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Responses of *Zinnia angustifolia* x *Z. violacea* Backcross Hybrids to Three Pathogens

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Abstract. **True-breeding lines of *Zinnia marylandica* Spooner, Stimart & Boyle [allotetraploids of *Z. angustifolia* H.B.K. and *Z. violacea* Cav. (2*n* = 4*x* = 46)] were backcrossed with autotetraploid *Z. angustifolia* (2*n* = 4*x* = 44) and *Z. violacea* (2*n* = 4*x* = 48). Seed-generated, backcross (BC_1) families were screened for resistance to alternaria blight (*Alternaria zinniae* Pape), bacterial leaf and flower spot [*Xanthomonas campestris* pv. *zinniae* (Hopkins and Dowson) Dye], and powdery mildew (*Erysiphe cichoracearum* DC. ex Merat). All BC_1 families exhibited high levels of resistance to alternaria blight and powdery mildew. BC_1 families derived from crossing *Z. marylandica* with autotetraploid *Z. angustifolia* were highly resistant to bacterial leaf and flower spot, whereas BC_1 families derived from crossing *Z. marylandica* with autotetraploid *Z. violacea* were susceptible to this disease. Our results suggest that one *Z. angustifolia* genome in BC_1 allotetraploids is sufficient to confer resistance to *A. zinniae* and *E. cichoracearum*, but at least two *Z. angustifolia* genomes are required in BC_1 allotetraploids to provide resistance to *X. campestris* pv. *zinniae*.**

The common zinnia [formerly *Z. elegans* Jacq., currently *Zinnia violacea* (McVaugh, 1984)] is subject to attack by three major pathogens: *Alternaria zinniae* (alternaria blight), *Erysiphe cichoracearum* (powdery mildew), and *Xanthomonas campestris* pv. *zinniae* (bacterial leaf and flower spot) (Andersen, 1971; Jones and Strider, 1979; Lipschutz, 1965; Torres, 1963). These pathogens incite moderate to severe epiphytotics within *Z. violacea* plantings, resulting in plant losses, decreased ornamental value, or both. The narrow-leaved zinnia [*Z. angustifolia* (McVaugh, 1984)] is highly resistant to these pathogens (Andersen, 1971; Jones and Strider, 1979; Lipschutz, 1965; Torres, 1963) and is a promising source of resistance genes for *Zinnia* breeding programs.

Crosses between *Z. angustifolia* (2*n* = 2*x* = 22) and *Z. violacea* (2*n* = 2*x* = 24) yield infertile allodiploids (2*n* = 2*x* = 23) (Boyle and Stimart, 1982; Terry-Lewandowski et al., 1984). Partially fertile, true-breeding allotetraploids (2*n* = 4*x* = 46) have been produced by treating allodiploids with colchicine (Boyle and Stimart, 1982; Terry-Lewandowski et al., 1984). Allotetraploids of *Z. angustifolia* and *Z. violacea* have been given the collective name *Z. marylandica* (Spooner et al., 1991). Terry-Lewandowski and Stimart (1983) found that *Z. marylandica* plants displayed high levels of resistance to *A. zinniae* and *E. cichoracearum*, and moderate to high levels of resistance to *X. campestris* pv. *zinniae*. Boyle (1996) obtained BC_1 hybrids when *Z. marylandica* was crossed with autotetraploid forms of *Z. angustifolia* (2*n* = 4*x* = 44) and *Z. violacea* (2*n* = 4*x* = 48). Our objective for this study was to determine the reactions of BC_1 plants to *A. zinniae*, *E. cichoracearum*, and *X. campestris* pv. *zinniae*.

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Materials and Methods

Plant material. Thirteen *Zinnia* accessions were used for this study (Table 1). The parentage of *Z. angustifolia* A88C, *Z. violacea* V86C, and *Z. marylandica* M1C and M2C, and the methods used to produce these lines, were reported previously (Boyle, 1996). Four BC_1 families were produced by crossing lines M1C and M2C (as females) with A88C and V86C. Henceforth, the parental material will be referred to by their genomic formulas instead of their chromosome numbers (Table 1).

Cultural procedures. Seeds were sown in plastic bedding plant trays (54.5 cm long × 28 cm wide × 6 cm deep). The propagation medium was maintained at ≈22 °C by bottom heat. Seedlings were transplanted into plastic cell-packs (eight packs per flat and six cells per pack), using one seedling per 92-cm^3 cell. A commercial soilless mix (Fafard Growing Mix No. 2; Conrad Fafard, Springfield, Mass.) was used for sowing seed and transplanting.

Plants in the glasshouse were fertilized at each irrigation with 20N–4.3P–16.6K at 200 mg N/L. The field plot used in Expt. 2 was amended with 10N–4.3P–8.3K at 50 $g \cdot m^{-2}$ before planting, and was fertilized twice (once in July and again in August) with 20N–4.3P–16.6K at 300 mg N/L.

Glasshouse temperature setpoints were 18/21/23 °C (heat/vent/exhaust). Actual glasshouse air temperatures in Expts. 1 and 3 were monitored with a LI-1000 datalogger (LI-COR, Lincoln, Neb.) equipped with an aspirated LI-1000-16 thermistor. The datalogger was configured with a sampling interval of 60 s and recorded mean temperatures at 1-h intervals. Shading compound (Kool Ray; Continental Products Co., Euclid, Ohio) was applied to the glass to maintain photosynthetic flux at ≤1200 $\mu mol \cdot m^{-2} \cdot s^{-1}$. Field plot temperatures (Expt. 2) were not recorded. Relative humidity (RH) was measured with a hygrometer (model 22-7059; Bacharach Instrument Co., Pittsburgh). All experiments were performed under natural daylengths (lat. 42°22.5′N).

Reactions to Alternaria zinniae *(Expt. 1).* Seed were sown on 14 Apr. 1991. Five-week-old seedlings were planted in a 1.1 × 14.0-m glasshouse raised bed that was filled with a soil-based growing medium (3 sphagnum peat : 1 coarse perlite : 2 coarse, loamy, mixed mesic Typic Fragiochrept). The bed was steam-pasteurized (≈80 °C for 30 min) 1 week before planting. The 12 entries were arranged in a randomized complete-block design with three blocks and one plot per block. Each plot consisted of 10 plants (two rows with five plants per row). Plants were spaced at a distance of 15 cm within rows and 30 cm between rows.

A single-spore isolate of *A. zinniae* obtained locally from diseased *Z. violacea* plants was used for inoculum. The fungus was cultured on carrot agar (200 g homogenized carrots and 15 g Difco Bacto-agar/L; Difco, Detroit) and incubated for 8 days in a growth chamber set at 25 ± 2 °C and providing ≈35 $\mu mol \cdot m^{-2} \cdot s^{-1}$ photosynthetic photon flux for 12 h daily from cool-white fluorescent lamps. Inoculum was prepared by gently scraping the surface of four petri plates with a spatula and washing the contents into 1 L of sterile distilled water containing 1 mL Tween 20/L (polyoxyethylene sorbitan monolaurate). The final concentration of the inoculum (determined by dilution-plating) was ≈1.2×10^3 colony-forming units (cfu)/mL. On 28 May, inoculum was applied with a pressurized hand sprayer onto the upper and lower leaf surfaces of each plant. Immediately after inoculation, a moist chamber was created by covering the ground bed with a white (0.1-mm-thick) polyethylene sheet. The polyethylene sheet was supported by two wires running parallel to the sides of the ground bed that were installed 1 m above the growing medium surface. Plants were misted at 2-h intervals to maintain a high (>95%) RH in the moist chamber (Terry-

Table 1. *Zinnia* accessions used for study and their chromosome numbers, genomic formulas, and sources.

Breeding line, cultivar, or hybrid	Chromosome no. (2*n*)	Genomic formula[z]	Source[y]
Z. angustifolia Classic	22	AA	BU
Linearis White	22	AA	SI
A88C	44	AAAA	MA
Z. violacea Canary Bird	24	VV	BO
Thumbelina Mini-Pink	24	VV	BO
V86C	48	VVVV	MA
Z. marylandica Rose Pinwheel	46	AAVV	BU
M1C	46	AAVV	MA
M2C	46	AAVV	MA
BC_1: *Z. marylandica* M1C x *Z. angustifolia* A88C	45[x]	AAAV	MA
BC_1: *Z. marylandica* M2C x *Z. angustifolia* A88C	45[x]	AAAV	MA
BC_1: *Z. marylandica* M1C x *Z. violacea* V86C	47[x]	AVVV	MA
BC_1: *Z. marylandica* M2C x *Z. violacea* V86C	47[x]	AVVV	MA

[z]A = *Z. angustifolia* (*n* = 11); V = *Z. violacea* (*n* = 12).
[y]BO = Bodger Seeds, Lompoc, Calif.; BU = W. Atlee Burpee Co., Warminster, Pa.; MA = Univ. of Massachusetts, Amherst; SI = Sutton and Sons (India) Pvt.,Calcutta, India.
[x]Chromosome numbers and genomic formulas for BC_1 plants are expected values and are not based on actual chromosome counts.

Lewandowski and Stimart, 1983). The moist chamber was removed 24 h after inoculation.

Disease reactions were assessed 14 days after inoculation. We used a 0 to 2 disease index scale similar to that used by Terry-Lewandowski and Stimart (1983): 0 = no symptoms on leaves or stems; 1 = small necrotic flecks (<2 mm in diameter) on leaves and stems but no sporulation; and 2 = large, sporulating lesions (≥2 mm in diameter) on leaves and stems. Plants with a disease index value ≤1 were scored as resistant, and those with a value >1 were scored as susceptible.

Reactions to Erysiphe cichoracearum *(Expt. 2).* Seeds were sown on 1 June 1988. Four-week-old seedlings were planted in a field plot consisting of a coarse, loamy, mixed mesic Typic Fragiochrept. The 13 entries were arranged in a randomized complete-block design with two blocks and one plot per block. Each plot contained 20 seedlings (four rows with five seedlings per row). Plants were spaced 30 cm apart.

Plants of *Z. violacea* severely infected with *E. cichoracearum* were harvested from the glasshouse. Field-grown plants were inoculated twice (4 and 6 Sept.) by shaking the infected plants over the canopy to release conidia. Disease reactions were assessed 21 days after the second inoculation. Plants with abundantly sporulating mildew colonies on the leaves and stems were scored as susceptible, and those devoid of mildew colonies were scored as resistant.

Reactions to Xanthomonas campestris *pv.* zinniae *(Expt. 3)*. Seeds were sown on 17 Apr. 1989. Five-week-old seedlings were planted in a 1.1 × 14.0-m glasshouse raised bed that had been steam-pasteurized (≈80 °C for 30 min) 4 days earlier. The 13 entries were arranged in a randomized complete-block design with three blocks and one plot per block. Each plot consisted of 10 plants (two rows with five plants per row). Rows were spaced 30 cm apart and plants were spaced 15 cm apart within rows.

Xanthomonas campestris pv. *zinniae* was recovered from diseased *Z. violacea* plants acquired locally. The bacterium was identified using standard morphological, biochemical, and physiological tests (Schaad, 1988). Pathogenicity was confirmed by inoculating *Z. violacea* plants with the bacterium. Bacteria were grown on sucrose-peptone agar (Lelliott and Stead, 1987) and were incubated for 48 h in a growth chamber kept at 27 ± 1 °C. Inoculum was prepared by flooding pure cultures of *X. campestris* pv. *zinniae* with sterile distilled water. The final concentration of the inoculum (determined by dilution-plating) was ≈1.8×10^8 cfu/mL. On 29 May, inoculum was applied to the upper leaf surfaces of each plant using a pressurized hand sprayer. A moist chamber was created by covering the ground bed with a sheet of white polyethylene, as described in Expt. 1. Plants remained in the moist chamber from 3 h before until 24 h after inoculation (Jones and Strider, 1979). Plants were misted at 3-h intervals to maintain a high (>95%) RH in the chamber.

Disease reactions were assessed 21 days after inoculation. The two leaves at the fourth node from the base were collected from the main stem of each plant, and the number of necrotic lesions with chlorotic margins was recorded. Leaf area differed significantly among the entries (Boyle, 1996), and to correct for these differences, the number of lesions per 10 cm^2 of leaf area was determined for each plant and entry. Leaf area was measured with an area meter (model LI-3000A; LI-COR). The percentage of plants showing disease symptoms (necrotic lesions) was calculated for each entry.

Results and Discussion

Experiment 1. During the 14 days from inoculation to disease assessment, glasshouse air averaged 19.0 ± 1.4 °C at night and 23.6 ± 2.9 °C during the day, and ranged from 17 to 33 °C. Entries varied in their responses to *A. zinniae* (Table 2). All three *Z. violacea* entries were susceptible to the pathogen. Inoculated plants of *Z. violacea* formed large, sporulating leaf lesions that frequently coalesced into necrotic patches, and stem lesions often girdled the stems, causing shoot tip dieback. Entries of *Z. angustifolia*, *Z. marylandica*, and the BC_1 hybrids contained from 93% to 100% resistant plants (Table 2). Resistant plants produced small, nonsporulating lesions on the leaves and stems, or were devoid of lesions.

One plant of *Z. angustifolia* 'Classic' developed several large, sporulating leaf lesions and was classified as susceptible. Spores were collected and recultured on carrot agar (as described previously), which confirmed the presence of *A. zinniae*. Neither Lipschutz (1965) nor Terry-Lewandowski and Stimart (1983) reported any cases of susceptibility to *A. zinniae* in *Z. angustifolia*. Our results demonstrate that *Z. angustifolia* 'Classic' is segregating for resistance to alternaria blight. Commercial *Z. angustifolia* cultivars, such as 'Classic', are open-pollinated and consist of highly self-incompatible genotypes (Boyle and Stimart, 1986). Although phenotypic variation within cultivars is limited, considerable genetic variation would be expected to occur within cultivars due to allogamy. Previous studies (Boyle et al., 1987; Boyle and Stimart, 1989a) have shown that *Z. angustifolia* cultivars also segregate for genes affecting seedling emergence, plant morphology, and flowering.

The two BC_1 entries derived from crossing *Z. marylandica* with *Z. violacea* yielded 93% to 100% resistant plants (Table 2). Two plants in one BC_1 family (M1C x V86C) formed large, sporulating leaf and stem lesions and were classified as susceptible; all other plants in these two BC_1 families were classified as resistant. These results suggest that one *Z. angustifolia* genome in BC_1 allotetraploids (AVVV) is sufficient to confer a high level of resistance to *A. zinniae*.

Primary inoculum of *A. zinniae* comes from infected zinnia seed or infested soil (Baker and Davis, 1950; Dimock and Osborn, 1943; Neergaard, 1945). Current control procedures include treating zinnia seeds with hot water (Baker and Davis, 1950), disinfesting the growing medium using steam or chemical fumigants, and applying chemical eradicants. However, these practices increase production costs and, in some cases, pose health risks. Genetic resistance to *A. zinnia* offers a cost-effective, safe alternative to the control methods used currently.

Experiment 2. Entries were either 100% resistant or 100% susceptible to *E. cichoracearum* (Table 2). The three *Z. violacea* entries were highly susceptible to powdery mildew and contained numerous, sporulating mildew colonies on the leaves and stems. In contrast, the *Z. angustifolia*, *Z. marylandica*, and BC_1 entries were devoid of mildew colonies on all plant parts. The responses we observed when *Z. angustifolia*, *Z. marylandica*, and *Z. violacea* were inoculated with *E. cichoracearum* are essentially identical with those reported by Terry-Lewandowski and Stimart (1983).

Erysiphe cichoracearum has a wide host range, and the strain that infects zinnias also attacks many other Compositae species (Schmitt, 1955). Although Baker and Locke (1946) demonstrated that seed transmission of

Table 2. Disease reactions of *Zinnia angustifolia*, *Z. violacea*, and their F_1 and BC_1 hybrids to *Alternaria zinniae* (alternaria blight), *Erysiphe cichoracearum* (powdery mildew), and *Xanthomonas campestris* pv. *zinniae* (bacterial leaf and flower spot).

		A. zinniae		*E. cichoracearum*	*X. campestris* pv. *zinniae*	
Entry	Genomic formula[z]	Disease index[y]	Resistant plants (%)[x]	Resistant plants (%)[w]	No. lesions/ 10 cm² leaf area[v]	Symptomless plants (%)
			Z. angustifolia			
Classic	AA	0.6 ± 0.6	97	100	0.0 ± 0.0	100
Linearis White	AA	0.8 ± 0.4	100	100	0.0 ± 0.0	100
A88C	AAAA	0.8 ± 0.4	100	100	0.0 ± 0.0	100
			Z. violacea			
Canary Bird	VV	2.0 ± 0.0	0	0	6.0 ± 4.1	0
Thumbelina Mini-Pink	VV	2.0 ± 0.0	0	0	4.4 ± 2.9	0
V86C	VVVV	2.0 ± 0.0	0	0	5.4 ± 3.0	0
			Z. marylandica			
Rose Pinwheel	AAVV	0.9 ± 0.4	100	100	0.3 ± 0.6	64
M1C	AAVV	1.0 ± 0.0	100	100	0.1 ± 0.2	70
M2C	AAVV	0.7 ± 0.5	100	100	0.2 ± 0.6	63
			Z. marylandica x Z. angustifolia *(BC_1)*			
M1C x A88C	AAAV	---	---	100	0.1 ± 0.3	96
M2C x A88C	AAAV	0.6 ± 0.5	100	100	0.1 ± 0.1	90
			Z. marylandica x Z. violacea *(BC_1)*			
M1C x V86C	AVVV	0.8 ± 0.4	100	100	7.0 ± 6.1	0
M2C x V86C	AVVV	0.8 ± 0.6	93	100	5.0 ± 3.0	0

[z]A = *Z. angustifolia* (*n* = 11); V = *Z. violacea* (*n* = 12).
[y]Mean ± SD. Disease index: 0 = no symptoms on leaves or stems; 1 = small necrotic flecks (<2 mm in diameter) on leaves and stems but no sporulation; and 2 = large, sporulating lesions (≥2 mm in diameter) on leaves and stems.
[x]The percentage of plants within each entry having a disease index of 0 or 1.
[w]The percentage of plants within each entry that were devoid of mildew colonies on leaves and stems.
[v]Mean ± SD. Lesions were counted on two leaves at the fourth node from the base of the main stem.

cleistothecia was possible, primary inoculum of *E. cichoracearum* for zinnia infections is likely to come from wild or cultivated Compositae growing nearby. Currently, the only practical method for controlling powdery mildew on zinnias is by applying chemical eradicants. Our data show that the levels of resistance present in *Z. angustifolia*, *Z. marylandica*, and the BC_1 entries are effective in controlling powdery mildew (Table 2). These results suggest that presence of one *Z. angustifolia* genome in BC_1 allotetraploids (AVVV) is enough to confer resistance to *E. cichoracearum*.

Experiment 3. During the 21 days from inoculation to disease assessment, glasshouse air averaged 18.9 ± 1.2 °C at night and 22.9 ± 2.5 °C during the day, and ranged from 15 to 33 °C. The entries differed in the number of lesions per 10 cm² leaf area and the percentage of symptomless plants (Table 2). Lesions were absent on all plants of the three *Z. angustifolia* entries (Table 2). The percentage of plants devoid of disease symptoms ranged from 64% to 96% in the three *Z. marylandica* lines and the two BC_1 families derived from *Z. angustifolia* A88C (AAAV). All of the plants in the three *Z. violacea* accessions and two BC_1 families derived from *Z. violacea* V86C (AVVV) exhibited disease symptoms, averaging 4.4 to 7.0 lesions per 10 cm² leaf area (Table 2). The disease responses reported here for *Z. angustifolia* and *Z. violacea* agree with those reported previously (Jones and Strider, 1979; Terry-Lewandowski and Stimart, 1983).

The results (Table 2) demonstrate the effect of gene dosage on resistance of tetraploid zinnias to *X. campestris* pv. *zinniae*. The autotetraploid line of *Z. angustifolia* (AAAA) was completely resistant, BC_1 allotetraploids with three *Z. angustifolia* genomes (AAAV) and F_1 allotetraploids with two *Z. angustifolia* genomes (AAVV) were highly resistant, and BC_1 allotetraploids with one *Z. angustifolia* genome (AVVV) and autotetraploid *Z. violacea* (VVVV) were moderately to highly susceptible to bacterial leaf and flower spot. Thus, a minimum of two *Z. angustifolia* genomes is necessary to confer a high level of resistance to *X. campestris* pv. *zinniae* in tetraploid zinnias.

Bacterial leaf and flower spot of zinnia was first reported in North America in 1972 (Sleesman et al., 1973) and has since become a major disease of *Z. violacea*. Primary inoculum of *X. campestris* pv. *zinniae* is chiefly or exclusively from infected seed (Strider, 1979a). Presence of a few infected seed in a seed package may yield >90% infected seedlings due to preemergence spread of the pathogen (Strider, 1979a). These conditions can result in substantial plant losses for bedding plant growers. It is possible to control this disease with seed treatments (Strider, 1979b, 1980), but disease control is difficult once seedlings or mature plants become infected. Hence, genetic resistance to this pathogen is highly desirable.

Zinnia angustifolia manifests high levels of resistance to *A. zinniae*, *E. cichoracearum*, and *X. campestris* pv. *zinniae* (Table 2), but present-day cultivars of this species do not exhibit the diversity in flower size and ray floret colors displayed by *Z. violacea* cultivars (Boyle and Stimart, 1989b). BC_1 hybrids of *Z. marylandica* and tetraploid *Z. violacea* (AVVV) have larger capitula, more ray florets, and a greater diversity of floral pigments than either *Z. angustifolia* or *Z. marylandica* (Boyle, 1996), and are nearly as resistant as *Z. angustifolia* to *A. zinniae* and *E. cichoracearum* (Table 2). Hence, in the BC_1 generation, it has been possible to combine the disease resistance of *Z. angustifolia* with many of the ornamental features of *Z. violacea*. Unfortunately, BC_1 hybrids of *Z. marylandica* and tetraploid *Z. violacea* are similar to *Z. violacea* in their reactions to bacterial leaf and flower spot (Table 2). Further breeding will be required to combine high levels of resistance to *X. campestris* pv. *zinniae* with the ornamental characteristics of *Z. violacea*.

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