Fusarium Wilt of Chrysanthemum: Effect of Nitrogen Source and Lime on Disease Development

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ABSTRACT

Nitrate-nitrogen decreased the severity of Fusarium wilt of Yellow Delaware chrysanthemum in comparison with half ammonium, half nitrate-nitrogen. Liming also reduced the severity of wilt. Nitrate-nitrogen together with calcium hydroxide decreased wilt additively. The

severity of disease generally decreased with increasing soil pH. A visual disease rating system correlated well with root and shoot growth and discoloration of vascular bundles and root systems.

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Fusarium wilt of chrysanthemum has been shown to be caused by two formae speciales, each of which attacks specific cultivars (2, 3, 4, 5, 6, 8). Symptomatology and disease reactions were described for 14 chrysanthemum cultivars in response to inoculation with the two recognized formae speciales as well as for several wild isolates from the cultivar Yellow Delaware (5). Specifically, Fusarium oxysporum (Schlecht.) f. sp. tracheiphilum (E. F. Sm.) Snyd. & Hans. race 1 Armst. & Armst. incites disease of Encore but not of Yellow Delaware (4, 5). Fusarium oxysporum (Schlecht.) f. sp. chrysanthemi Litt., Armst., & Armst. causes wilt of Yellow Delaware and Encore (4).

Higher levels of nitrogen were shown to cause greater disease severity than lower levels (6). Severity of Fusarium wilt in other host plants was decreased by nitrate-nitrogen in comparison with ammonium (1), and by liming (7). Applications of micronutrients, especially zinc and certain combinations of iron, maganese, and zinc were found (7, 9) to increase the severity of Fusarium wilt of tomato. In vitro growth and sporulation of tomato wilt Fusarium was reported to be dependent on adequate supplies of manganese and zinc (9).

This investigation was undertaken to find cultural control procedures for Fusarium wilt of chrysanthemum. Since the disease problem has been most severe with Yellow Delaware, this cultivar and wild isolates of the pathogen from the cultivar were used in the study.

Yellow Delaware rooted cuttings were planted, four/6-inch plastic pot containing a growing medium composed of 75% virgin Leon fine sand and 25% shredded peat by volume. Liming materials were thoroughly incorporated in the growing medium and allowed to equilibrate in a moist condition for 2 weeks prior to planting. Two liming regimes were employed, namely, 1 g CaCO₃/kg growing medium and 1 g CaCO₃ + 2 g Ca(OH)₂/kg. Plants were irrigated as required with well water, pH 7.5, and a total salts content of 770 ppm. Pots were watered twice weekly with 100 ml of a nutrient solution made up with either 3.2 g NaNO₃ or 1.5 g NH₄NO₃ as the nitrogen source and 1.0 g NaH₂PO₄·H₂O₅ 0.85 g

KC1 and 0.2 g MgSO₄ ·7H₂O dissolved in deionized water and diluted to 1 liter. Plants were inoculated 2 weeks after the transplanting into pots, at which time they were well established and had developed good root systems. Roots of the four plants were cut 1 inch from stems 2 inches deep. Two trenches were made across the pot in one direction and two other trenches were made by cutting at right angles. Fifty ml of suspension containing 6 X 10⁷ microconidia were poured into the trenches in each inoculated pot; 50 ml water were poured into each noninoculated pot. The inoculum was produced from a composite culture of three wild isolates of Fusarium (f. sp. unidentified) from diseased Yellow Delaware plants. Pathogenicity was established for each isolate by root-dip inoculation of Yellow Delaware. The isolates were grown on nutrient agar, using galactose as the carbon source. We prepared a spore suspension by washing each petri plate with 5 ml sterile water and blending combined washing water briefly to prepare a uniform suspension.

Pots were held in a pad-cooled greenhouse 33 ± 2 C daily maxima and 25 ± 2 C minima. Disease index ratings were made at 16 days and each week thereafter for 3 subsequent weeks. A rating procedure was adopted as follows: 0 = no symptoms; 1 = characteristic chlorosis; 2 = chlorosis plus curvature of leaf or stem; 3 = chlorosis and curvature plus wilt; 4 = chlorosis, curvature, and wilt plus obvious stunting; and 5 = dead plant.

Plants received long day photoperiods and were in a vegetative condition at the conclusion of the experiment 9 weeks after planting. Fresh weights were recorded for roots recovered by a standard washing procedure using a strong stream of water. Root discoloration was rated on a 0 to 5 scale where 0 = normal light color of roots of noninoculated control plants and 5 = dark brown color of the most severely diseased roots. Vascular browning of the stem 2-3 cm above the root crown was rated on a 0 to 5 scale where 0 = normal white-green coloration; 1 = very light brown or tan; 2 and 3 = increasing degrees of brown coloration; 4 = dark brown; and 5 = very dark brown, found in dead plants.

In this experiment, as in others (5), disease

TABLE 1. Effect of nitrogen source and liming on soil pH and the severity
of Fusarium wilt disease in Yellow Delaware chrysanthemum

N source	Liming material ^a (g/kg soil)	Final soil pH	Vascular browning ^b	Root browning ^b	Fresh wt, g	
					Shoots	Roots
NaNO	1	5.9	3.7	3.4	21	16
NaNO ₃	3	7.3	2.7	2.7	51	37
NaNO ₃	1	4.9	5.0	4.9	13	9
NH ₄ NO ₃ NH ₄ NO ₃	3	6.3	4.7	4.7	25	14
NaNO ₃ Noninoculated	1	6.1	0.0	0.0	70	23
LSD, 5% level			0.8	0.4	13	4

 $a_1 = 1 \text{ g CaCO}_3$; $3 = 1 \text{ g CaCO}_3 + 2 \text{ g Ca(OH)}_2$.

b Browning rated on a 0 to 5 scale where 0 = normal color and 5 = maximum browning.

developed rapidly and uniformly following root inoculation, with soil conditions that favored the development of Fusarium wilt. The progression of development of the wilt syndrome was as follows: 7 to 10 days after inoculation, upper leaves developed chlorosis and externally visible vein discoloration that usually affected only parts of leaves and one side of the plant. Vascular browning was apparent in the petioles of detached leaves showing the early symptoms of chlorosis. Areas of leaves affected were

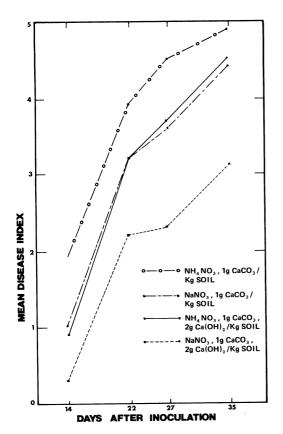


Fig. 1. Effect of N-source and lime on severity of r'usarium wilt in chrysanthemum.

uniformly chlorotic in a manner distinguishable from nutritional symptoms, because of the very rapid development of chlorosis in a few of the upper leaves, affecting only limited areas of individual leaves. Leaves and stems commonly exhibited a curvature toward the initially diseased portion of the plant. Wilting developed later, followed by a noticeable stunting. Death of most plants occurred within 4 weeks after inoculation under the soil conditions most favorable to disease development.

Soil pH was raised by NaNO₃ (Table 1) as well as by the greater amount of liming material provided by CaCO₃ plus Ca(OH)₂. Ammonium nitrate lowered the pH in contrast to NaNO₃. The NaNO₃ treatment with 1 g CaCO₃ was selected as the noninoculated control for the experiment reported. Results from other experiments conducted in a similar manner, but not reported here, indicated that normal and fairly equal growth could be obtained with the treatments in Table 1. High soil pH did, however, necessitate foliar applications of micronutrients, particularly of iron. Micronutrient deficiencies were avoided in the present experiment by foliar sprays applied as required. Vascular browning and root browning were inversely related to soil pH. Where NH₄NO₃ was used with the high level of lime, a greater degree of browning occurred independent of pH effects. All NO₃-N and the high level of lime decreased disease severity singly and additively based on the data on vascular and root browning, weight of shoots and roots, and disease index (Table 1, Fig. 1). Nitrogen derived from NH4NO3 and the low level of lime amendment increased disease severity singly and additively on the basis of browning, weight of plant parts, and disease index ratings. The progression of disease development responded consistently to varied N-source and levels of lime with the passage of time (Fig. 1). The same relationship of effects existed for disease ratings over a period of 35 days.

The nutrition, physiology, and pathogenicity-resistance patterns of the pathogen and host are probably altered in a complex manner by varying N source and liming procedures. Although the literature (1, 7, 9) suggests several explanatory mechanisms that are likely to be involved, a clear exposition of the cause-effect situation will probably

await additional research conducted so as to separate the many factors involved.

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