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RESEARCH PAPER

Powdery mildew (*Podosphaera xanthii*) resistance in melon is categorized into two types based on inhibition of the infection processes

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Abstract

Physiological races of powdery mildew (*Podosphaera xanthii*) cause different symptoms in eight melon lines. Infection by races 1, 2, and 5 was examined in different melon lines. After a compatible reaction, conidia germination, haustorium initiation from the germ tube, germ tube branching, and sporulation occurred within 12, 24, 48, and 120 h, respectively, and the conidia matured within 240 h. In contrast, type i and ii inhibition were identified through incompatible reactions. The germ tube and haustorium were initiated from conidia, but no germ tube branching occurred in the lines with type i resistance within 48–240 h. In type ii resistance, germ tube branching was observed within 120 h, but no sporulation was observed within 240 h. The number of fluorescing epidermal cells was higher within 24 h in type i, and within 48–120 h in type ii resistance lines than in susceptible lines. Callose accumulation around the haustorium was detected in type ii resistance lines within 48–120 h. This suggests that the rapid hypersensitive response (HR) within 24 h has an important role in the type i response, while HR and callose accumulation in the type ii response occur slowly between 48 and 120 h. Of the resistant lines, PMR 45 and WMR 29 showed a type i incompatible response; the PI 414723 response was entirely type ii; and PMR 5, PI 124112, and MR-1 showed different responses depending on the race. Therefore, the two types of incompatible responses were intermixed in the same germplasm.

Key words: *Cucumis melo* L., *Podosphaera xanthii*, powdery mildew resistance, stages of conidial development, type of incompatible reaction.

Introduction

Worldwide, powdery mildew is a serious disease in melon (*Cucumis melo* L.) throughout major areas of cultivation. Two causal agents of powdery mildew on melon have been identified: *Podosphaera xanthii* (formerly *Sphaerotheca fuliginea* Schlech ex Fr. Poll.) and *Golovinomyces cichoracearum* (formerly *Erysiphe cichoracearum* DC ex Merat). Both fungi have been reported in Europe (Epinat *et al*., 1993; Krístková *et al*., 2004), while many other studies pertain to outbreaks of *P. xanthii* alone (Sowell, 1982; Cohen *et al*., 1984; McCreight *et al*., 1987). In Japan, *P. xanthii* is the major causal fungus of powdery mildew in melon (Hosoya *et al*., 1999, 2000).

Physiological races of *P. xanthii* have been identified in melon. Bardin *et al*. (1999) identified seven races based on the responses of eight different lines of melon: Vedrantais, PMR 45, WMR 29, Edisto 47, PI 414723, PMR 5, PI 124112, and MR-1. Many other races have been identified in Japan (Hosoya *et al*., 1999, 2000) and the Czech Republic (Krístková *et al*., 2004) using these eight lines. A race shift over the cultivation period or in different years has been reported (Sowell, 1982; Thomas *et al*., 1984; Hosoya *et al*., 1999, 2000). It is necessary to introduce durable resistance for many races, such as the barley *Mlo* locus for powdery mildew resistance (Jørgensen, 1992),

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Abbreviation; HR, hypersensitive response.

or rapid and effective resistance to susceptible lines of interest. Accumulating information on the genetics and mechanisms of resistance genes is necessary prior to their introduction.

Much effort has been expended in screening for resistant accessions to powdery mildew in melon. The response of accessions from India, which is a secondary diversification centre of melon (Kitamura, 1951; Mallick and Masui, 1986), to melon powdery mildew has been investigated. Through screening, many resistant accessions have been detected, such as PI 414723 and PI 124112, or bred, such as MR-1 (Thomas, 1986). Resistance has also been introduced into breeding lines, e.g. PMR 45 (Jagger and Scott, 1937), WMR 29 (Bohn *et al*., 1980), and PMR 5 (Pryor *et al*., 1946). Accessions from Spain, another secondary diversification centre, have also been screened, and three resistant and two moderately resistant accessions have been reported (Floris and Alvarez, 1996).

There have been many reports on the inheritance of resistance genes, although some are confusing, in part because the races responsible have not been clearly identified (Pitrat *et al*., 1998). Bardin *et al*. (1999) noted that the confusion arises because melon lines possess several resistance genes.

This confusion can be resolved by identifying each resistance gene genetically or by relating each resistance gene to the interaction between resistant melon lines and races of powdery mildew fungi. Reports on resistance mechanisms (Cohen and Eyal, 1988; Cohen *et al.*, 1990; Floris and Alvarez, 1996; Pérez-García *et al.*, 2001) have addressed races 1 and 2.

In this study, conidial development and the response of melon epidermal cells was observed during the infection of eight different melon lines by three races of powdery mildew fungi. A leaf disc assay was used which has recently become a common method for observing the responses of plants to powdery mildew (Cohen, 1993; Epinat *et al.*, 1993; Bertrand, 2002; Krístková *et al*., 2004). The mechanisms of resistance are discussed with reference to resistance genes.

Materials and methods

Podosphaera xanthii *races used in the inoculation tests*

One isolate each of the three physiological races 1, 2, and 5 (Hosoya *et al*., 1999) of *P. xanthii* was studied. Conidia were obtained from monospore culture and maintained on the cotyledons of melon cultivar Fuyu 3 placed on MS medium (Murashige and Skoog, 1962) in an 8 cm Petri dish under a 16 h light/8 h dark cycle at 24 °C. The conidia were subcultured on new cotyledons every 2 weeks. Two to three weeks after subculture, conidia were used as inocula for the subsequent tests.

Plant materials

Eight genotypes of melon (*Cucumis melo* L.) were investigated: Fuyu 3, PMR 45, WMR 29, Edisto 47, PI 414723, PMR 5, PI 124112, and MR-1. Fuyu 3 is susceptible to *P. xanthii* races 1, 2, and 5. PMR 45 is resistant to race 1, but not to races 2 and 5. WMR 29 and Edisto 47 are resistant to races 1 and 2, but not to race 5. PI 414723, PMR 5, PI 124112, and MR-1 are resistant to all three races. The plants were grown in a growth chamber at 26 °C on a 16 h light/8 h dark cycle.

Inoculation of conidia on leaf segments

A fully expanded leaf obtained from the 4th to the 6th leaf beneath the apex of each plant was cut into pieces for inoculation. The pieces were placed on MS medium in a Petri dish. Conidia were picked up with a brush from a single colony on a cotyledon and inoculated onto each segment. The inoculation was repeated three times for each race. The Petri dishes were incubated at 23 °C on a 16 h light/8 h dark cycle.

Observation of the infection process

Leaves of the melon lines Fuyu 3, PMR 45, WMR 29, Edisto 47, PI 414723, PMR 5, PI 124112, and MR-1 were removed 12, 24, 48, 120, and 240 h after inoculation, and cut into small segments (20–64 mm^2), which were then decolorized in 99% ethanol for 10–15 min at 95 °C and stained with 0.01% trypan blue in equal volumes of lactic acid, glycerine, phenol, and water to observe the infection process. Stained segments were observed under a light microscope at ×200–400 magnification. The spore density was 6–12 mm^{-2} for each segment. Other segments of these melon lines were placed in 0.005% aniline blue in equal volumes of lactic acid, glycerine, phenol, and water, and stored for >2 d under room temperature to observe callose accumulation under fluorescence. Stained segments were examined under fluorescence microscopy at ×200–400 magnification.

Results

The symptoms caused by race 1 of *P. xanthii* were visible to the naked eye after 7–10 days on leaves of the susceptible line Fuyu 3, but not on the resistant lines (Fig. 1). The symptoms of races 2 and 5 were also visible after the same period (data not shown).

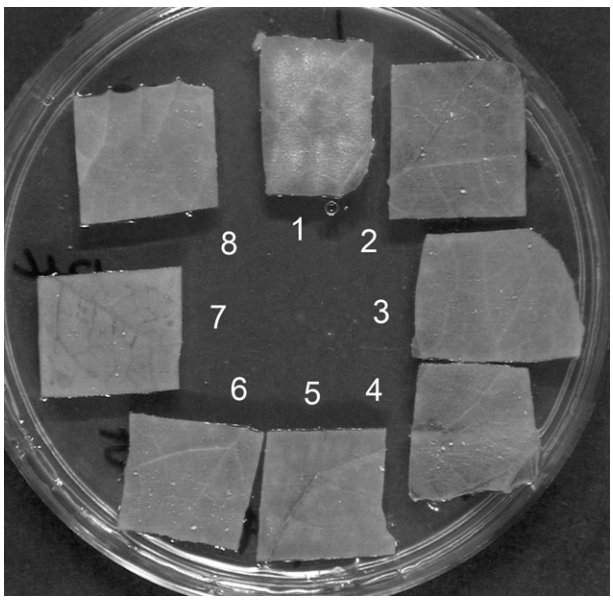

Fig. 1. Responses of eight melon lines to *Podosphaera xanthii* race 1 in the leaf disc test 5 d (120 h) after inoculation. 1, Fuyu 3; 2, PMR 45; 3, WMR 29; 4, Edisto 47; 5, PI 414723; 6, PMR 5; 7, PI 124112; 8, MR-1.

During a compatible reaction on discs inoculated with race 1, the conidia underwent five stages of development (Fig. 2; Table 1): conidia germinated within 12 h of inoculation [stage 1: Fig. 2 (1)], formed a haustorium within 24 h [stage 2: Fig. 2 (2)], and began germ tube branching and hyphae elongation within 48 h [stage 3: Fig. 2 (3)]. New conidia were initiated within 120 h [stage 4: Fig. 2 (4)], and the conidia matured within 240 h [stage 5: Fig. 2 (5)]. These results were generalized to other races undergoing a compatible reaction. Well-developed haustoria seemed to consist of the haustorium body surrounded by numerous haustorial lobes and separated from epidermal cells by an extrahaustorial membrane within 120–240 h of the compatible reaction [Fig. 3 (1)].

For the resistant lines, the incompatible reaction was categorized into two types, based on the stage of conidial development.

On WMR 29 inoculated with race 1, conidia grew to stage 2 [Fig. 2 (1 and 2), Table 1]. Conidia germination and haustorium formation occurred within 24 h, as in the compatible reaction, but no or very little (0.3% of 300 conidia counted) germ tube branching was observed, even after 48 and 120 h of inoculation. The haustoria in incompatible reactions seem to be undeveloped; no haustorial

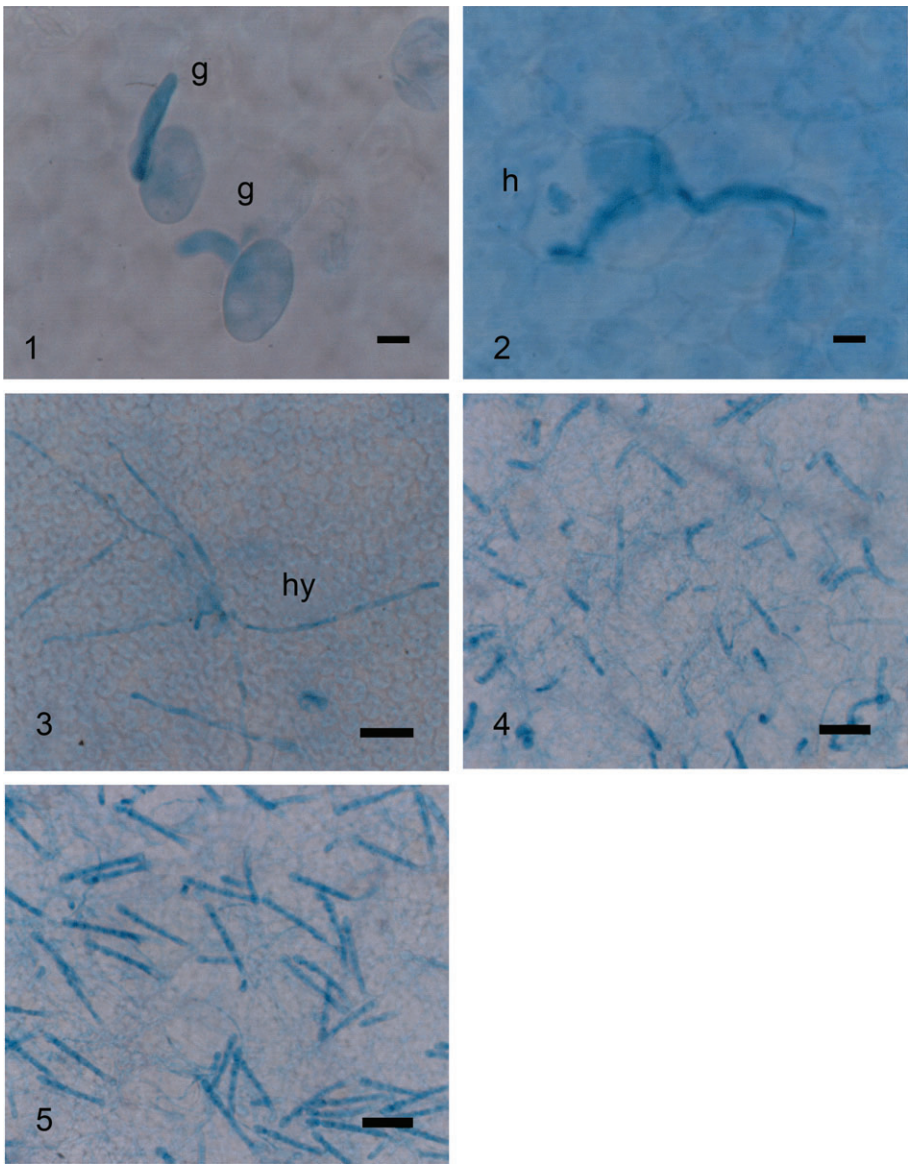

Fig. 2. Histological observation of *Podosphaera xanthii* race 1 infection on leaf discs of Fuyu 3. (1) Germ tube initiation 12 h after inoculation. (2) Haustoria formation within 24 h of inoculation. (3) Hyphal development within 48 h after inoculation. (4) New conidia initiation 120 h after inoculation. (5) Mature conidia 240 h after inoculation. G, germ tube; h, haustorium; hy, hyphae. The scale bars represent 10 μm in 1 and 2, 50 μm in 3, and 100 μm in 4 and 5.

Table 1. *Stages of infection of powdery mildew fungus* Podosphaera xanthii *race 1 after conidia inoculation onto susceptible and resistant melon lines*

The numbers given indicate the stages of infection as in Fig. 2.

Lines	Response[a]	Time after inoculation (h)					Type of resistance[a]
		12	24	48	120	240	
Fuyu 3	S	1	2	3	4	5	
PMR 45, WMR 29, Edisto 47, PMR 5, PI 124112, MR-1	R	1	2	2	2	2	i
PI 414723,	R	1	2	3	3	3	ii

[a] R, resistant; S, susbceptible; i, type i resistance; ii, type ii resistance.

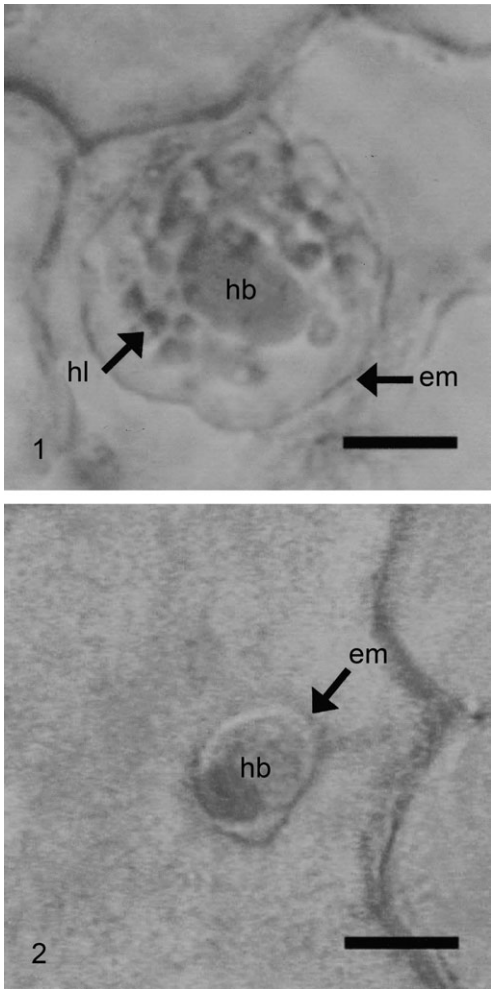

Fig. 3. Haustorium of *Podosphaera xanthii* race 1 on susceptible and type i resistance leaf discs 120 h after inoculation, viewed by differential interference contrast microscopy. (1) Well-developed haustorium of Fuyu 3. (2) Undeveloped haustorium of WMR 29. Scale bar=10 μm. hb, Haustorial body; hl, haustorial lobes; em, extrahaustorial membrane.

lobes were visible around the haustorial body [Fig. 3 (2)] within 120 h. This type of resistance was denoted type i. Type i resistance behaviour was also observed for the following incompatible reactions: PMR 45 with race 1, although few conidia (3.6% of 300 conidia counted) elongate after 120 h; WMR 29 with race 2, Edisto 47 with races 1 and 2, PMR 5 with race 1, PI 124112 with races 1 and 2, and MR-1 with races 1 and 2 showed an incompatible reaction of type i resistance (Table 2).

On PI 414723 inoculated with race 1, the conidia grew to stage 3 [Fig. 2 (1–3), Table 1]. Conidia germination and haustorium formation were observed within 24 h, and some inoculates grew second germ tubes and hyphae within 48–120 h (59% of 300 conidia counted). However, no sporulation was observed on the leaves within 120 h. The haustoria seemed to develop slowly, as haustorial lobes were few or lacking within 120 h (data not shown). This type of resistance was denoted type ii. Type ii resistance behaviour was also observed for the following incompatible reactions: PI 414723 with races 2 and 5, PMR 5 with races 2 and 5, PI 124112 with race 5, and MR-1 with race 5 (Table 2).

Callose accumulation and autofluorescence was observed in epidermal cells (Fig. 4). To clarify the different responses of epidermal cells for each compatible or incompatible reaction, the amount of fluorescence cells with haustoria was counted 24, 48, and 120 h after inoculation for compatible line Fuyu 3, type i resistance line WMR 29, and type ii resistance line PI 414723 inoculated with race 1 (Fig. 5). No fluorescing cells were observed within 12 h on any melon leaves. In the compatible reaction of Fuyu 3, a few fluorescing epidermal cells were observed within 24 h when conidia formed a haustorium (stage 2). The number of fluorescing cells was almost constant with conidial development, followed by germ tube branching and hyphal elongation within 48 h (stage 3) and 120 h (stage 4). Callose accumulation around the haustorial neck was observed within 120 h, but not in all haustoria.

Conversely, there were significantly more fluorescing epidermal cells with a haustorium or adjacent cell within 24 h on type i resistance lines with race 1 on WMR 29 than on Fuyu 3 [Figs 4 (1) and 5]. This difference seems to be most important between compatible and incompatible reactions of type i. These fluorescing cells were visible on every compatible reaction with type i resistant lines for races 1 and 2 within 24–48 h. Accumulations of callose around the haustorial neck were detected within 24–48 h. The number of fluorescing cell was constantly large within 24–48 h.

Table 2. *Grouping of resistant melon lines by the types of resistance to three races of* Podosphaera xanthii

Lines	*P. xanthii* races			Group
	1	2	5	
Fuyu 3	S[a]	S	S	
PMR 45	i	S	S	1
WMR 29	i	i	S	1
Edisto 47	i	i	S	1
PI 414723	ii	ii	ii	2
PMR 5	i	ii	ii	3
PI 124112	i	i	ii	3
MR-1	i	i	ii	3

[a] S, susceptible; i, type i, resistance; ii, type ii resistance.

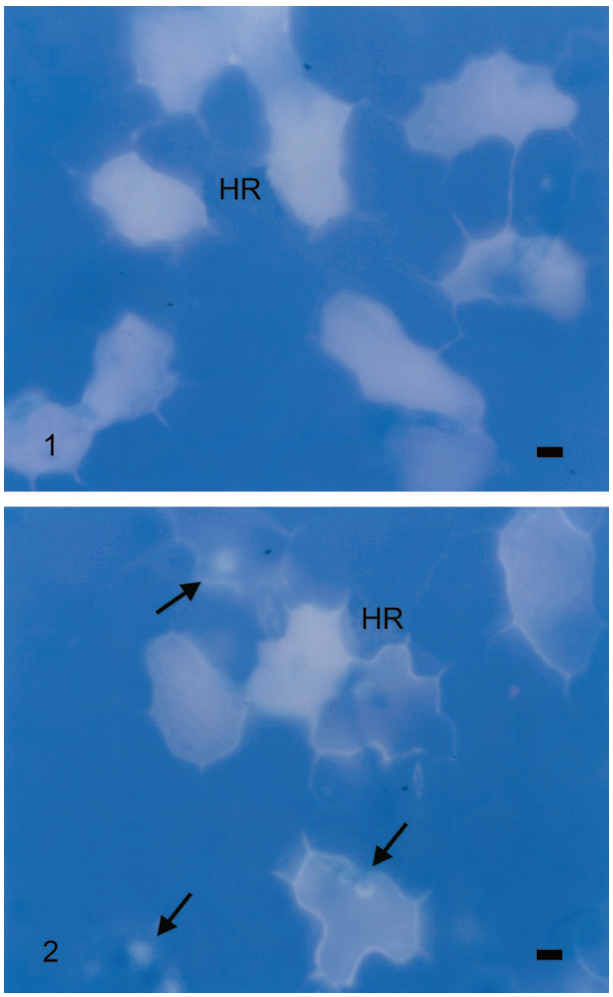

Fig. 4. Responses of epidermal cells of the different melon lines to *Podosphaera xanthii* race 1. (1) Fluorescing WMR 29 epidermal cells 24 h after inoculation. (2) PI 414723 epidermal cells fluorescing and showing callose accumulation 48 h after inoculation. F, fluorescing cell. Arrow indicates callose accumulation. Scale bars=10 μm.

In the type ii resistance line PI 414723 with race 1, fluorescing epidermal cells were observed within 24 h though the number of fluorescing cells was smaller than for the type i incompatible reaction on WMR 29 (Fig. 5).

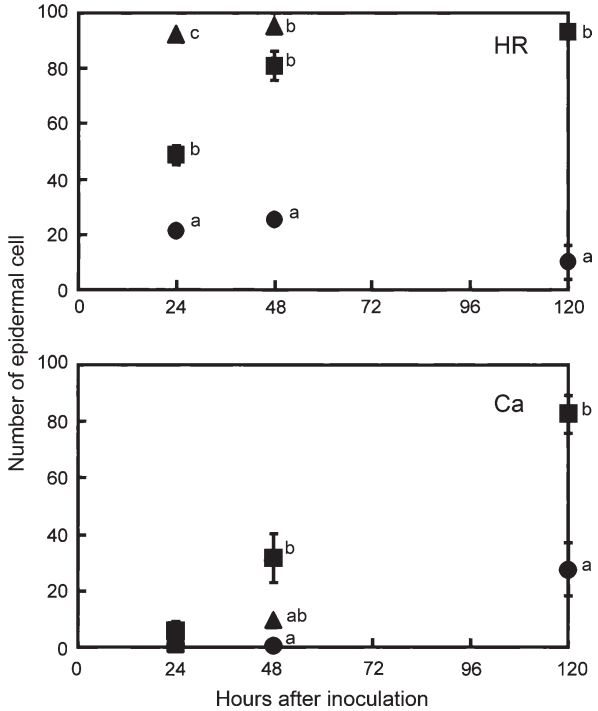

Fig. 5. Number of epidermal cells displaying susceptible lines (filled circles), type i resistance lines (filled triangles) and type ii resistance lines (filled squares) beneath haustoria on melon lines inoculated with race 1 of *Podosphaera xanthii* 24, 48, and 120 h after inoculation. HR, number of fluorescing cells; Ca, number of cells with callose accumulation. Three segments were observed each hour; 100 conidia were counted for each segment. Each value is the mean ±SE.

The number of fluorescing cells increased within 48 h and was constant within 120 h. Callose accumulation around haustoria increased within 48 [Fig. 4 (2)] to 120 h. The callose accumulation was larger than in Fuyu 3 and WMR 29 during 48–120 h. Callose accumulation and fluorescing cells were visible on other lines that showed type ii resistance with race 5 within 48–120 h.

The resistant lines were grouped into three categories, based on the combination of the type of resistance against the inoculated races (Table 2). Group 1 included PMR 45, WMR 29, and Edisto 47, which showed only type i resistance for every avirulent race. Group 2 comprised PI 414723, which showed only type ii resistance to each race. PMR 5 responded to race 1 with type i resistance, and to races 2 and 5 with type ii resistance. PI 124112 and MR-1 showed type i resistance to races 1 and 2, and type ii resistance to race 5. These three lines were placed in group 3.

Discussion

Conidial development of powdery mildew fungus (*P. xanthii*) was observed through compatible and incompatible reactions. We were able to categorize the incompatible reaction against powdery mildew in melon histologically

into two types, based on hyphal elongation and sporulation: types i and ii (Fig. 6). In the compatible reaction, five stages of the infection process were observed. In the case of the incompatible reaction with type i resistance, conidial development was inhibited at the stage of germ tube germination within 24–48 h after haustorium formation, but they were undeveloped and without haustorial lobes. In the case of the incompatible reaction with type ii resistance, conidial development was restricted at the stage of new conidia initiation within 48–120 h after hyphal elongation. The development of conidia was the same on whole plant leaves and leaf segments (data not shown).

The main part of the type i incompatible reaction seemed to occur before 24–48 h after inoculation. The number of HR cells was significantly higher between 24 and 48 h. In the case of accumulation of pathogenesis-related proteins in resistance line PMR 6 showing a type i-like response, β-1,3-glucanases accumulated within 24–48 h of inoculation (Rivera *et al.*, 2002). This suggests that the early event within 24–48 h is important for type i resistance.

The mechanisms of resistance to powdery mildew on Indian lines, PI 124111, PI 124112, and PMR 5 resistant to race 2 (Cohen and Eyal, 1988) and PI 124111F, PI 124112, PMR 45, and PMR 6 resistant to races 1 and 2 (Cohen *et al*., 1990), were assumed by observing the responses of epidermal cells. In these lines, conidia development was suppressed at the time of germ tube formation, but not at the time of hyphal elongation, as in the incompatible reaction of type i lines in our study; this suggested that the suppression was associated with hypersensitive cell death (Cohen and Eyal, 1988), callose accumulation, and liginification (Cohen *et al*., 1990) of epidermal cells. In our study, the cells with callose accumulation were few and localized around the haustorial neck visible under the light microscope at 24 and 48 h. These phenomena suggest that type i resistance is conferred more by rapid HR than by callose accumulation. Conversely, the findings in PMR 5 differed markedly from the observations of Cohen and Eyal (1988). It is likely that the physiology of our race 2 and their race 2 is different; we therefore need to compare these races under the same conditions.

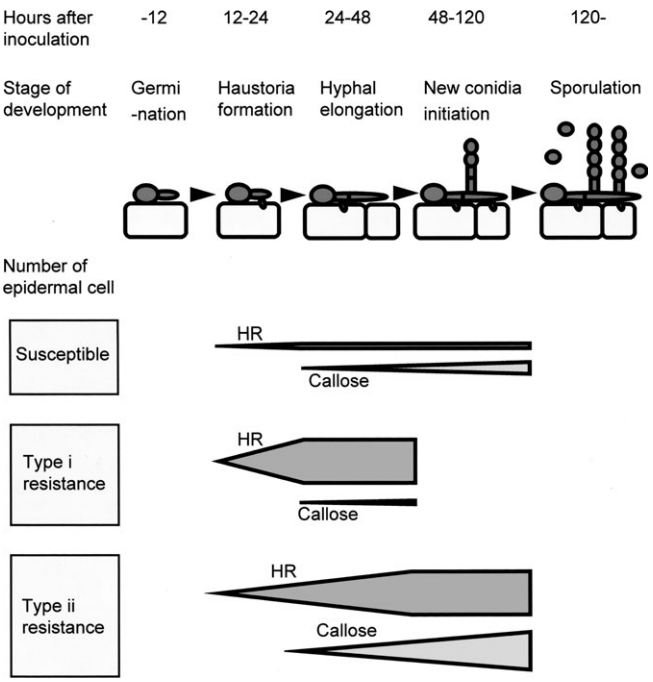

Fig. 6. A schematic representation of the process of *Podosphaera xanthii* infection on melon leaves and the steps leading to the initiation of different types of resistance to powdery mildew in melon.

The rate of conidial development was higher on PMR 45 than other typical type i lines such as WMR 29, although the rate on PMR 45 was much lower than the type ii incompatible reaction on PI 414723 with race 1 in 48 h. The number of HRs was the same in WMR 29 and PMR 45 at 24 and 48 h, but the intensity of fluorescence was faint on PMR 45 (data not shown). The resistance gene locus *Mla* for powdery mildew (*Erysiphe graminis* f.sp. *hordei*) in barley has 28 alleles (Jørgensen, 1994), and different alleles induce resistant responses of differing intensity, which are correlated with the timing of hypersensitive cell death (Boyd *et al*., 1995). As with the *Mla* locus, different genes in lines PMR 45 and WMR 29 or other type i lines probably cause the different timing or intensity of HR, while both have type i resistance.

Fluorescing cells and callose accumulation were also observed on type ii resistance lines, although the timing of these events differed from that in type i resistance lines. Floris and Alvarez (1996) attempted to screen the resistance mechanisms of the Spanish lines Negro, Moscatel Grade, and Amarillo, and found that the hyphae elongate, but that sporulation is inhibited in these Spanish resistant lines like the type ii incompatible reaction in this study. They also compared the mechanisms of resistance in Indian lines PI 124111 (revealing type i-like resistance) and Spanish lines (type ii-like resistance) by observing the fluorescence of epidermal cells, and concluded that the mechanisms differed. This suggests that the resistance mechanisms differ in Indian lines with the HR and in Spanish lines without the HR, although the resistance process in the Spanish lines is likely to be type ii resistance.

The differences in the stage of fungus development seen in incompatible reactions of types i and ii resistance seem to be due to differences in the timing of the HR. On the other hand, callose accumulation was observed and continued to increase within 120 h in a type ii incompatible reaction, while little accumulation was observed in a type i incompatible reaction on WMR 29 even within 48 h. Therefore, the mechanism of type ii resistance is much more complex compared with type i; several reactions seemed to happen simultaneously in the type ii incompatible reaction and occur slowly compared with the type i incompatible reaction. Determination of the reactions to

powdery mildew of F_1 hybrids of type i and type ii lines will provide some information on the difference in mechanisms between these two types.

There have been many reports on the resistance genes for races 1 and 2 (Kenigsbuch and Cohen, 1992; Epinat *et al*., 1993; Floris and Alvarez, 1996). PMR 45 and WMR 29 have a *Pm-A* resistance gene for *P. xanthii* race 1 (Epinat *et al*., 1993). Our results suggest that locus *Pm-A* controls type i resistance. PI 124112 has *Pm-5* (Kenigsbuch and Cohen, 1992) for *P. xanthii* race 1. This gene also seems to control type i resistance. PI 414723 has the *S6* gene for resistance to race 1 (Pitrat *et al*., 1998), and this gene seems to control type ii resistance. The relationships between the two types of resistance will be clarified by genetic analyses of the resistance genes controlling each type.

PMR 5, PI 124112, and MR-1 resist powdery mildew using both types of resistance. These two resistance processes correspond to different fungus races, suggesting that there are at least two types of gene-mediated resistance to powdery mildew in the same germplasm.

PI 124112 appears to possess three genes for resistance to *P. xanthii* (Bardin *et al*., 1999). *Locus A* is the gene for resistance to races 1 and 2, *locus B* is the gene for resistance to races 1, 2, and 4, and *locus C* is the gene for resistance to races 1, 2, 4, and 5. The actions of these genes support our hypothesis that *locus A* and *locus B* confer resistance to races 1 and 2 by controlling type i resistance, and *locus C* confers resistance to race 5 by controlling type ii resistance. More information will be obtained by observing the resistance mechanisms of *locus C* for races 1, 2, and 4.

In conclusion, incompatible reactions with powdery mildew on different melon lines and the resistance mechanisms involved were investigated. Two types of incompatible reaction are sometime maintained in the same germplasm. The HR appears to play an important role in one type, while callose accumulation is the underlying mechanism for the other resistance type. The analysis of resistance mechanisms and their genetics will provide useful information on race determination, virulence shifting, and the durability of resistance to *P. xanthii*.

Acknowledgements

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