lesion development on geranium in the present study. Lesion development on geranium commonly occurred underneath stomata (Fig. 10A) until the eventual rupture of the epidermis (Fig. 10B). Thus, on tearing, it is plausible that the exposed epidermal cells would senesce while a layer of suberized cells was redifferentiated below (Fahn, 1982) (Fig. 14). Although lesions on geranium are not anatomically similar to lenticels, it is possible that this physiological response may serve a similar function in facilitating increased gas exchange to the underlying tissues.

Bat-faced cuphea

Morphology. Lesion development on bat-faced cuphea occurred on both the adaxial and abaxial surface of the leaf,

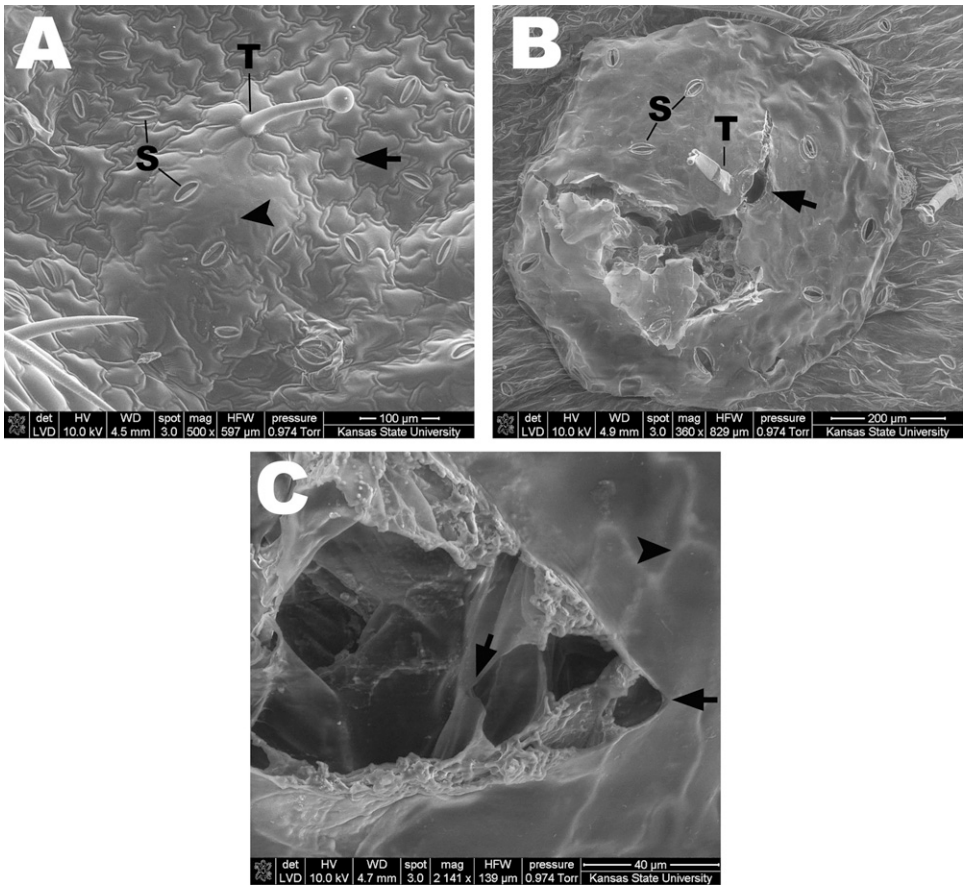

Fig. 10. Field emission scanning electron microscopy of the abaxial leaf surface of interspecific hybrid geranium. (**A**) Lesion development initiated as epidermal cells were stretched (point) as a result of the expansion of underlying mesophyll cells. A loss in the definition of these stretched epidermal cells was apparent when compared with the surrounding unaffected cells (arrow). Additional labeling includes stoma (S) and trichome (T). (**B**) The lower epidermis was torn (arrow) across the lesion surface as a result of pressure from mesophyll expansion below. (**C**) Evidence of epidermal tearing resulting from tension, where epidermal cells have lost definition (point) and torn (arrows) resulting from underlying mesophyll cell expansion.

Fig. 11. Interspecific hybrid geranium leaf displaying a small circle of senesced cells (arrow) on the adaxial surface where a lesion had previously collapsed on the abaxial surface directly below.

but was predominantly found on the adaxial surface during this study. This is in contrast to previous literature, where Jaworski et al. (1988) reported that this disorder mostly developed on the abaxial leaf surface. In our research, the more prevalent adaxial lesions were primarily observed and documented. Similar to lesion development on geranium, leaves that were nearing full expansion or mature were most susceptible to this disorder, whereas the younger, underdeveloped leaves showed no signs of lesion development. These findings are similar to those of Jaworski et al. (1988), who found that lesions were absent from very young leaves on susceptible cuphea species. Early stages of lesion development appeared as light green bumps that formed sporadically across the leaf surface (Fig. 15A–B). Under severe cases of development, lesions would coalesce and form large groupings or masses (Fig. 15A). Lesion development initiated as individual epidermal cells underwent hypertrophy (Fig. 16A), similar to lesion initiation on tomato. These individual hypertrophic cells would continue to expand and elongate resulting in the formation of large masses (Fig. 16A).

The intermediate stages of lesion development were characterized by the senescence of lesions (Fig. 15C–D). This senescence began at the lesion apex and would spread to encompass the remainder of the cells over time (Fig. 16B). It appeared that this senescence may have been caused by cellular rupture or collapse (Fig. 16B). The latter stages of lesion development depicted continued senescence of the lesions, which ultimately resulted in cellular collapse (Fig. 15E–F). Under severe cases of lesion development, large senescent regions were formed as collapse occurred (Fig. 15F). Similar to tomato, this cellular collapse was observed on both the abaxial and adaxial surface of the leaf, indicating that all cell layers were ultimately affected by lesion development.

Anatomy. Bat-faced cuphea leaf tissue had a single-layered upper and lower epidermis, a single-layered palisade parenchyma, and a loosely arranged spongy parenchyma (Fig. 17A). Lesion development involved the extensive hypertrophy of upper epidermal cells as well as the elongation of palisade parenchyma cells (Fig. 17B). With the upper epidermal cells undergoing significant hypertrophy, it is highly probable that cell rupture eventually occurred. This would have led to the senescence and collapse of the lesions observed during the intermediate and latter stages of development. Lesions on the abaxial surface were also occasionally observed with development predominantly involving the hypertrophy of spongy parenchyma cells (Fig. 17C). When lesion development occurred on the abaxial surface, it appeared that spongy mesophyll cells elongated and forced pressure on the lower epidermis. This pressure may eventually have led to the rupture of the lower epidermis, resulting in the senescence that was later observed. Thus, although development on the adaxial

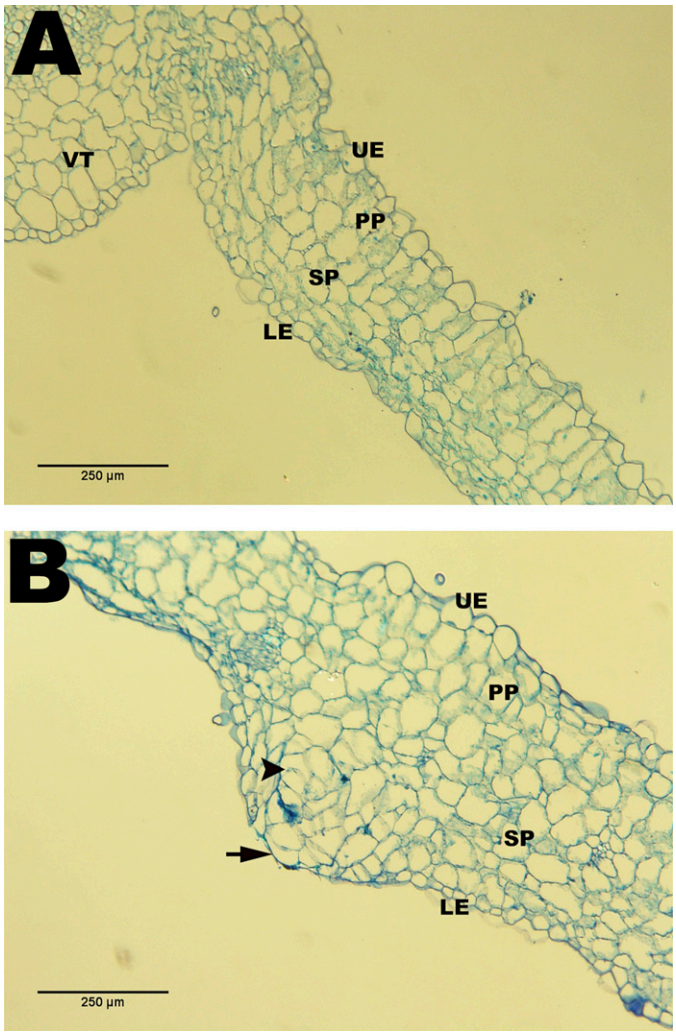

Fig. 12. Light microscopy cross-sections stained with Toluidine blue O of lesions on the abaxial leaf surface of interspecific hybrid geranium. (**A**) Asymptomatic leaf; tissue with no lesion development. Additional labeling includes the lower epidermis (LE), palisade parenchyma (PP), spongy parenchyma (SP), upper epidermis (UE), and vascular tissue (VT). (**B**) Hypertrophy and hyperplasia of spongy parenchyma cells (point) applied pressure to the lower epidermis. Evidence for tearing of the lower epidermis (arrow) as the epidermal cells became increasingly thin from the underlying mesophyll cell expansion.

Fig. 13. Western flower thrips (arrow) feeding damage on the abaxial leaf surface of interspecific hybrid geranium. Damage (point) appears similar to the latter stages of lesion development on geranium, suggesting a potential wounding response.

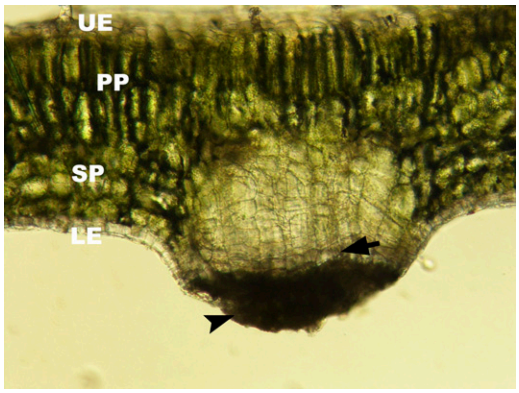

Fig. 14. Cross-section (200 µm thick) of a lesion on the abaxial leaf surface of interspecific hybrid geranium. Senescing cells at the apex of the lesion appear black (point), whereas stacked cells underneath may act as a protective layer and be suberized (arrow). Additional labeling includes the lower epidermis (LE), palisade parenchyma (PP), spongy parenchyma (SP), and upper epidermis (UE).

surface closely related to the lesion development observed on tomato and ornamental sweetpotato, development on the abaxial surface more closely resembled that of geranium. Evidence of hyperplasia was also seen within the spongy parenchyma cells when lesions developed on the abaxial surface (Fig. 17C).

Light microscopy cross-sectional measurement comparisons. Lesion development on ornamental sweetpotato was more than three times greater in estimated mean height compared with those on geranium and tomato and more than two times greater compared with lesions on bat-faced cuphea (Table 1). The difference in cellular expansion between species was further observed by the estimated average width measurements, because geranium and bat-faced cuphea lesions were roughly two times wider than those on ornamental sweetpotato with tomato lesions measuring roughly three times wider (Table 1). We propose that the greater average height for the growths on ornamental sweetpotato and bat-faced cuphea was the result of the cells involved predominantly being the palisade parenchyma. With this cell layer being more tightly packed together than the spongy parenchyma, the hypertrophic cells tended to elongate upward as a result of this constraint. On the contrary, the naturally occurring air spaces within the spongy parenchyma of tomato leaves allowed for greater horizontal cell expansion and possibly division, resulting in the shorter height and more horizontal lesion development. Additionally, the greater lesion width in geranium might be explained either by an increase in periclinal divisions of the spongy parenchyma or by the restriction of outward growth from the lower epidermis, causing cellular expansion to spread horizontally until the epidermis was torn. The greater lesion width in bat-faced cuphea was likely related to a greater number of cells undergoing hypertrophy within an area.

The average lesion area for ornamental sweetpotato was more than double those on geranium, bat-faced cuphea, and tomato (Table 1). This was also the result of the tendency of lesions on ornamental sweetpotato to display greater vertical elongation of hypertrophic cells, resulting in a greater lesion area. Intuitively, with much of the cellular growth occurring horizontally within the mesophyll in geranium and tomato, the

total lesion area above the epidermis for those two species would be significantly lower. In the case of bat-faced cuphea, elongation of the palisade parenchyma was not as severe as that observed in ornamental sweetpotato, leading to less total area measured above the lamina surface.

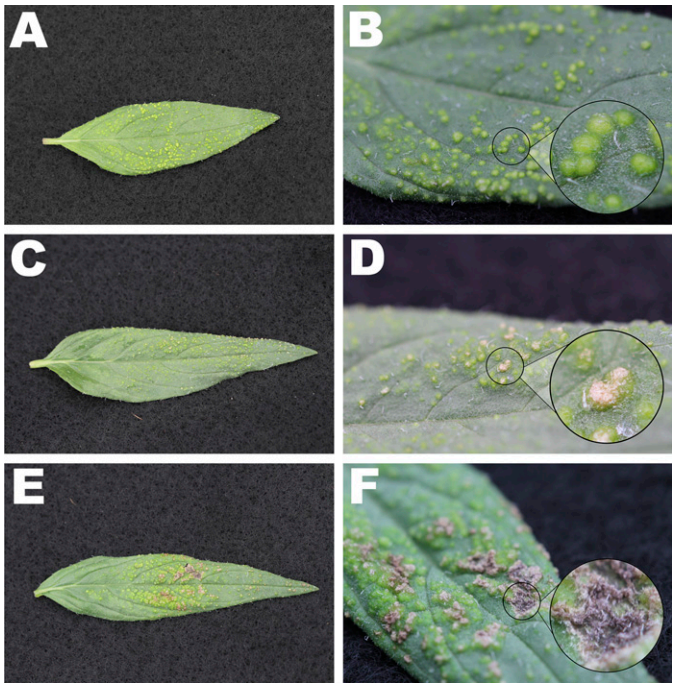

Fig. 15. Digital images of three stages of lesion development on the adaxial leaf surface of bat-faced cuphea. (**A**) Leaf displaying the early stages of lesion development as small bumps formed sporadically on the surface both individually and in small groupings. (**B**) Leaf area close-up characterizing lesions that appeared as light green bumps. (**C**) Leaf displaying the intermediate stages of development as lesions began to senesce. (**D**) Leaf area close-up characterizing lesions that became light brown as a result of senescence, typically beginning at the lesion apex. (**E**) Leaf displaying the later stages of lesion development as senescent regions began to appear where groupings of lesions had collapsed. (**F**) Leaf area close-up characterizing a collapsed region where multiple lesions previously developed.

Conclusions

An understanding of the causative factors related to lesion development on each species further assists in providing a comprehensive characterization and comparison. Light quality, specifically ultraviolet B (UVB) radiation, was found to be directly related to preventing or reducing lesion development on ornamental sweetpotato (J.K. Craver, C.T. Miller, K.A. Williams, and N.M. Bello, unpublished data) and tomato (Lang and Tibbitts, 1983; Morrow and Tibbitts, 1987, 1988; Rud, 2009). On the contrary, UVB was found to have no effect regarding lesion development on geranium (Rangarajan and Tibbitts, 1994). Rather, lesion development on geranium is thought to be affected by water relations; specifically, that high humidity, warm soils, and poor ventilation are conducive to development (Balge et al., 1969; Lang and Tibbitts, 1983; Metwally et al., 1970; Rangarajan and Tibbitts, 1994). This finding led Rangarajan and Tibbitts (1994) to propose that "oedema injury" on geranium may have different causative factors and be regulated by different physiological systems than on solanaceous species.

With lesions on tomato, ornamental sweetpotato, and bat-faced cuphea, the epidermis was often subjected to the same hypertrophy apparent in the underlying cells. However, in geranium the epidermis resisted the expansion of the underlying cells, resulting in the eventual tearing of the tissue layer. Because lesions on geranium are thought to be caused by water congestion and high humidity, a plant response that results in a function similar to that provided by lenticels, as proposed by Pinkard et al. (2006), is an appropriate hypothesis. Because leaves were unable to transpire at rates equivalent to the amount of water uptake, the cells underneath the stomata may have undergone a response to facilitate increased aeration. Thus, swelling and division of spongy parenchyma cells would have continued until eventual tearing of the lower epidermis. This response involving facilitated gas exchange does not seem to be related to lesion development on tomato, because epidermal cells underwent significant hypertrophy, and this hypertrophy did not seem to instigate around stomata. Additionally, lesion development on tomato resulted in the ultimate senescence of leaf tissue, which does not support the idea of a wound response or response to facilitate gas exchange for this species. However, lesions on ornamental sweetpotato appeared to initiate around the stomata as guard cells underwent hypertrophy. The occurrence of the lesions around the stomata is similar to the development on geranium and may point toward the involvement of air quality, hormones, or solutes in the initiation of lesion development. Although tearing of the epidermis was not apparent in lesions on

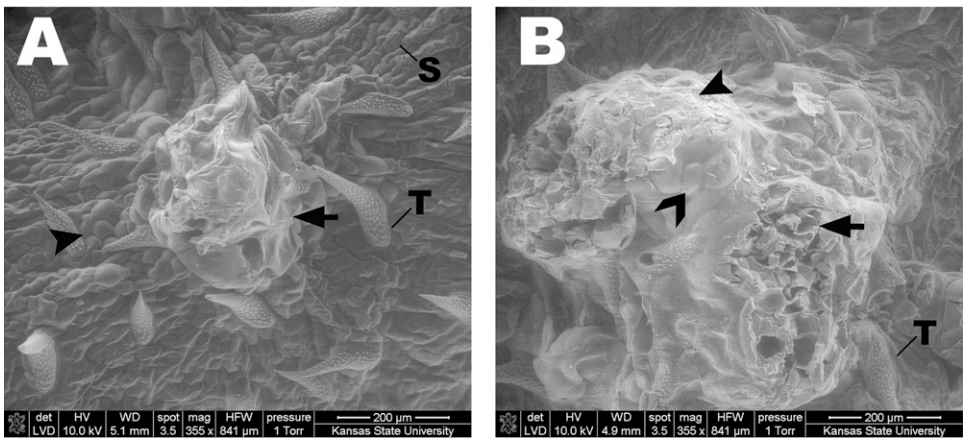

Fig. 16. Field emission scanning electron microscopy of the adaxial leaf surface of bat-faced cuphea. (**A**) The initiation of lesion development occurred as epidermal cells underwent hypertrophy (point) on the leaf surface. Cells would continue to undergo hypertrophy and coalesce to form large lesions (arrow). Additional labeling includes stoma (S) and trichome (T). (**B**) Lesion undergoing senescence near the apex during the intermediate stages of development (point). Some hypertrophic cells involved in lesion development appear to have ruptured or collapsed (arrow), whereas others remain turgid (caret).

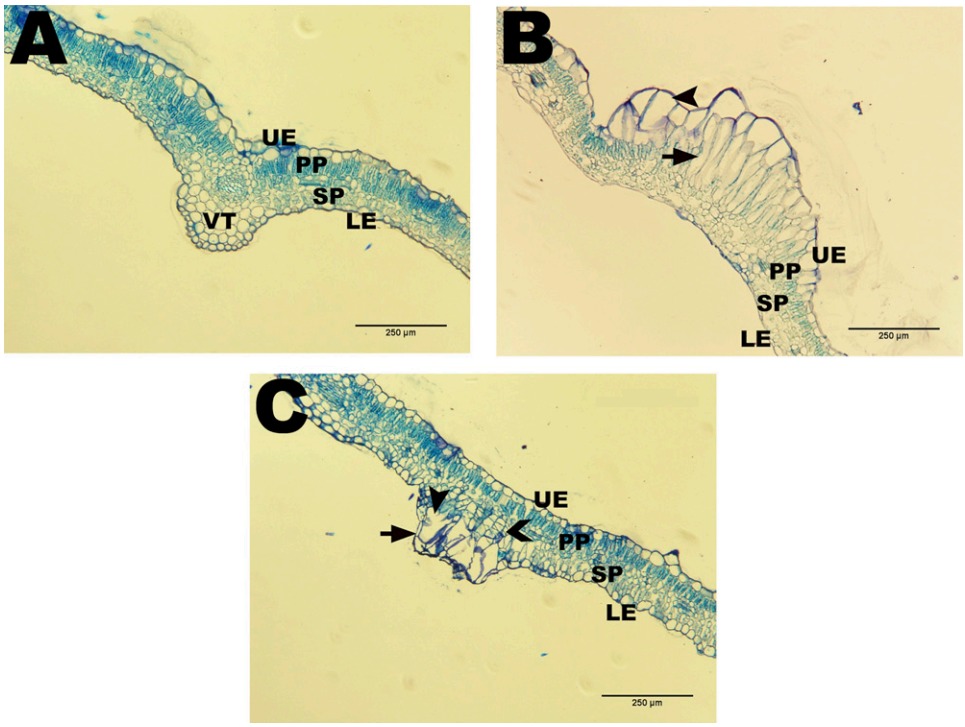

Fig. 17. Light microscopy cross-sections stained with Toluidine blue O of lesions on the adaxial and abaxial leaf surfaces of bat-faced cuphea. (**A**) Asymptomatic leaf; tissue with no lesion development. Additional labeling includes the lower epidermis (LE), palisade parenchyma (PP), spongy parenchyma (SP), upper epidermis (UE), and vascular tissue (VT). (**B**) Lesion developing on the adaxial surface displaying extensive hypertrophy of the upper epidermis (point) as well as the elongation of palisade parenchyma cells (arrow). (**C**) Lesion developing on the abaxial surface displaying the hypertrophy of spongy parenchyma cells (point), which are placing pressure on the lower epidermis and potentially leading to rupture (arrow). Evidence for hyperplasia in the spongy parenchyma is also evidenced (caret).

ornamental sweetpotato, it is probable that the epidermis was compromised around the stomata as the underlying palisade parenchyma elongated outward. This also may account for the multiple observations of stomata at the apex of lesion development on ornamental sweetpotato. However, the development of lenticel-like structures on ornamental sweetpotato seems unlikely because palisade parenchyma cells underwent significant hypertrophy above the epidermis soon after the initiation of lesion development with no apparent signs of cellular suberization.

UVB has been found to reduce stomatal density and opening on rice [*Oryza sativa* (Dai et al., 1995)], which would seemingly result in lower levels of transpiration and increased hypertrophic lesions if water congestion were the causative factor for lesion development on tomato and ornamental sweetpotato. Rather, UVB radiation has been found to be a preventive measure for these two species. Thus, it appears that lesion development on these species is not directly related to the need for increased gas exchange, but may involve a different physiologically mechanism from geranium lesion development altogether.

In conclusion, it is important to consider that plants possess a limited means by which to respond to stress. In other words, although the occurrence of hypertrophy was apparent in lesion development on all four species, the cause and mechanism by which these abnormal growths occur may differ between species. Thus, it is useful to consider the causative factors alongside morphological and anatomical aspects of lesion development to most accurately determine the appropriate terminology for these physiological disorders. Our results point toward a differentiation in lesion terminology. Specifically, that lesion development on geranium would be referred to as "oedema," whereas lesion development on tomato and ornamental sweetpotato would be referred to as "intumescence." Lesion development on geranium has previously been found to be closely related to water status within the plant and appeared to result in a wound response or provide a means of facilitated gas exchange. On the contrary, development of lesions on ornamental sweetpotato and tomato were found to involve light quality. Lesions on these two species resulted in cellular abnormalities, which often included the epidermis, that ultimately caused cell and tissue senescence. Lesion development on bat-faced cuphea has not been extensively studied to date. As a result, very little is known concerning causative factors resulting in development for this species. However, in terms of the morphological and anatomical aspects of development on the adaxial surface, this disorder more closely resembles lesions on ornamental sweetpotato and tomato. Therefore, we suggest that "intumescence" is the appropriate term. Future research should investigate the role of UVB radiation regarding intumescence development on cuphea species.

Literature Cited

Balge, R.J., B.E. Struckmeyer, and G.E. Beck. 1969. Occurrence, severity and nature of oedema in *Pelargonium hortorum* Ait. J. Amer. Soc. Hort. Sci. 94:181–183.

Chen, Y. and K.A. Williams. 2006. Quantifying western flower thrips (*Frankliniella occidentalis* Pergande) (Thysanoptera: Thripidae) damage on ivy geranium [*Pelargonium peltatum* (L.) L'Her ex Ait.] (Geraniaceae Juss.) with Adobe® Photoshop™ and Scion® image software. J. Kans. Entomol. Soc. 79:83–87.

Cloyd, R.A. 2010. Western flower thrips management on greenhouse-grown crops. 2 Apr. 2014. <http://www.ksre.ksu.edu/>.

Dai, Q., S. Peng, A.Q. Chavez, and B.S. Vergara. 1995. Effects of UVB radiation on stomatal density and opening in rice (*Oryza sativa* L.). Ann. Bot. (Lond.) 76:65–70.

Dale, E. 1905. Further experiments and histological investigations on intumescences, with some observations on nuclear division in pathological tissues. Proc. R. Soc. Lond., B 76:587–588.

Eisa, H.M. and A.K. Dobrenz. 1971. Morphological and anatomical aspects of oedema in eggplants (*Solanum melongena* L.). J. Amer. Soc. Hort. Sci. 96:766–769.

Esau, K. 1977. Plant anatomy. 2nd Ed. Wiley, New York, NY.

Fahn, A. 1982. Plant anatomy. 3rd Ed. Pergamon Press, Elmsford, NY.

Jaworski, C.A., M.H. Bass, S.C. Phatak, and A.E. Thompson. 1988. Differences in leaf intumescences between *Cuphea* species. HortScience 23:908–909.

La Rue, C.D. 1932. Intumescences on poplar leaves. I. Structure and development. Amer. J. Bot. 20:1–17.
Lang, S.P., B.E. Struckmeyer, and T.W. Tibbitts. 1983. Morphology and anatomy of intumescence development on tomato plants. J. Amer. Soc. Hort. Sci. 108:266–271.
Lang, S.P. and T.W. Tibbitts. 1983. Factors controlling intumescence development on tomato plants. J. Amer. Soc. Hort. Sci. 108:93–98.
Metwally, A.W., G.E. Beck, and B.E. Struckmeyer. 1970. The role of water and cultural practices on oedema of *Pelargonium hortorum* Ait. J. Amer. Soc. Hort. Sci. 95:808–813.
Morrow, R.C. and T.W. Tibbitts. 1987. Induction of intumescence injury on leaf disks. J. Amer. Soc. Hort. Sci. 112:304–306.
Morrow, R.C. and T.W. Tibbitts. 1988. Evidence for involvement of phytochrome in tumor development on plants. Plant Physiol. 88:1110–1114.
Pinkard, E., W. Gill, and C. Mohammed. 2006. Physiology and anatomy of lenticel-like structures on leaves of *Eucalyptus nitens* and *Eucalyptus globulus* seedlings. Tree Physiol. 26:989–999.
Rangarajan, A. and T.W. Tibbitts. 1994. Exposure with far-red radiation for control of oedema injury on 'Yale' ivy geranium. HortScience 29:38–40.
Rud, N.A. 2009. Environmental factors influencing the physiological disorders of edema on ivy geranium (*Pelargonium peltatum*) and intumescences on tomato (*Solanum lycopersicum*). Master's thesis, Kansas State Univ., Manhattan, KS.
Schrenk, H. 1905. Intumescences formed as a result of chemical stimulation. Missouri Bot. Garden Annu. Rpt. 16:125–148.
Trotter, A. 1904. Intumeszenze fogliari di "Ipomoea batatas.". Ann. Bot. (Lond.) 1:362–364.
Wallace, R.H. 1928. Histogenesis of intumescences in the apple induced by ethylene gas. Amer. J. Bot. 15:509–524.
Wetzstein, H.Y. and J.J. Frett. 1984. Light and scanning electron microscopy of intumescences on tissue-cultured, sweetpotato leaves. J. Amer. Soc. Hort. Sci. 109:280–283.
White, P.R. 1951. Neoplastic growth in plants. Qtrly. Rev. Biol. 26: 1–16.