

Effects of the Host, the Pathogen, the Environment and Their Interactions, on Fusarium Wilt in Carnation

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Fusarium oxysporum f.sp. *dianthi* causes severe wilting in carnation (*Dianthus caryophyllus* L.) worldwide. The pathogen is present in the soil profile in which carnation roots are distributed and may infect the plants at any time during the growing season. To minimize the losses induced by Fusarium wilt, growers use carnation cuttings free of *Fusarium* spp. and fumigate the soil with methyl bromide prior to planting. The severity of epidemics and the resulting losses are governed by the main and interacting effects of the three components of the disease syndrome: the host, the pathogen and the environment. Host variables include the type and the degree of cultivar resistance (*i.e.*, complete, partial or tolerance); pathogen variables include the race, its virulence and infectivity, and the amount of initial inoculum; environmental variables include solar radiation intensity, photoperiod, temperature and the growth substrate. In the present review the information available on the effect of the host, the pathogen and the environment, and their interactions, on Fusarium wilt in carnation is summarized.

KEYWORDS: *Fusarium oxysporum* f.sp. *dianthi*, photoperiod; growth substrate; solar radiation; epidemiology.

INTRODUCTION

Fusarium oxysporum Schlechtend.:Fr. f.sp. *dianthi* (Prill. & Delacr.) W.C. Snyder & H.N. Hans. causes severe wilting in carnation (*Dianthus caryophyllus* L.) worldwide. The pathogen is present in the soil profile in which carnation roots are distributed and may infect the plants at any time during the growing season. However, the disease is observed when the conditions are favorable for the expression of symptoms. To minimize the losses induced by Fusarium wilt, growers use carnation cuttings free of *Fusarium* spp. (47) and fumigate the soil with methyl bromide prior to planting (10,52). However, in greenhouses where susceptible cultivars of carnations have been grown as monoculture for more than 10–15 years, these practices are not always enough to prevent Fusarium-induced losses. Another means of disease management is to use cultivars resistant to the pathogen. A number of studies have shown that the inheritance of resistance to *F.o.* f.sp. *dianthi* is partial, and relies on a complex system of host reactions (3,22,26,42,48). In partial resistance, a wide range of responses among cultivars is apparent, from highly susceptible to highly resistant (20). Because the highly resistant cultivars currently available in the market have a lower yield or are commercially undesirable, growers prefer to plant susceptible cultivars whenever possible. In greenhouses where severe Fusarium wilt

Contribution from the Agricultural Research Organization. No. 1937-E, 1996 series. Received Dec. 19, 1996; received in final form April 30, 1997.

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occurred during the previous season, growers are advised to plant only cultivars with high resistance, even though they are less profitable (20).

The severity of epidemics and the resulting losses are governed by the main and interacting effects of the three components of the disease syndrome: the host, the pathogen and the environment – both biotic and abiotic. Host variables include the type and degree of cultivar resistance (*i.e.*, complete, partial or tolerance); pathogen variables include the race, its virulence and infectivity, and the amount of initial inoculum; environmental variables include solar radiation intensity, photoperiod, temperature and the growth substrate. In the present review we summarize the information available on the effect of the host, the pathogen and the environment – primarily the abiotic, and their interactions, on *Fusarium* wilt in carnation. We focused mainly on wilt response of new cultivars following natural or artificial inoculations.

THE HOST

In hosts where complete resistance to wilt pathogens exists, *e.g.* resistance of tomato to *F. oxysporum* f.sp. *lycopersici* or to *Verticillium dahliae* (2,12,27,38), the most important factor influencing wilt development is, obviously, the reaction of the cultivar to the pathogen. Following inoculations, resistant cultivars remain healthy whereas susceptible ones become diseased. The severity of wilt in the susceptible cultivars is governed by the pathogen and the environment.

In carnation, resistance to *F.o.* f.sp. *dianthi* race 2 is partial (3,9,22,42). Due to the role of the pathogen and the environment on disease expression, contradictory conclusions may be derived from tests conducted under different conditions. For example, Harling *et al.* (35) inoculated six commercial cultivars with different backgrounds of resistance to *F.o.* f.sp. *dianthi* at two temperatures, 22° and 26°C. At 22°C the cultivars were clearly differentiated into three groups (highly resistant, moderately resistant, and susceptible) based on the severity of the symptoms; however, this differentiation was not apparent at 26°C, at which temperature most of the cultivars exhibited a susceptible reaction. The inoculation technique may also affect the outcome severity. Several methods have been proposed for artificial inoculation of carnation cultivars with *F.o.* f.sp. *dianthi*. These include root-dip inoculation in a spore suspension (37), stem inoculation with a spore suspension (8), and inoculation of the substrate with spores before (49,50) or after (48) planting. However, wilt response following artificial inoculations in greenhouses does not always coincide with the plant response in the field (20). Ben-Yephet *et al.* (15) classified cultivar response in artificially inoculated plants in the greenhouse and compared the outcome with that of a test conducted in a naturally infested field. Similarity in cultivar response between the tests was found only in the highly susceptible cultivars. For cultivars with some degree of resistance, results of the artificial inoculation test did not coincide with those of the field test (Table 1). No further increase in disease incidence in inoculated plants of various cultivars was observed when the growth period was extended up to 180 days after planting. The reason for this inconsistency is probably due to the differences in environmental conditions and the inoculation procedures among the pathogenicity tests.

TABLE 1. Wilt reactions of 17 cultivars in greenhouse and field tests^z

| Cultivar | Disease incidence (%) | |
|----------------|-----------------------|------------|
| | Greenhouse test | Field test |
| Fantasia | 100 | 99 |
| Raggio di sole | 95 | 88 |
| Lior | 9 | 90 |
| Galit | 0 | 41 |
| Pallas | 0 | 13 |
| Eveline | 0 | 7 |
| Libnatt | 100 | 97 |
| Alpinia | 100 | 89 |
| Orili | 100 | 80 |
| Spring Tween | 100 | 88 |
| Wily | 67 | 72 |
| Amir | 42 | 61 |
| Dona | 25 | 47 |
| Aviv | 0 | 67 |
| Golden Queen | 0 | 81 |
| Mark Queen | 0 | 34 |
| Pink Tween | 0 | 28 |

^zIn the greenhouse with diffusible solar light $ca\ 500\ \mu E\ m^{-2}\ s^{-1}$, and temperature ranging from 23° (minimum) to 30°C (maximum), plants were artificially inoculated and planted in 3-l pots filled with steamed tuff (crushed volcanic stones). Disease was recorded 60 days after planting. In the field, plants were planted in late June in naturally infested soil and disease was recorded after 180 days (15).

The effects of cultivar resistance on the incidence of *Fusarium* wilt and the population densities of *F.o. f.sp. dianthi* on carnation and in the soil were studied recently in the field (18). In general, population densities of the pathogen on plant stems were linearly related to disease incidence: the more severe the disease, the more colony-forming units (cfu) of *F.o. f.sp. dianthi* were recovered. A similar relationship was observed in soil sampled from beneath plants, except that the increase in cfu was not linear and the pathogen cfu leveled off gradually at high disease incidence. The pathogen was also recovered from symptomless plants (18). In several studies of the defense mechanism to *Fusarium* wilt it was observed that resistance of carnations to infection is characterized by a localization mechanism involving vascular occlusion by brown gums. The involvement of lignification and suberization in the localization was suggested as an important role in the mechanism of resistance in carnation to *Fusarium* wilt. In resistant cultivars the infection is quickly localized and fungal growth remains low (4,5,6,7,34). Nevertheless, pathogen existence in symptomless plants may play a substantial role in the epidemiology of the disease, because the existence of cfu of the pathogen in these plants may provide inoculum for the following season.

THE PATHOGEN

Among the eight known races of *F.o. f.sp. dianthi* (32), race 2 is the most prevalent worldwide (1,14,32,40). The role of the amount of initial inoculum in wilt has been well demonstrated in many pathosystems. Most studies involved susceptible hosts and

artificial inoculation (21,28,36,41,43,46). Generally, increasing the amount of initial inoculum enhances disease severity and reduces the time required for maximal disease development. Root-dip and stem-wound inoculations with *F.o. f.sp. dianthi* induced wilt symptoms in carnation more quickly than did inoculation by infestation of the soil (11,37). This probably was due to the immediate penetration of spores into the xylem following root-dip inoculation, as compared with penetration occurring following root infections in field-grown plants. In another study, Ben-Yephet and Shtienberg (19) reported that under optimal conditions of temperature and radiation intensity, increasing the inoculum concentration of *F.o. f.sp. dianthi* (from 10^3 to 10^6 spores/ml) reduced the time for development of wilt in a highly susceptible carnation cultivar ('Hermon'), but 55 days after inoculation wilt incidence reached 100% even with the lowest inoculum level used.

The relationship between the amount of initial inoculum and the resulting wilt was recently studied using large containers filled with naturally infested soil (13). Disease incidence increased significantly with increasing initial inoculum levels: for initial *F.o. f.sp. dianthi* inoculum levels of 6, 25, 120, 770 and 3500 cfu/g soil, disease incidence 180 days after planting was 2%, 5%, 13%, 34% and 57%, respectively. Analysis of the linear relationship between inoculum level and Fusarium wilt incidence revealed that wilt symptoms can be incited by an inoculum density lower than 6 cfu/g soil.

THE ENVIRONMENT

Effect of temperature

Wilt induced by *Fusarium* spp. is affected markedly by soil temperature. However, the effect of temperature on stem colonization and wilt occurrence may vary in different pathosystems (23,28,31,33,36,41,46). In carnation, no symptoms and very little colonization were observed at 14–15°C; nearly all stems were colonized at 18–20°C but they remained symptomless; at temperatures of 23–26°C, wilt symptoms were severe (30,35,37). The interval between inoculation and appearance of wilt symptoms decreased progressively with increasing air or soil temperatures between 15 and 32°C (37). In a recent study (19), a parabolic relationship was described between substrate temperature and disease intensity, indicating that there were lower and upper temperature extremes at which symptoms did not develop, and an optimum temperature at which the most severe disease occurred. The optimal temperature was 25–26°C; the upper and lower temperature extremes were influenced by solar radiation intensity and the inoculum concentration used. For example, at 85% shade ($200 \mu\text{E m}^{-2} \text{s}^{-1}$), the lower and upper temperatures at which symptoms did not develop were, respectively, 21.5 and 30.7°C for 10^3 spores/ml, but 18.6 and 33.0°C for 10^6 spores/ml. Harling *et al.* (35) suggested that temperatures alter the balance between the host and the pathogen; temperatures that favor the host metabolism to increase relative to that of the pathogen, would induce a resistant reaction, whereas temperatures that favor the pathogen would induce a susceptible reaction resulting in wilt symptoms.

Photoperiod and solar radiation intensity

Photoperiod and solar radiation intensity were reported to affect wilt induced by several soilborne pathogens in various hosts. In inoculated tomato (38) or potato (25,51) seedlings, symptoms of *Verticillium* spp. developed faster under short-day than under long-day

conditions. Similarly, wilt induced by *V. dahliae* in watermelon (11) was more pronounced at low than at high solar radiation intensities. Recently, Pennypacker *et al.* (44) studied the effect of photosynthetic photon flux density on the pathogenicity of *V. albo-atrum* to susceptible and partially resistant alfalfa clones. They found that pathogenicity on the susceptible clone was not influenced by photosynthetic photon flux density, whereas the other clone lost its resistance with less than 40% photosynthetic photon flux density of the ambient radiation. Ben-Yephet and Shtienberg (19) reported that wilt induced by *F.o. f.sp. dianthi* in a susceptible cultivar of carnations was negatively correlated with solar radiation. Severe epidemics (wilt incidence of 100%) developed under low solar radiation intensities ($200\text{--}300 \mu\text{E m}^{-2} \text{s}^{-1}$), whereas at solar intensities above $1000 \mu\text{E m}^{-2} \text{s}^{-1}$, inoculated plants remained symptomless although they were colonized by the pathogen. The above-ground biomass and stem circumference of plants grown under the low light intensity were smaller than those of plants grown under high solar radiation intensity, but the former were taller. The mechanisms by which photoperiod or solar radiation intensity affect disease severity are not known. Solar radiation affects the rate of net photosynthesis (29). It is more likely that reduction in net photosynthesis predisposes the host to become more susceptible, rather than altering the virulence of the pathogen (44).

The growth substrate

One of the variables that may affect disease severity is the growth substrate. Carnations are grown in most places mainly on natural soils, but growth substrates are used as well. Contradictory results on the effects of the growth substrate on wilt incidence were reported. For example, peat was reported in one study (49) to be suppressive to the pathogen (*i.e.*, it was associated with a mild wilt reaction), but in another study (16) it was found to be conducive (*i.e.*, it induced a severe wilt reaction). The effect of the growth substrate tuff (crushed volcanic stones) or tuff mixed with various proportions of peat on wilt incidence was studied in five carnation cultivars with different degrees of resistance to the pathogen (17). For the highly susceptible cultivar ('Hermon'), severe epidemics developed in all growth substrates and disease incidence reached 100% within approximately 3 weeks after inoculation. However, for the less susceptible cultivars the growth substrate had a very strong influence on disease development. Intermittent or mild epidemics developed in moderately resistant ('Galit') or highly resistant ('Eveline' and 'Candy') cultivars, respectively, when plants were grown in the tuff substrate. However, severe epidemics developed in all cultivars grown in the 1:1 and 1:3 tuff-peat mixture substrates. The mechanism by which peat affects disease severity could be direct (through effects on the host or the pathogen), indirect (*via* its effect on the microbial population in the growth substrate), or both. Regardless of the mechanism of the effect of the growth substrate, this effect emphasizes the complex interaction among variables involved in the Fusarium wilt syndrome in carnations.

INTERACTIONS AMONG THE HOST, THE PATHOGEN AND THE ENVIRONMENT

The individual effect of each variable and the interaction among the variables lead to a complex host-pathogen relationship. Katan (39) reviewed the interacting effects of several biotic and abiotic variables on vascular wilt diseases and demonstrated that a change in one variable may affect any of the other variables, leading to an increase, a decrease, or

to no change in disease severity. Cirulli and Ciccarese (27) studied the effects of several variables on wilt induced by *F.o. f.sp. lycopersici* on tomato. The variables were host resistance (completely and moderately resistant cultivars), seedling age, inoculum density, and temperature. They found that plants with complete resistance were not affected and remained symptomless, regardless of the other variables. However, disease severity in the moderately resistant cultivars was affected by each of the factors examined.

Fig. 1. Effects of substrate temperature and solar radiation intensity on Fusarium wilt in carnation, induced by *Fusarium oxysporum* f.sp. *dianthi*. At values greater (outside) than the depicted curves, symptoms of Fusarium wilt did not develop in carnation plants (cv. 'Hermon') inoculated with spore suspensions of *F.o. f.sp. dianthi* varying from 10^3 to 10^6 spores/ml (19).

Analysis of the interaction of the effects of the host, the pathogen and the environment in the *F.o. f.sp. dianthi* – carnation pathosystem revealed that it may be understood in terms of two general concepts: the law of minimum (24) and compensation (45). Liebig (24) codified the concepts of the law of minimum as follows: "If several factors affecting outcome are present in abundance and one factor is deficient, adding more of the abundance factor will change the outcome little." With respect to the *F.o. f.sp. dianthi* – carnation pathosystem, effects of solar radiation and temperature on wilt incidence were consistent with the law of minimum whenever these variables were extreme (Fig. 1). For example, when temperatures were lower than 18°C or higher than 34°C, plants remained symptomless even when solar radiation intensity was optimal for wilt incidence. Similarly, at high solar radiation intensities, above $1000 \mu\text{E m}^{-2} \text{ s}^{-1}$, plants remained

symptomless even at temperatures optimal for *F.o. f.sp. dianthi* activity. Within the range of these variables, the concepts of compensation rather than the law of minimum are more applicable. Rotem (45) developed the theory and concepts of compensation, which postulate that a highly favorable state of one factor, essential for development of a given phase in the life cycle of a pathogen, can compensate for the limitations imposed by the simultaneously less favorable state of another factor. Compensation was evident in the carnation – *F.o. f.sp. dianthi* system when the abiotic variables were at intermediate levels. The amount of inoculum concentration was a factor in the compensation phenomenon as well (Fig. 1).

The interacting effects of solar radiation intensity, temperature and growth substrate were studied in artificial inoculation experiments (17). Disease incidence and disease severity varied significantly among the experiments (due mainly to differences in temperature), among the solar radiation treatments and among the cultivars tested. The three-way interaction term (*i.e.*, cultivar \times shade treatment \times experiment [temperature]) was highly significant ($P < 0.001$) when both disease incidence and disease severity were considered, indicating that no single variable was predominant in determining disease severity.

CONCLUSIONS

In carnation with partial resistance to *F.o. f.sp. dianthi*, the host – pathogen interactions are affected considerably by the environment. Severity of the induced wilt is governed by the particular set of conditions. This could explain the contradictory results reported from different places regarding cultivar response following artificial inoculations. Moreover, there are differences between the effects of variables on disease severity in the greenhouse and in the field. For example, in the field, disease symptoms developed when solar radiation intensity exceeded $1000 \mu\text{E m}^{-2} \text{ s}^{-1}$, but potted plants grown in the greenhouse following artificial inoculation remained symptomless under similar conditions. Do growth conditions modify expression of wilt response due to increasing virulence of the pathogen, or through alteration of the defense mechanisms of the host, or both? This remains to be clarified.

The implications of these findings are important mainly in breeding programs which require a simple, quick and reliable method for screening wilt response of new plant materials. Artificial inoculation of potted plants is one such method but, as indicated above, it may not be reliable enough in predicting the wilt response that would occur under natural conditions. Whether it would be possible to identify the specific conditions that would enable accurate predictions of wilt response in the field, is yet to be evaluated.

ACKNOWLEDGMENT

We would like to thank M. Reuven, M. Lampel, A. Zveibil, Y. Szmulewich and Y. Nitzani for their technical assistance.

REFERENCES

1. Aloi, C. and Baayen, R.P. (1993) Examination of the relationships between vegetative compatibility groups and races on *Fusarium oxysporum f.sp. dianthi*. *Plant Pathol.* 42:839-850.

2. Alon, H., Katan, J. and Kedar, N. (1974) Factors affecting penetrance of resistance to *Fusarium oxysporum* f.sp. *lycopersici* in tomatoes. *Phytopathology* 64:455-461.
3. Arus, P., Liaurado, M. and Pera, J. (1992) Progeny analysis of crosses between genotypes resistant and susceptible to *Fusarium oxysporum* f.sp. *dianthi* race 2. *Acta Hort.* 307:57-65.
4. Baayen, R.P. (1986) Regeneration of vascular tissues in relation to Fusarium wilt resistance of carnation. *Neth. J. Plant Pathol.* 92: 273-285.
5. Baayen, R.P. (1988) Responses related to lignification and intravascular periderm formation in carnations resistant to Fusarium wilt. *Can. J. Bot.* 66: 784-792.
6. Baayen, R.P. and Elgersma, D.M. (1985) Colonization and histopathology of susceptible and resistant carnation cultivars infected with *Fusarium oxysporum* f.sp. *dianthi*. *Neth. J. Plant Pathol.* 91: 119-135.
7. Baayen, R.P., Elgersma D.M., Demmink, J.F. and Spranaaij, L.D. (1988) Differences in pathogenesis observed among susceptible interactions of carnation with four races of *Fusarium oxysporum* f.sp. *dianthi*. *Neth. J. Plant Pathol.* 94: 81-94.
8. Baayen, R.P. and Schrama, R.M. (1990) Comparison of five stem inoculation methods with respect to phytoalexin accumulation and Fusarium wilt development in carnation. *Neth. J. Plant Pathol.* 93:315-320.
9. Baayen, R.P., Sparnaaij, L.D., Jansen, J. and Niemann, G.J. (1991) Inheritance of resistance in carnation against *Fusarium oxysporum* f.sp. *dianthi* races 1 and 2, in relation to resistance components. *Neth. J. Plant Pathol.* 97:73-86.
10. Baker, R. (1980) Measures to control Fusarium and Phytophthora wilt of carnation. *Plant Dis.* 64:743-749.
11. Ben-Yephet, Y. (1979) Isolate source and daylight intensity effects in the pathogenicity of *Verticillium dahliae* in watermelon seedlings. *Phytopathology* 69:1069-1072.
12. Ben-Yephet, Y. and Pilowsky, M. (1979) A method for determining susceptibility of tomatoes to *Verticillium dahliae* in the greenhouse. *Plant Dis. Rep.* 63:66-69.
13. Ben-Yephet, Y., Reuven, M. and Genizi, A. (1994) Effects of inoculum depth and density on Fusarium wilt in carnations. *Phytopathology* 84:1393-1398.
14. Ben-Yephet, Y., Reuven, M., Lampel, M., Nitzani, Y. and Mor, Y. (1992) *Fusarium oxysporum* f.sp. *dianthi* races in carnation. *Phytoparasitica* 20:225 (abstr.).
15. Ben-Yephet, Y., Reuven, M. and Mor, Y. (1993) Selection methods for determining resistance of carnation cultivars to *Fusarium oxysporum* f.sp. *dianthi*. *Plant Pathol.* 42:517-521.
16. Ben-Yephet, Y., Reuven, M., Szmulewich, Y., Nitzani, Y. and Mor, Y. (1992) Susceptibility reaction of carnation cultivars to *Fusarium oxysporum* f.sp. *dianthi* in an infested field and in spore suspension. *Acta Hort.* 307:83-89.
17. Ben-Yephet, Y., Reuven, M., Zviebil, A. and Shtienberg, D. (1996) Effect of abiotic variables on the response of carnation cultivars to *Fusarium oxysporum* f.sp. *dianthi*. *Plant Pathol.* 45:98-105
18. Ben-Yephet, Y., Reuven, M., Zviebil, A. and Shtienberg, D. (1996) Effects of initial inoculum and cultivar resistance on incidence of Fusarium wilt and population densities of *Fusarium oxysporum* f.sp. *dianthi* on carnation and in soil. *Phytopathology* 86:751-756.
19. Ben-Yephet, Y. and Shtienberg, D. (1994) Effect of solar radiation and temperature on Fusarium wilt in carnation. *Phytopathology* 84:1416-1421.
20. Ben-Yephet, Y., Shtienberg, D., Reuven, M. and Mor, Y. (1993) Response of carnation cultivars to *Fusarium oxysporum* f.sp. *dianthi* in the field. *Neth. J. Plant Pathol.* 99:3-12.
21. Bhatti, M.A. and Kraft, J.M. (1992) Effects of inoculum density and temperature on root rot and wilt of chickpea. *Plant Dis.* 76:50-54.

22. Blanc, H. (1983) Carnation breeding for resistance to *Fusarium oxysporum* f.sp. *dianthi*: practical achievement of resistant cultivars. *Acta Hortic.* 141:43-47.
23. Bosland, P.W., Williams, P.F. and Morrison, R.H. (1988) Influence of soil temperature on the expression of yellows and wilt of crucifers by *Fusarium oxysporum*. *Plant Dis.* 72:777-80.
24. Browne, C.A. (1942) Leibig and the Law of the Minimum. in: Moulton, F.R. [Ed.] Leibig and after Leibig. *Publ. Am. Assoc. Adv. Sci.* 16: 71-82.
25. Busch, L.V. and Edgington, L.V. (1967) Correlation of photoperiod with tuberization and susceptibility of potato to *Verticillium albo-atrum*. *Can. J. Bot.* 45:691-693.
26. Carrier, L.M. (1977) Breeding carnations for disease resistance in southern California. *Acta Hortic.* 71:182-187.
27. Cirulli, M. and Ciccarese, F. (1980) Influenza di alcuni fattori sull'espressione delle resistenze poligenica e monogenica a *Fusarium oxysporum* f.sp. *lycopersici* nel pomodoro. *Inf. Fitopatol.* 11-12:49-53.
28. Elmer, W.H. and Lacy, M.L. (1987) Effects of inoculum densities of *Fusarium oxysporum* f.sp. *apii* in organic soil on disease expression in celery. *Plant Dis.* 71:1086-1089.
29. Farquhar, G.D. and von Caemmerer, S. (1982) Modelling of photosynthetic response to environmental conditions. in: Lange, O.L., Nobel, P.S., Osmond, C.B. and Ziegler, H. [Eds.] *Physiological Plant Ecology II. Water Relations and Carbon Assimilation.* pp. 549-587. Springer-Verlag, New York, NY.
30. Fletcher, J.T. and Martin, J.A. (1972) Spread and control of Fusarium wilt of carnations. *Plant Pathol.* 21:182-187.
31. Gardiner, D.C., Horst, R.K. and Nelson, P.E. (1989) Influence of night temperature on disease development in Fusarium wilt of chrysanthemum. *Plant Dis.* 73:34-37.
32. Garibaldi, A.E. (1983) Resistenza di cultivar di garofano nei confronti di otto patogeni di *Fusarium oxysporum* f.sp. *dianthi* (Prill. et Del.) Snyd. et Hans. *Riv. Fruttic. Ortofloric. Ital.* 67:261-270.
33. Garibaldi, D.C. and Nelson, P.E. (1989) Influence of night temperature on disease development in Fusarium wilt of chrysanthemum. *Plant Dis.* 73:34-37.
34. Harling, R. and Taylor, G.S. (1985) A light microscope study of resistant and susceptible carnations infected with *Fusarium oxysporum* f.sp. *dianthi*. *Can. J. Bot.* 63: 638-646.
35. Harling, R., Taylor, G.S., Matthews, P. and Arthur, A.E. (1988) The effect of temperature on symptom expression and colonization in resistant and susceptible carnation cultivars infected with *Fusarium oxysporum* f.sp. *dianthi*. *J. Phytopathol.* 121:103-117.
36. Harris, A.R. and Ferris, H. (1991) Interaction between *Fusarium oxysporum* f.sp. *tracheiphilum* and *Meloidogyne* spp. in *Vigna unguiculata*. I. Effects of different inoculum densities on Fusarium wilt. *Plant Pathol.* 40:445-456.
37. Hood, J.R. and Stewart, R.N. (1957) Factors affecting symptom expression in Fusarium wilt of *Dianthus*. *Phytopathology* 47:173-178.
38. Jones, J.P., Crill, P. and Volin, R.B. (1975) Effect of light duration on Verticillium wilt of tomato. *Phytopathology* 65:647-648.
39. Katan, J. (1988) Soil temperature interactions with the biotic components of vascular wilt diseases. in: Tjamos, E.C. and Beckman, C.H. [Eds.] *Vascular Wilt Disease of Plants, Basic Studies and Control.* pp. 352-366. Springer-Verlag, New York, NY.
40. Manulis, S., Kogan, N., Reuven, M. and Ben-Yephet, Y. (1994) Use of the RAPD technique for the identification of *Fusarium oxysporum* f.sp. *dianthi* from carnation. *Phytopathology* 84:98-101.

41. Marois, J.J. and Mitchell, D.J. (1981) Effects of fumigation and fungal antagonists on the relationships of inoculum density to infection incidence and disease severity in Fusarium crown rot of tomato. *Phytopathology* 71:167-170.
42. Niemann, G.J. (1992) The mechanism of resistance of carnation to wilt diseases. *Acta Hort.* 307:29-37.
43. Paplomatas, E.J., Bassett, D.M., Broome, J.C. and DeVay, J.E. (1992) Incidence of Verticillium wilt and yield losses of cotton cultivars (*Gossypium hirsutum*) based on soil inoculum density of *Verticillium dahliae*. *Phytopathology* 82:1417-1420.
44. Pennypacker, B.W., Knievel, D.P., Risius, M.L. and Leath, K.T. (1994) Photosynthetic photon flux density x pathogen interaction in growth of alfalfa infected with *Verticillium albo-atrum*. *Phytopathology* 84:1350-1358.
45. Rotem, J. (1978) Climatic and weather influences on epidemics. *in*: Horsfall, J.G. and Cowling, E.B. [Eds.] *Plant Diseases: An Advanced Treatise*. Vol. II, pp. 317-337. Academic Press, New York, NY.
46. Rush, C.M. and Kraft, J.M. (1986) Effects of inoculum density and placement on Fusarium root rot of peas. *Phytopathology* 76:1325-1329.
47. Scovel, G. (1987) Improved agrotechnical and sanitation methods versus resistant cultivars as a means of avoiding Fusarium wilt. *Acta Hort.* 216:55-61.
48. Sparnaaij, L.D. and Demmink, J.F. (1977) Progress towards Fusarium resistance in carnation. *Acta Hort.* 71:107-113.
49. Tramier, R., Anthoni, A. and Bettachini, A. (1987) Variation of the tolerance level of carnation cultivars against *Fusarium oxysporum* f.sp. *dianthi* depending on the substrate. *Acta Hort.* 216:105-109.
50. Tramier, R., Anthoni, A., Bettachini, A. and Metay, C. (1983) Studies of Fusarium wilt resistance in carnation. *Acta Hort.* 141:49-54.
51. Tsrer (Lahkim), L., Livescu, L. and Nachmias, A. (1990) Effect of light duration and growth season on Verticillium wilt in potato. *Phytoparasitica* 18:331-339.
52. Vigodsky Haas, H. and Klein, L. (1976) Influence of methyl bromide soil fumigation method on fungicidal efficacy and bromide residues. *Phytoparasitica* 4:123-129.