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Tylose formation in elms after inoculation with an aggressive or a non-aggressive strain of Ophiostoma ulmi or with a non-pathogen to elms

D. M. ELGERSMA and H. J. MILLER

Phytopathological Laboratory 'Willie Commelin Scholten', Baarn

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After inoculation of elms with *Ophiostoma ulmi* (Buisman) Nannf., the causal organism of Dutch elm disease, it was found that tylose formation in vessels of the susceptible elm clone Belgica occurred later than in clone 390 (Elgersma, 1973) which is resistant to the so-called non-aggressive strains of *O. ulmi*. It was suggested that tylose formation might serve as a resistance mechanism by preventing the spread of the pathogen; it was further assumed that in compatible elm-*O. ulmi* combinations the pathogen produces a compound which inhibits tylose formation.

To evaluate this hypothesis, 2-year-old nursery grown callus cuttings of clone Belgica, which is susceptible to all strains of *O. ulmi*, and of clone 390, which is resistant to the non-aggressive, but susceptible to the aggressive strains of *O. ulmi* (Elgersma, 1976), were inoculated with the aggressive strain H6 or with the non-aggressive strain E2 of *O. ulmi* with the techniques described earlier (Elgersma, 1969). The inoculum contained 10^6 conidia per ml. Additional inoculations were carried out with *Fusarium oxysporum* Schlecht. f. sp. *lycopersici* (Sacc.) Sny. & Hans., a vascular parasite of tomatoes, but non-pathogenic to elms.

On the 3rd, 4th and 5th day after inoculation, 2 cm-stem pieces were cut 5 cm above the site of inoculation, using 10 plants in each combination at a time, which were fixed in formalin-ethanol (70%) – propionic acid (5:90:5) and sectioned transversely at 20 µm thickness. The total number of vessels in the latest developed annual ring was counted and the proportion containing tyloses determined as described earlier (Elgersma, 1973).

Results are shown in Table 1. No significant differences in tylose formation could be found within the clone Belgica after inoculation with either of the three fungi. Similar results were obtained in clone 390 after inoculation with either one of these fungi. However, the numbers and percentages of vessels with tyloses in clone 390 are approximately double those found in clone Belgica. Plants treated with water instead of a conidium suspension showed only a small and insignificant number of tyloses. Tylose formation in clone Belgica as well as in clone 390 is not inhibited in case of a compatible host-parasite combination, but seems to be genetically determined by the elm clone. The increased rate or intensity of tylose formation in clone 390 as compared with clone Belgica still lends support to the belief of many authors including Elgersma (1973), that tylose formation plays a role in resistance. Differences

Table 1. Tylose formation in vessels of elm clones Belgica and 390 after inoculation with an aggressive (H6) strain or a non-aggressive (E2) strain of *O. ulmi* or with a non-pathogen (*Fusarium oxysporum* f. sp. *lycopersici*). The first figure in each column represents the actual number of vessels with tyloses (average of 10 replicates), the figure between the brackets represents the percentage of vessels with tyloses.

Number of days after inoculation	*U. hollandica* cl. Belgica			*U. hollandica* cl. 390		
	O. ulmi		*F. oxysporum* f. sp. *lycopersici*	*O. ulmi*		*F. oxysporum* f. sp. *lycopersici*
	strain H6	strain E2		strain H6	strain E2	
3	17 (2.4)	20 (3.3)	22 (2.7)	42 (5.0)	40 (6.8)	42 (6.0)
4	58 (7.7)	58 (7.7)	35 (6.1)	89 (10.3)	97 (12.5)	71 (9.9)
5	67 (7.4)	53 (6.0)	59 (7.4)	109 (11.6)	104 (10.6)	106 (10.9)

Tabel 1. Thyllenvorming in vaten van iepeklonen Belgica en 390 na inoculatie met een agressieve (H6) stam of een niet-agressieve (E2) stam van O. ulmi of met een niet pathogeen (Fusarium oxysporum f. sp. lycopersici). Het eerste getal in iedere kolom geeft het aantal vaten met thyllen weer (gemiddelde van 10 herhalingen), het getal tussen haakjes geeft het percentage van vaten met thyllen weer.

in susceptibility can also be related to the differences in anatomy (Elgersma, 1969, 1970; McNabb et al., 1970; Sinclair et al., 1975). As no significant differences appear to exist between the tyloses induced by *O. ulmi* strains E2 and H6 in clone 390, we must assume that a bypassing of obstacles such as tyloses is probable. This was suggested by Miller and Elgersma (1976) as being due to a more rapid growth and penetration of the vessel pits by the aggressive strain.

Samenvatting

Thyllenvorming in iepen na inoculatie met een agressieve, een niet-agressieve stam van Ophiostoma ulmi of met een niet-pathogeen voor iepen

De hypothese dat de thyllenvorming in de houtvaten van de iep als reactie op een infectie met *Ophiostoma ulmi* alleen in de compatibele waardplant-pathogeen combinatie vertraagd zou zijn, werd getoetst. Er bleek geen significant verschil in snelheid van thyllenvorming op te treden als de vatbare kloon Belgica geïnoculeerd werd met een agressieve en een niet-agressieve stam van *O. ulmi* of met *Fusarium oxysporum* f. sp. *lycopersici*, een niet-pathogeen voor iepen. Overeenkomstige resultaten werden verkregen wanneer kloon 390, die resistent is tegen de niet-agressieve stam van *O. ulmi*, geïnoculeerd werd met deze drie schimmels. In deze gevallen wordt de snelheid van thyllenvorming alleen bepaald door de aard van de betreffende iepekloon.

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Adresses

D. M. Elgersma: Phytopathologisch Laboratorium 'Willie Commelin Scholten', Javalaan 20, Baarn, the Netherlands.
H. J. Miller: Plantenziektenkundige Dienst, Geertjesweg 15, Wageningen, the Netherlands.

Book review

H. Wheeler, 1975. Plant pathogenesis. Springer-Verlag, Berlin, Heidelberg, New York. 106 pp., 19 figs. Price DM 39.

This concise and very clearly written book presents an excellent account of the process of plant pathogenesis. The author defines it as: 'the sequence of events that occur during the development of a disease'. It is based on work with fungal pathogens.

After a short introductory chapter on concepts (4 pp.), a review is given of the mechanisms of pathogenesis (28 pp.). Penetration phenomena together with the 'chemical weapons' (enzymes, hormones, toxins) used by the pathogen are discussed. In Chapter 3 follow the 'responses of plants to pathogens' (35 pp.) as manifested by pathological alterations in structure, function and metabolism. Much attention is paid to recent additions to our knowledge on the ultrastructural level. Chapter 4 on 'disease resistance mechanisms' (11 pp.) deals mainly with resistance induced by inoculation with a non-pathogen and pays much attention to phytoalexins. Chapter 5 describes the genetics of pathogenesis (13 pp.) with an illuminating discussion of the gene for gene concept. The biochemical basis for pathogen specificity receives special attention. In a short final chapter on the nature of the physiological syndrome (5 pp.), the author speculates on the nature of the responses of the host to pathogens on the cellular level. Changes in the distribution of negative charges on the cell wall, changes in permeability and cell wall synthesis may be the initial events which trigger subsequent pathogenic processes. A list of 195 references, most of them quite recent, concludes the book.

The book is very stimulating in that it presents not merely facts (in a very systematic way), but also the author's interpretation thereof. The author is to be congratulated on his use of examples to illustrate the phenomena described and to make his points clear. One can thereby easily forgive the emphasis given to examples drawn from his own work *(Helminthosporium victoriae)*.The excellent integration of biochemical, ultrastructural and genetic information is an important aspect of this book.

Its reading can be recommended to all phytopathologists.

T. Limonard