lower than 100 ppm and IAA, 10 to 100 ppm, were less effective in producing parthenocarpic fruit, and fruit development was relatively slow. GA_3 and GA_{4+7} slowed down the early rate of fruit development as compared with pollinated fruit, but were very effective in accelerating fruit development during later stages, as was BA.

High percentage of flowers abscissed when 4-CPA and β-NOA were applied to the entire plant when the first flower was at anthesis. However, flower buds treated at earlier developmental stages produced parthenocarpic fruits. Cucumber plants treated with β-NOA, showed relatively slow increase in the rate of fruit set.

4-CPA at high rates caused epinasty of plant apices 1 day after application and slowed down plant growth. Growth was renewed later, usually in the shape of abnormally thick and small leaves. There was only a slight evidence of this phenomenon after β-NOA treatment. Treatment with BA caused dark-green leaves, and did not reduce the rate of fruit growth. One day after GA application tendrils straightened out. Later the growth pattern changed from spreading to erect growth, growth rate later accelerated, the nodes became more elongated than usual and the foliage pale green.

Fruit development on treated whole plants resulted in different observations; 4-CPA produced parthenocarpic fruit, while GA was only slightly effective in fruit development (Table 2). Similar results were obtained in experiments performed under conditions of open pollination in the field (Table 3). GA treatment also decreased the number of developing fruits by ca 400% as compared with open pollinated control.

Table 2. The effect of single 4-CPA and GA_3 treatments, applied to whole plants, on the development of parthenocarpic fruit in cucumbers (bees excluded).

Compound	Rate (ppm)	No. of fruits per plant	
		In the field	In net enclosures
4-CPA	200	3.0 a[z]	3.6 a
	100	2.0 a	3.4 a
	50	2.8 a	2.8 a
GA_3	2,000	0.2 b	0.4 b
	1,000	0.0 c	0.0 c
	500	0.2 b	0.4 b
Unpollinated control		0.0 c	0.0 c

[z]Mean separation by Student-Neuman-Keul's range test, 5% level.

The nature of the endogenous growth regulators active at various stages of fruit development is not well understood (1, 3). Nitsch (3), however, emphasized that natural parthenocarpy is characterized by the development of seed coats and nucellus. In the present study, seed coats were present in all fruits which developed in response to treatments. It is conceivable that seed coats might play a role in the development of parthenocarpic cucumber fruits.

Application of GA to individual flowers and to the entire plant produced contradictory results. In the last case, growth was accelerated and it seemed likely that GA accumulated in the plant apex and served as a metabolic sink. In the first case, however, the ovary itself may serve as a metabolic sink, thus promoting the development of parthenocarpic fruits. Such an hypothesis agrees with current ideas about the importance of site-dependent hormonal activity during fruit development.

Table 3. The effect of growth regulators on fruit development under natural bee pollination in the field (plant population: 20 plants per m^2).

Compound	Rate (ppm)	No. of fruits per m^2
4-CPA	50	41.4 a[z]
β-NOA	100	31.1 a
BA	10	30.7 a
GA_3	500	8.1 b
Control (bee-pollinated)		32.0 a

[z]Mean separation by Student-Neuman-Keul's range test, 5% level.

Literature Cited

1. Crane, J. C. 1969. The role of hormones in fruit set and development. *HortScience* 4:108-111.
2. Jones, C. M. 1965. Effects of benzyladenine on fruit set in muskmelon. *Proc. Amer. Soc. Hort. Sci.* 87:335-340.
3. Nitsch, J. P. 1965. Physiology of flower and fruit development. Encyc. plant physiol. 15(1):1537-1647.
4. Palevitch, D., E. Pressman, and J. Rudich. 1972. Induction of parthenocarpy by triiodobenzoic acid in cucumbers (*Cucumis sativus* L.). *Z. Pflanzenphysiol.* 67:457-459.
5. Quebedaux, B., and E. M. Beyer, Jr. 1972. Chemically induced parthenocarpy in cucumber by a new inhibitor of auxin transport. *HortScience* 7:474-476.
6. Robinson, R. W., D. J. Cantliffe, and S. Shannon. 1971. Morphactin-induced parthenocarpy in cucumber. *Science* 171:1251-1252.
7. Wong, C. Y. 1941. Chemically induced parthenocarpy in certain horticultural plants with special reference to watermelon. *Bot. Gaz.* 103:64-84.

Control of Seed-borne *Botrytis cinerea* (Pers ex Fr.) on *Gerbera jamesonii* Bolus[1]

L. Orlikowski[2], J. Hetman[2], and B. Tjia[3]
University of Kentucky, Lexington

Abstract. Botrytis cinerea was shown to be seed-borne on gerbera seeds. Inoculation of seeds increased damping-off of seedlings from 5 to 90%. Benlate or Thiram at a concentration of 0.1% applied as a dip for 1, 5 or 10 minutes eliminated or reduced the post-germination mortality of seedlings.

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[2]Research Specialist, Agricultural Academy, Lublin, Poland.

[3]Assistant Professor, Department of Horticulture, University of Kentucky, Lexington, Kentucky 40506.

Gerbera, at all stages, is very susceptible to *Botrytis cinerea* infection. The fungus invades plants from infested soil or from other nearby diseased plant parts causing significant losses in greenhouse and field production (2). The most serious aspect of the disease is crown rot (7). From the crown tissue the pathogen invades the roots and leaves. Affected parts of plants turn brown or dark brown. The fungus produces conidia on the surface of infected leaves and other plant parts. Flowers are also susceptible. Following infection, the florets develop very small black spots (4) or slightly slender dark lesions on petioles (7). Conidia can spread easily to the seeds from infected organs.

The use of fungicide for protection of seeds against *Botrytis cinerea* was successfully shown by Belcher et al. (1) and Volger (8) on conifer seedlings. Some fungicides were evaluated on gerbera but only on growing plants. Garthwaite (2, 3) used N-(trichloromethylthio)-4-cyclohexene-1,2 dicarboximide (Captan) to protect gerbera cuttings, but the results were not successful. Van Doesburg (7) used 0.5% (N-dichlorofluoromethyl-thio-N,N-dimethyl-N-phenyl sulfamide (Euparen) to protect gerbera flowers, but florets were scorched. The objectives of this investigation were to evaluate the magnitude of seed transmission by *Botrytis cinerea;* to determine pathogenicity of the fungus to gerbera seeds and seedlings; and to determine

the effectiveness of fungicides for protection.

The methods described by Lacicowa (5) were essentially followed. Gerbera seeds were placed on petri dishes containing malt agar. All fungi growing from the seeds were transferred to PDA medium and identified. Isolations were made from different sources of seeds over a 3-year period.

Botrytis cinera cultures isolated from seeds were further used for pathogenicity tests. Ten surface-sterilized seeds were plated on sterilized petri dishes containing 5 layers of filter paper. Each seed was inoculated with 1 ml sterilized distilled water containing 600 conidia. Control plates were treated with 10 ml of sterilized distilled water only. Treatments were replicated 5 times. Plates were incubated at 22 ± 1°C. Seeds were checked for germination at the end of 5 days. Disease observations, rated as to the degree of severity were made after 10 days.

Two fungicides were evaluated: methyl 1-butylcarbamoyl-2 benzimidazole carbamate (Benlate) at 0.025, 0.05 and 0.1% and tetramethyl thiram disulfide (Thiram) at 0.1, 0.2 and 0.3%. We dipped 200 seeds in each fungicide suspension for 1, 5 and 10 min. After chemical treatments, seeds were plated on petri dishes containing 10 layers of filter paper and 15 ml of distilled water. Four replications were used. The plates were incubated at 22 ± 1°C. Germination and Botrytis infection were evaluated after 5 days.

The results obtained over 3 years of isolations indicated that about 7, 6 and 9 of gerbera seeds were infected with *Botrytis cinerea* (Table 1). Since 100 of each seed were examined each year, this amounts to an average of 7% Botrytis infection. The fungus did not inhibit seed germination (Table 2) but adversely affected the growth of the young seedlings. The mortality rate of inoculated seedlings was 89.5%. The pathogen caused post-germination damage to roots and/or cotyledons. Infected seedlings died within a few days. Therefore, we assume that seeds not treated with fungicides can be associated with the spread of gray mold fungus on germinating seedlings, as our results have indicated. In the actual production of gerbera, seeds are grown under 20 – 24°C and 70% relative humidity or more, and this provides optimum environmental conditions for germination as well as Botrytis infection.

Thiram and Benlate dips gave satisfactory control of Botrytis infection on gerbera seedlings, although complete control on all treatments was not found. Benlate seemed to give better results than Thiram, however statistical analysis did not give significantly better results with Benlate than Thiram (Table 3). Benlate at 0.1% gave also no more protection than Benlate at 0.05 or 0.025% in terms of significant differences. Therefore, Benlate (0.025 – 1%) as well as Thiram (0.1 – 3%) can be used as effectively to protect gerbera seeds from Botrytis.

Table 1. Isolation of fungi from commercial gerbera seeds.

Year[z]	All fungus[y] isolates (colonies)	*Botrytis cinerea* isolates No.	*Botrytis cinerea* isolates %	No. of seeds contaminated with *Botrytis cinerea*
1970	93	9	9.7	7
1971	114	16	14.0	6
1972	109	11	10.0	9

[z]3 lots each year.
[y]mean of 100 seeds.

Table 2. Effect of *Botrytis cinerea* infection on germination and growth of gerbera seedlings.

Seed treatment	Germination (%)	Seedling mortality (%)
Noninoculated	80a[z]	5.0a
Inoculated	78a	89.5b

[z]Mean separation in columns by Duncan multiple range test, 1% level.

Table 3. Effect of chemical dips on germination and seedling infection of gerbera.[z]

Fungicides	Concn (%)	Germination[y] 1 min	5 min	10 min	Avg infected seedlings
Control		178a	188a	182a	12a
Benlate	0.025	182a	182a	186a	2b
	0.050	184a	186a	182a	2b
	0.100	185a	181a	187a	0b
Thiram	0.1	188a	191a	193a	4b
	0.2	184a	185a	184a	2b
	0.3	189a	189a	189a	2b

[z]Mean separation in columns by Duncan's multiple range test, 5% level.
[y]From 200 seeds.

Literature Cited

1. Belcher, J., and L. W. Carlson. 1968. Seed-treatment fungicides for control of conifer damping-off. Laboratory and greenhouse tests, 1961. *Can. Plant Dis. Surv.* 48:47-52.
2. Garthwaite, J. M. 1960. Gerbera propagation. *Expt. Hort.* 3:65-68.
3. ———, ———. 1962. Culture of gerbera and the avoidance of Botrytis. *Expt. Hort.* 8:114.
4. Garibaldi, A. 1968. Uira incosueta alterazione delle infiorescenze di gerbera. *Phytopath. Medit.* 7:91-92.
5. Lacicowa, B. 1969. Metoda laboratoryjna pozwalajacana szybka ocene odpornosci jeczmienia na H. sativum P. *K et. B. Biluletyn IHAR 3-4.*
6. McLean, N. A. 1949. New host for *Botrytis cinerea* and *B. elliptica* in the Pacific Northwest. *Phytopathology* 39:949-950.
7. Van Doesburg, J. 1967. Proefstation voor de bloemistery in Nederland te Aalsmeer. *Jaarverslag.* 1966.
8. Volger, C. 1959. Ein systemischer effect des fungiziden wirkstoffes TMTD. *Naturwiss* 46:148-149.

A Nutritional Disorder of Red Oak Seedlings[1]

P. Fenn and R. D. Durbin[2]
University of Wisconsin, Madison

Abstract. **Seedlings of red oak (*Quercus rubra* L.) grown in various planting media without nutritional supplements developed marginal necrosis and puckering on their first whorl of leaves. These symptoms were prevented by supplying Ca++ to seedlings growing in a vermiculite-peatmoss mixture. An exogenous supply of Ca++ was also required for initial growth in silica sand.**

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[2]Research Assistant, Professor and Laboratory Leader, respectively, Department of Plant Pathology, University of Wisconsin and Agricultural Research Service, U.S. Department of Agriculture, Madison, Wisconsin.

The initial growth of red oak seedlings is thought to depend largely on cotyledonary reserves thus making them essentially independent of the external nutritional environment (4, 5). However we have observed a nutritional disorder in the early development of red oak seedlings grown in various planting media. The abnormality was first noticed on seedlings grown in a crossed gradient controlled environment room (University of Wisconsin Biotron) where 16 light and temperature environments were simultaneously maintained. Temperature ranged from 17 – 30°C