MOLECULAR PLANT PATHOLOGY (2001) **2**(6), 303–309

Pathogen profile

The tomato powdery mildew fungus *Oidium neolycopersici*

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SUMMARY

Pathogen: Powdery mildew fungus; Ascomycete although sexual stage is yet to be found; an obligate biotroph.

Identification: Superficial mycelium with hyaline hyphae; unbranched erect conidiophores; conidia, ellipsoid-ovoid or doliform, 22–46 × 10–20 μm, lack fibrosin bodies; conidia formed singly, rarely in short chains of 2–6 conidia; appressoria lobed to multilobed, rarely nipple-shaped. *Pseudoidium* species.

Host range: Broad, reported to attack over 60 species in 13 plant families, particularly members of the Solanaceae and Curcubitaceae.

Symptoms: Powdery white lesions on all aerial plant parts except the fruit. In severe outbreaks the lesions coalesce and disease is debilitating.

Agronomic importance: Extremely common in glasshouse tomatoes world wide but increasing in importance on field grown tomato crops.

Control: Chemical control and breeding programmes for disease resistance.

DISEASE SYMPTOMS

Oidium neolycopersici is a highly polyphagous powdery mildew fungus which infects tomatoes. It causes powdery white lesions on the adaxial tomato leaf surface. The fungus can also infect abaxial surfaces, petioles and the calyx but the fruit remains uninfected (Fig. 1). Severe infections lead to leaf chlorosis, premature senescence and a marked reduction in fruit size and quality (Whipps *et al.*, 1998). *Oidium neolycopersici* currently poses a significant threat to glasshouse-grown tomatoes and is of increasing importance on field-grown tomato crops.

THE PATHOGEN

A new powdery mildew disease on tomato plants was reported in the UK in 1986 (Fletcher *et al.*, 1988) but it has now spread world-wide. However, its true identity was uncertain due to the lack of a sexual stage and varying reports of its structure, particularly whether conidia were formed singly or in chains. Consequently, the new mildew pathogen on tomato plants was variously termed *O. lycopersicum, Erysiphe orontii* or *E. cichoracearum* (Bélanger and Jarvis, 1994; Boiteux, 1994; Koike and Saenz, 1999) or was simply described as *Erysiphe* sp. (Arredondo *et al.*, 1996; Karasevicz and Zitter, 1996; Kiss, 1996; Neshev, 1993; Olalla and Torés, 1998; Pernezny and Sonoda, 1998; Smith *et al.*, 1997;

Fig. 1 Powdery white lesions of *O. neolycopersici* on tomato (var. Moneymaker) leaves but the fruit is uninfected.

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Vakalounakis and Papadakis, 1992). The first appropriate description of the fungus, *Oidium lycopersicum,* appeared to come from Australia (Cooke and Massee, 1888), and the name was redesignated, in 1999, as *Oidium lycopersici*, in accordance with the International Code of Botanical Literature (Mieslerova and Lebeda, 1999). However, confusion remained over classification based on morphological characteristics. Consequently, we analysed the internal transcribed spacer regions of the nuclear rRNA genes from the new tomato powdery mildew pathogen and were able to differentiate *Oidium (neo)lycopersici* from *E. orontii* and *E. cichoracearum* (Jones *et al*., 2000). Moreover, we found *O. (neo)lycopersici* to be a sister taxon of *E. aquilegia* var. *ranunculi* (Jones *et al*., 2000). This was confirmed by Kiss *et al*. (2001) from a study of tomato powdery mildew fungi from Europe, North and South America, and Asia. Importantly, Kiss *et al*. (2001) recognized that all recent outbreaks of tomato powdery mildew reported outside Australia were caused by a species that formed conidia singly, or, in high relative humidity, in pseudo-chains of 2–6 conidia, and so created a new species, *O. neolycopersici*, for this pathogen. The Australian isolates, which always formed conidia in chains, retained the name *Oidium lycopersici*.

ORIGINS

The origins of *Oidium neolycopersici* are still unclear. Huang *et al*. (2000) proposed that *O. neolycopersici* may have 'jumped' hosts by acquiring one or more pathogenicity factors but, as yet, there is no direct evidence to substantiate this suggestion. However, there are descriptions of resistance to *Oidium neolycopersici* identified in wild *Lycopersicon* species, including *L. hisutum, L. pennelli* and *L. parviflorum* (Ciccarese *et al*., 1998; Kozik, 1993; Laterrot *et al*., 1995; Lindhout *et al*., 1994a,b; Mieslerova *et al*., 2000; van der Beek *et al*., 1994) which could indicate that the appearance of *O. neolycopersici* is not a recent phenomenon, but rather one that has just become more apparent. This is also implied from the identification of *O. neolycopersici* from herbarium specimens from Asia since 1947 (Kiss *et al*., 2001). Nevertheless, this does not answer the question of why *O. neolycopersici* has become such a problem recently and why it has spread so rapidly around the world. The increased movement of plants though the international horticulture trade could be responsible, but so too could the aerial dispersal of conidia and their subsequent survival on numerous alternative hosts.

PATHOGEN MORPHOLOGY AND DEVELOPMENT ON THE HOST

The ellipsoidal-shaped spores of *O. neolycopersici* are approximately 30 μm × 15 μm (Jones *et al*., 2000). The conidial surface is covered by irregular arrays of ribbon-like and rounded projections (Fig. 2A) but, on germination, a smooth-surfaced germ tube emerges from the body of the conidium (Fig. 2B). This tube elongates at the tip and becomes lobed or 'clover-leaf' like in configuration. This appressorial structure is commonly found at the junction of three epidermal cells (Fig. 2C). Subsequently, a peg emerges from the centre of the 'palm' of the lobed appressorium and penetrates the host plant, leaving a penetration pore of approximately 0.2 μm diameter (Fig. 2D). Rapid colonization of the host plant follows as secondary hyphae radiate from both the conidial body and the primary appressorium. Secondary appressoria of *O. neolycopersici* develop either singly or in pairs (Fig. 2E) from the hyphae which ramified over the host surface. The asexual life-cycle is completed by the formation of conidiophores which 'stand' perpendicular to the host surface on a straight cylindrical foot cell, supporting a meristematic zone of immature conidia and which carry a single mature ellipsoidal conidium at the apex of the column (Fig. 2F). During germling morphogenesis the three key developmental changes are staged, with germination occurring 3–5 h after inoculation (hai), appressorium differentiation 6–8 hai and penetration at approximately 11 hai (Jones, 2001).

PATHOGENESIS

Host-derived signals

Since *Oidium neolycopersici* is an obligate biotroph, the development of mature, sporulating colonies depends upon successful initial penetration of the host and then continued haustorial formation and functioning. But, does the conidium perceive host signals to initiate germination and does the germling require additional signals to differentiate functional appressoria? Furthermore, does *O. neolycopersici* sense chemical and physical features on the host surface which drive differentiation and cue penetration? Much effort has been expended in attempts to identify features inductive to fungal plant pathogen development. These include leaf topography in *Uromyces* sp. (Hoch *et al*., 1987), hydrophobicity in *Magnaporthe grisea* (Howard *et al*., 1991) surface hardness, wax and ethylene in *Colletotrichum gloeosporioides* (Flaishman and Kolattukudy, 1994; Flaishman *et al*., 1995; Kim *et al*., 1998), cutin monomers in *Blumeria graminis* (Francis *et al*., 1996), cellulose breakdown products in *Erysiphe pisi* (Carver *et al*., 1996) and flavanoids in *Nectria haematococca* (Bagga & Straney, 2000). Preliminary analysis of differentiation on various artificial substrata revealed that hydrophobic surfaces and cellulose, as well as host wax, induce germling morphogenesis in *O. neolycopersici* (Jones, 2001). Furthermore, small peaks in *O. neolycopersici* cutinase activity, monitored both *in vivo* (on tomato leaf discs) and *in vitro* (in multiwell plate assays), 'coincided' at 4 and 11 hai, notably at the peak time for germination and for host penetration, respectively. This data suggests a role for cutinase activity in either the perception of host-derived cutin breakdown products, found in a range of plant

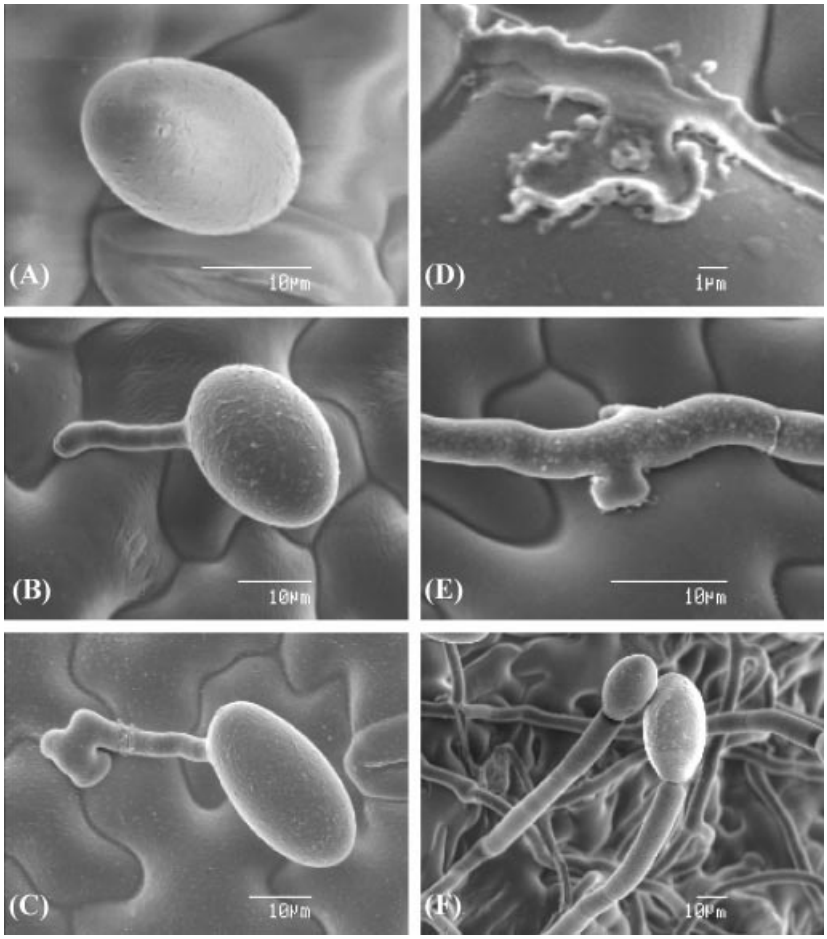

Fig. 2 Low temperature scanning electron micrographs of cryofixed tissue. (A) Ellipsoidal conidium lying obliquely on the plant surface. (B) A single smooth-surface germ tube has emerged from the conidium, 5 hai. (C) A lobed and hooked appressorium has developed, 10 hai. A 'craggy' collar delimits the conidial body and germ tube from the primary appressorium. (D) Imprint left by peeling (with gelatin) the superficial hyphae and a single hyphal appressorium from the infection site to expose the fungal extracellular matrix both in hyphal tracks and the outline of the lobed appressorium and central 'O'ring, which delimits the penetration pore. (E) Paired hyphal appressoria. (F) Two conidiophores bearing a single apical and near mature ellipsoidal conidium.

pathogenic fungi including other powdery mildews (Fan and Koller, 1998; Francis *et al.*, 1996; Fric and Wolf, 1994; Rumbolz *et al.*, 2000) or else in the use of enzymic means to breach the host (Maiti and Kolattukudy, 1979; Podila *et al.*, 1995; Rogers *et al.*, 1994).

Adhesion

How does this pathogen adhere to the plant? Deposits of extracellular matrix (ECM) material lie beneath the *O. neolycopersici* germ tubes, hyphae, around the margins of the appressorium and surrounding the O-ring at the site of host penetration, but not beneath ungerminated spores (Jones *et al.*, 2000). Such material is assumed to be fungal in origin and is reminiscent of the patterns of ECM seen in *Blumeria graminis* (Carver *et al.*, 1995a,b). Herein may lie a clue to its presence, as Carver proposes that the ECM boosts the adhesion of *B. graminis* to its host and that the ECM may provide a medium for the localization of enzymes involved in penetration (Carver *et al.*, 1995b). In binding assays with *O. neolycopersici*, the adhesion of ungerminated conidia to the host surface was compared with that of differentiated appressorial-stage tissue. Here, the presence of ECM seemed pivotal for successful adhesion, as appressorial stage tissue remained attached to the host, whilst ungerminated conidia did not (Jones, 2001).

Recently, it has been suggested that dimeric *trans*-membrane proteins, termed integrins, may play a key role in cell adhesion, recognition, organization of the cytoskeleton and intracellular signalling in a range of animal and microbial systems (Bendel and Hostetter, 1993; Hostetter, 2000; Schoenwaedler and Burridge, 1999). In *Candida albicans* the application of the tripeptide arginine-glycine-aspartic acid (RGD) blocks both adhesion to, and perception of, the host (Bendel and Hostetter, 1993). Moreover, in *Uromyces appendiculatus,* exogenous application of RGD prevents fungal perception of the topographical features of the host surface (Corrêa *et al.*, 1996; Hoch *et al.*, 1987). The colocalization of integrins with stretch-activated Ca^{2+} channels suggests an involvement of integrins in intracellular signalling in *Saprolegnia ferax* (Levina *et al.*, 1994). Therefore, integrins have been proposed to be involved in specific interactions of pathogenic fungi with their hosts and to initiate intracellular signalling cascade(s) (Corrêa *et al.*, 1996). Preliminary work with

O. neolycopersici demonstrated that the tetra-peptide, arginine-glycine-aspartate-serine (RGDS), was a more potent inhibitor of germination than RGD and led to aberrant appressorial differentiation in *O. neolycopersici*. Here, the swollen appressoria plasmolysed more easily than controls when exposed to reduced external osmotic pressure, suggesting that a putative integrin may exist for the perception of the host surface, in accordance with work carried out on *U. appendiculatus* (Corrêa *et al*., 1996; Jones, 2001).

Penetration

Does *O. neolycopersici* breach the host cuticle and epidermal cell wall by enzymic activity, by mechanical force, or by a combination of both? The small peak in cutinase activity at the time of penetration and the appearance of the smooth-edged penetration hole (Fig. 2D) suggest that enzymes may play a role in penetration. (Jones *et al*., 2000; Jones, 2001). Nevertheless, development of increased turgor pressure in appressoria has been found to be the key feature of penetration in a range of plant pathogenic fungi including powdery mildews (de Jong *et al*., 1997; Howard *et al*., 1991; Money and Howard, 1996; Money *et al*., 1998; Money, 1995; Pryce-Jones *et al*., 1999). For *O. neolycopersici*, we have strong data supporting the use of mechanical force in penetration. Cell turgor measurements, determined by cytorrhysis and plasmolysis experiments, revealed that mature *O. neolycopersici* appressoria produce a maximum turgor pressure of approximately 3 MPa, coincident with host cell penetration from the appressoria, at 11 hai. This turgor pressure is in near concordance with the maximum pressure in *Blumeria graminis* appressoria recorded at the time of host penetration (Pryce-Jones *et al*., 1999). It is, however, significantly lower than the 8 MPa recorded in the melanized appressoria of the rice blast fungus *Magnaporthe grisea* (Howard *et al*., 1991). There is no evidence to support the presence of melanin in the hyaline appressoria of either *O. neolycopersici* or *Blumeria graminis* and so just how the powdery mildew fungi withstand such osmotic pressure remains a mystery.

HOST RANGE

Host range studies with *Oidium lycopersici* have held two main objectives. Firstly, to assess the risk posed by *O. neolycopersici* to a wide-range of horticultural crops and, secondly, to distinguish it from other powdery mildew species known to infect tomatoes. In 1988, Fletcher *et al*. found that *O. neolycopersici* infected all the tomato varieties tested, in addition to aubergine, potato and tobacco. However, a more extensive host range study was carried out by Whipps *et al*. (1998), who tested economically important plant species and also those purported to be hosts of *E. orontii*. Such work revealed that members of 13 families were alternative hosts for *O. neolycopersici*. Furthermore, this work and morphological characteristics led Whipps *et al*. (1998) to propose that *O. neolycopersici* was distinct from *E. orontii*.

However, not all workers agree on the exact host range of *O. neolycopersici* (Fletcher *et al*., 1988; Huang *et al*., 2000; Kiss, 1996; LaMondia, 1999; Mieslerova and Lebeda, 1999; Smith *et al*., 1997; Whipps *et al*., 1998) perhaps suggesting that different pathotypes exist, further complicating the situation (Huang *et al*., 2000; Lebeda and Mieslerová, 1999). Such disparity serves to highlight the limitations of identification of powdery mildews by host range alone. It calls for a combination of host-range work, molecular analyses and detailed morphological characterization prior to naming a given pathogen.

CHEMICAL CONTROL

When *O. neolycopersici* appeared it spread rapidly around the world. All commercial tomato cultivars tested were found to be susceptible to *O. neolycopersici* (Lindhout *et al*., 1994a, 1994b; van der Beek *et al*., 1994), and initially, good control of the disease was only achieved by the use of fungicides. Effective active ingredients include benomyl, bitertanol, bupirimate, carbendazim, fenarimol, pyrazophos, thiabendazol, triforine and various sulphur preparations, although relative efficacy appears to vary (Mieslerova and Lebeda, 1999). Currently, in the UK, 'off-label' approval has been granted for the use of bupirimate (Nimrod, from Zeneca), fenarimol (Rubigan, from DowAgrosciences) and sulphur (Thiovit, from Novartis) (Whitehead, 2001). In addition, we found the quinoline fungicide Quinoxyfen (Fortress, from DowAgrosciences), which is known to prevent infection by other powdery mildews (Wheeler *et al*., 2000), showed high efficacy in the inhibition of both germination and differentiation of *O. neolycopersici* (Jones, 2001) but we have not undertaken extensive glasshouse tests of this compound.

PROSPECTS

Resistant cultivars

Over the last decade, much research effort has focused on testing wild tomato species for their resistance to *Oidium neolycopersici* infection. Resistance at various levels has been found in *Lycopersicon cheesmanii, L. chilense, L. chmielski L. hisutum, L. minutum, L. parviflorum, L. pennelli* and *L. peruvianum* (Huang *et al*., 1998; Laterrot *et al*., 1995; Lindhout *et al*., 1994a; Mieslerova *et al*., 2000). These wild species are grouped into the 'peruvianum' and 'esculentum' complexes, according to whether they can be crossed easily with the commercial tomato, *L. esculentum* (Rick and Chetelat, 1995). The 'esculentum' complex comprises *L. esculentum*, *L. cheesmanii, L. hirsutum, L. pimpinellifolium*, *L. parviflorum* and *L. pennelli*, and these species form the main

focus of disease resistance screening against *O. neolycopersici*, due to the relative ease with which they can be crossed with commercially grown tomato cultivars (Lebeda and Mieslerová, 1999; Mieslerova *et al*., 2000).

Particular attention has been paid to resistance in *L. hirsutum* (Laterrot *et al*., 1995; Lindhout *et al*., 1994a) and the high degree of disease resistance of a pure line of *L. hirsutum* was attributed to the incompletely dominant *Ol*-1 gene, found to co-segregate with two other resistance genes (Lindhout *et al*., 1994b; van der Beek *et al*., 1994). Whilst breeding for monogenic resistance has met with some success, the commercial tomato hybrid DRW 4061 (*L. hirsutum* × *L. esculentum*) exhibits variable resistance to *O. neolycopersici* (Mieslerova *et al*., 2000). In addition, resistance to infection by *O. neolycopersici* has also been found in *L. esculentum* var. *cerasiforme* and appeared to be due to a single recessive *Ol*-2 gene (Ciccarese *et al*., 1998). Clearly, more hybrids with more durable disease resistance are needed.

Although the 'esculentum' complex forms the current focus for breeding programmes, work on introgressing resistance from the 'peruvianum' complex into *L. lycopersicon* is continuing (Mieslerova *et al*., 2000). Lindhout *et al*. (1994a) propose that the two complexes may carry different mechanisms of disease resistance to *Oidium neolycopersici* infection, due to their differing geographical locations which may have prevented the co-segregation of resistance genes. Clearly, the basis for the mechanisms of resistance to *O. neolycopersici* in these groups deserves further study.

Biological control

A final area that may develop in the next few years is the use of biological or integrated control for *O. neolycopersici*. A number of mycoparasites with activity against powdery mildews are known, including *Ampelomyces quisqualis, Sporothrix flocculosa, Stephanoascus rugulosus, Tilletiopsis* species and *Verticillium lecanii* (Falk *et al*., 1995; Hajlaoui *et al*., 1992; Hijwegen, 1992; Verhaar *et al*., 1997) and there is the potential to combine these with foliar sprays of phosphate and potassium salts, acting both through direct effects on the pathogen and induced resistance in the plant (Reuveni *et al*., 1994, 1996). Preliminary experiments have shown that foliar sprays of *Sporothrix flocculosa* reduce the development of *O. neolycopersici* on tomato in the glasshouse (J.M. Whipps, unpublished data) and this could be a useful approach for sustainable control of this pathogen.

ACKNOWLEDGEMENTS

H.J. is a BBSRC special CASE student with Horticulture Research International. We extend our particular thanks to Barry Thomas and Tim Carver at IGER for their expertise and help in gathering the SEM images.

REFERENCES

Arredondo, C.R., Davis, R.M., Rizzo, D.M. and Stahmer, R. (1996) First report of powdery mildew of tomato in California caused by *Oidium* sp. *Plant Dis.* **80**, 1303.

Bagga, S. and Straney, D. (2000) Modulation of cAMP and phosphodiesterase activity by flavanoids which induce spore germination of *Nectria haematococca* MP VI (*Fusarium solani*). *Physiol. Mol Plant Pathol.* **56**, 51–61.

van der Beek, L.G., Pet, G. and Lindhout, P. (1994) Resistance to powdery mildew (*Oidium lycopersicum*) in *Lycopersicon hirsutum* is controlled by an incompletely-dominant gene *Ol*-1 on chromosone 6. *Theor. Appl. Genet.* **89**, 467–473.

Bélanger, R.R. and Jarvis, W.R. (1994) Occurrence of powdery mildew (*Erysiphe* sp.) on greenhouse tomatoes in Canada. *Plant Dis*. **78**, 640.

Bendel, C.M. and Hostetter, M.K. (1993) Distinct mechanism of epithelial adhesion for *Candida albicans* and *Candida tropicalis. J. Clin. Invest.* **92**, 1840–1849.

Boiteux, L.S. (1994) Powdery mildew of potato caused by *Erysiphe cichoracearum* in Brazil. *Plant Dis.* **78**, 830.

Carver, T.L.W., Ingerson, S.M. and Thomas, B.J. (1996) Influences of host surface features on development of *Erysiphe graminis* and *Erysiphe pisi*. In *Plant Cuticles—an Integrated Functional Approach* (Kersteins, G., ed.). Oxford, UK: Bios Scientific Publishers, pp. 255–266.

Carver, T.L.W., Ingerson-Morris, S.M. and Thomas, B.J. (1995a) Early interactions during powdery mildew infection. *Can. J. Bot.* **73**(Suppl. 1), S632–S639.

Carver, T.L.W., Thomas, B.J. and Ingerson-Morris, S.M. (1995b) The surface of *Erysiphe graminis* and the production of extracellular material at the fungus–host interface during germling and colony development. *Can. J. Bot.* **73**, 272–287.

Ciccarese, F., Amenduni, M. and Schiavone, D. (1998) Cirulli Occurrence and inheritance of resistance to powdery mildew (*Oidium lycopersici*) in *Lycopersicon* sp. *Plant Pathol.* **47**, 417–419.

Cooke, M.C. and Massee, G. (1888) Australasian fungi. *Grevillea*, **16**, 114.

Corrêa, A., Staples, R.C. and Hoch, H.C. (1996) Inhibition of thigmostimulated cell differentiation with RGD-peptides in *Uromyces* germlings. *Protoplasma*, **194**, 91–102.

Falk, S.P., Gadoury, D.M., Pearson, R.C. and Seem, R.C. (1995) Partial control of grape powdery mildew by the mycoparasite *Ampelomyces quisqualis. Plant Dis.* **79**, 483–490.

Fan, C.-Y. and Koller, W. (1998) Diversity of cutinases from plant pathogenic fungi: differential and sequential expression of cutinolytic esterases by *Alternaria brassicicola. FEMS Microbiol. Lett.* **158**, 33–38.

Flaishman, M.A., Hwang, C.-S. and Kolattukudy, P.E. (1995) Involvement of protein phosphorylation in the induction of appressorium formation in *Colletotrichum gloeosporioides* by its host surface wax and ethylene. *Physiol. Mol. Plant Pathol.* **47**, 103–117.

Flaishman, M.A. and Kolattukudy, P.E. (1994) Timing of fungal invasion using host's ripening hormone as signal. *Proc. Natl Acad. Sci. USA*, **91**, 6579–6583.

Fletcher, J.T., Smewin, B.J. and Cook, R.T.A. (1988) Tomato powdery mildew. *Plant Pathol.* **37**, 594–598.

Francis, S.A., Dewey, F.M. and Gurr, S.J. (1996) The role of cutinase in germling development and infection by *Erysiphe graminis* f.sp. *hordei. Physiol. Mol. Plant Pathol.* **47**, 201–211.

Fric, F. and Wolf, G. (1994) Hydrolytic enzymes of ungerminated and germinated conidia of *Erysiphe graminis* DC f.sp. *hordei* Marchal. *J. Phytopathol.* **140**, 1–10.

Hajlaoui, M.R., Benhamou, N. and Belanger, R.R. (1992) Cytochemical study of the antagonistic activity of *Sporothrix flocculosa* on rose powdery mildew, *Sphaerotheca pannosa* var. *rosae. Phytopathol*, **82**, 583–589.

Hijwegen, T. (1992) Biological control of cucumber powdery mildew with *Tilletiopsis minor* under greenhouse conditions. *Neth. J. Plant Pathol.* **98**, 221–225.

Hoch, H.C., Staples, R.C., Whitehead, B., Comeau, J. and Wolf, E.D. (1987) Signaling for growth orientation and cell differentiation by surface topography in *Uromyces. Science*, **235**, 1659–1662.

Hostetter, M.K. (2000) RGD-mediated adhesion in fungal pathogens of humans, plants and insects. *Curr. Opin. Microbiol.* **3**, 344–348.

Howard, R.J., Ferrari, M.A., Roach, D.H. and Money, N.P. (1991) Penetration of hard surfaces by a fungus employing enormous turgor pressures. *Proc. Natl Acad. Sci. USA*, **88**, 11281–11284.

Huang, C.-C., Biesheuvel, J., Lindhout, P. and Niks, R.E. (2000) Host range of *Oidium lycopersici* occuring in the Netherlands. *Eur. J. Plant Pathol.* **106**, 465–473.

Huang, C.-C., Groot, T., Meijer-Dekens, F., Niks, R.E. and Lindhout, P. (1998) The resistance to powdery mildew (*Oidium lycopersicum*) in *Lycopersicon* species is mainly associated with hypersensitive response. *Eur. J. Plant Pathol.* **104**, 399–407.

Jones, H.E. (2001) *Identification and development of the tomato powdery mildew fungus, Oidium lycopersici*. DPhil thesis. University of Oxford.

Jones, H.E., Whipps, J.M., Thomas, B.J., Carver, T.L.W. and Gurr, S.J. (2000) Initial events in the colonization of tomatoes by *Oidium lycopersici*, a distinct powdery mildew fungus of *Lycopersicon* species. *Can J. Bot.* **78**, 1361–1366.

de Jong, J.C., McCormack, B.J., Smirnoff, N. and Talbot, N.J. (1997) Glycerol generates turgor in rice blast. *Nature*, **389**, 244–245.

Karasevicz, D.M. and Zitter, T.A. (1996) Powdery mildew occurrence on greenhouse tomatoes in New York. *Plant Dis*, **80**, 709.

Kim, Y.-K., Li, D. and Kolattukudy, P.E. (1998) Induction of Ca^{2+}- calmodulin signaling by hard surface contact primes *Colletotrichum gloeosporioides* conidia to germinate and form appressoria. *J. Bacteriol.* **180**, 5144–5150.

Kiss, L. (1996) Occurrence of a new powdery mildew fungus (*Erysiphe* sp.) on tomatoes in Hungary. *Plant Dis.* **80**, 224.

Kiss, L., Cook, R.T.A., Saenz, G.S., Cunnington, J.H., Takamatsu, S., Pascoe, I., Bardin, M., Nicot, P.C., Sato, Y. and Rossman, A.Y. (2001) Identification of two powdery mildew, *Oidium neolycopersici* sp. nov. and *Oidium lycopersici*, infecting tomato in different parts of the world. *Mycol. Res.* **105**, 684–697.

Koike, S.T. and Saenz, G.S. (1999) Powdery mildew of spearmint caused by *Erysiphe orontii* in California. *Plant Dis.* **8**, 399.

Kozik, E. (1993) Resistance to powdery mildew *Oidium lyopersici* in tomato. *Tomato Genet. Coop. Rep.* **43**, 26.

LaMondia, J.A. (1999) Host range of *Oidium lycopersicum* on selected solanaceous species in Connecticut. *Plant Dis.* **83**, 341–344.

Laterrot, H., Moretti, A., Conus, M. and Mas, P. (1995) Breeding for *Oidium lycopersicum* resistance. *Tomato Genet. Coop. Rep.* **45**, 30.

Lebeda, A. and Mieslerová, B. (1999) Identification, occurrence and host range of tomato powdery mildew (*Oidium lycopersici*) in the Czech republic. *Acta Phytopathol. Entol. Hung.* **34**, 15–27.

Levina, N., Lew, R.R. and Heath, I.B. (1994) Cytoskeltal regulation of ion channel distribtuion in the tip-growing organism *Saprolegnia ferax. J. Cell Sci.* **107**, 127–134.

Lindhout, P., Pet, G. and van der Beek, J.G. (1994a) Screening wild *Lycopersicon* species for resistance to powdery mildews (*Oidium lycopersicum*). *Euphytica*, **72**, 43–49.

Lindhout, P., van der Beek, J.G. and Pet, G. (1994b) Wild *Lycopersicon* species as sources for resistance to powdery mildew (*Oidium lycopersicum*). mapping of the resistance gene OL-1 on chromosone of *L. hirsutum. Acta Hort.* **376**, 387–394.

Maiti, I.B. and Kolattukudy, P.E. (1979) Prevention of fungal infection of plants by specific inhibition of cutinase. *Science*, **205**, 507–508.

Mieslerova, B. and Lebeda, A. (1999) Taxonomy, distribution and biology of the tomato powdery mildew (*Oidium lycopersici*). *J. Plant Dis. Prot.* **106**, 140–157.

Mieslerova, B., Lebeda, A. and Chetelat, R.T. (2000) Variation in response of wild *Lycopersicon* and *Solanum* sp. against tomato powdery mildew (*Oidium lycopersici*). *J. Phytopathol.* **148**, 303–311.

Money, N.P. (1995) Turgor pressure and the mechanics of fungal penetration. *Can J. Bot.* **73**(Suppl. 1), S96–S102.

Money, N.P. and Howard, R.J. (1996) Confirmation of a link between fungal pigmentation, turgor pressure, and pathogenicity using a new method of turgor measurement. *Fung. Gen. Biol.* **20**, 217–227.

Money, N.P., That, T.-C.C.-T., Frederick, B. and Henson, J.M. (1998) Melanin synthesis is associated with changes in hyphopodial turgor, permeability, and wall rigidity in *Gaeumannomyces graminis* var. *graminis. Fung. Gen. Biol.* **24**, 240–251.

Neshev, G. (1993) Powdery mildew (*Oidium* sp.) on tomatoes in Bulgaria. *Phytoparasitica*, **21**, 339–343.

Olalla, L. and Torés. J.A. (1998) First report of powdery mildew of tomato caused by an *Erysiphe* sp. in Spain. *Plant Dis.* **82**, 592.

Pernezny, K. and Sonoda, R.M. (1998) Powdery mildew of field grown tomatoes in Florida. *Plant Dis.* **82**, 262.

Podila, G.K., Rosen, E., San Franciscom, J.D. and Kolattukudy, P.E. (1995) Targeted secretion of cutinase in *Fusarium solani* f.sp. *pisi* and *Colletotrichum gloeosporioides. Phytopathology*, **85**, 238–242.

Pryce-Jones, E., Carver, T. and Gurr, S.J. (1999) The role of cellulase enzymes and mechanical force in host penetration by *Erysiphe graminis* f.sp. *hordei. Physiol. Mol. Plant Pathol.* **55**, 175–182.

Reuveni, M., Agapov, V. and Reuveni, R. (1994) Induced systemic protection to powdery mildew in cucumber by phosphate and potessium fertilisers. effects of inoculum concentration and post-inoculation treatment. *Can. J. Plant Pathol.* **17**, 247–251.

Reuveni, M., Agapov, V. and Reuveni, R. (1996) Controlling powdery mildew caused by *Sphaerotheca fuliginea* in cucumber by foliar sprays of phosphate and potassium salts. *Crop. Prot.* **15**, 49–53.

Rick, C.M. and Chetelat, R.T. (1995) Utilization of related wild species for tomato improvement. *Acta Hortic.* **412**, 21–38.

Rogers, L.M., Flaishman, M.A. and Kolattukudy, P.E. (1994) Cutinase gene disruption in *Fusarium solani* f. sp. *pisi* decreases its virulence on pea. *Plant Cell*, **6**, 101–109.

Rumbolz, J., Kassemeyer, H.-H., Steinmetz, V., Deising, H.B., Mendgen, K., Mathys, D., Wirttz, S. and Guggenheim, R. (2000) Differentiation of infection structures of the powdery mildew fungus *Uncinula necator. Can. J. Bot.* **78**, 409–421.

Schoenwaedler, S.M. and Burridge, K. (1999) Bidirectional signaling between the cytoskeleton and integrins. *Curr. Opin. Cell Biol.* **11**, 274–286.

Smith, V.L., Douglas, S.M. and LaMondia, J.A. (1997) First report of powdery mildew of tomato caused by an *Erysiphe* sp. in Connecticut. *Plant Dis.* **81**, 229–263.

Vakalounakis, D.J. and Papadakis, A. (1992) Occurrence of a new powdery mildew of greenhouse tomato in Greece, caused by *Erysiphe* sp. *Plant Pathol.* **41**, 372–373.

Verhaar, M.A., Ostergaard, K.K., Hijwegen, T. and Zadoks, J.C. (1997) Preventative and curative applications of *Verticillium lecanii* for biological control of cucumber powdery mildew. *Biocontrol. Sci. Technol.* **7**, 543–551.

Wheeler, I., Holloman, D.W., Longhurst, C. and Green, E. (2000) Quinoxyfen signals a stop to infection by powdery mildews. *The BCPB Conference—Pests and Diseases*, British Crop Protection Council, pp. 841–846.

Whipps, J.M., Budge, S.P. and Fenlon, J.S. (1998) Characteristics and host range of tomato powdery mildew. *Plant Pathol.* **47**, 36–48.

Whitehead, R. (2001) *The UK Pesticide Guide 2001*. Wallingford, UK: CABI Publishing.