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A Light and Electron Microscopical Study of Compatible and Incompatible Interactions between *Phytophthora capsici* and Tomato (*Lycopersicon esculentum*)

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Phytophthora capsici 균주와 토마토의 친화적, 불친화적 상호작용에 대한 광학 및 전자현미경적 연구

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ABSTRACT : Stem tissues of tomato plants (cv. Kwangyang) inoculated with *Phytophthora capsici* were examined by light and electron microscopy to compare early cytological differences between compatible and incompatible interactions of tomatoes with the fungus. Twenty four hours after inoculation, the compatible isolate S 197 colonized severely the epidermis, cortex, and xylem vessels of stem tissue, whereas only few fungal cells colonized the stem tissues inoculated with the incompatible isolate CBS 178.26. Fragmented plasma membrane, distorted chloroplast, degraded cell wall, remnants of host cytoplasm were early ultrastructural features of the damaged host cell observed both in the compatible and incompatible interactions, although damages were less severe in the incompatible interaction. In the incompatible interaction, a number of vesicles were distributed in the space between fungal cell walls and plasma membrane. The degradation of host cell walls by *P. capsici* was more pronounced in the compatible than the incompatible interactions. The incompatible interactions of tomato cells with *P. capsici* were characterized by formation of host cell wall apposition in the cortical parenchyma cells, indicating that the apposition of electron-dense material from the host cell walls may function as a plant defense reaction to the fungus. The fungal cells encased by wall appositions had abnormal cytoplasm and separated plasma membranes. The haustorium which formed from the fungal hyphae did not further penetrate through the host wall apposition and cytoplasmic aggregation, especially in the incompatible reactions. In contrast, the haustorium of the compatible isolate S 197 was not encased by wall appositions.

***Key words* : *Phytophthora capsici*, tomato, ultrastructural differences.**

Phytophthora capsici Leonian, one of the most destructive soilborne pathogens, is mainly pathogenic on solanaceae species including tomato (*Lycopersicon esculentum* Mill.) and pepper (*Capsicum annuum* L.)(26). The pathogen causes crown rot, root rot, fruit rot, and foliar blight in tomato plants (9, 24, 26). In our previous study, typical dark brown lesions were produced in tomato stems inoculated with isolates of *P. capsici* (17). In particular, the tomato cultivar 'Kwangyang' significantly differed in susceptibility to the isolates of *P. capsici*, when inoculated by using stem-wound techniques. The isolate S 197 was highly virulent to the cultivar Kwangyang, whereas the isolate CBS 178.26 was less virulent or avirulent at all stages of plants. Tomato plants of the cultivar Kwangyang inoculated with compatible (susceptible) to the isolate S 197 had a brownish stem-discoloration rapidly extending upward from the wound-inoculated site accompanied by a sudden wilt of the entire plant, defoliation, or damping-off

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in juvenile plants. In contrast, the tomato plants inoculated with incompatible (resistant) to the isolate CBS 178.26 were symptomless or had a hypersensitive, dark-brown girdling rot that developed very slowly on the stems.

Some cytological and ultrastructural studies have been made of the interaction of pepper plants with *P. capsici* (13, 14, 18, 21), but there has been little information about the fine structures of tomato-*P. capsici* interactions. In our previous investigations, degeneration of cell organelles, dissolution of host cell walls, and deposition of papilla-like material were observed in roots and stems of pepper infected with *P. capsici* by using light and electron microscopy (14, 21). Our ultrastructural study of the mode of action of metalaxyl in pepper against *P. capsici* also demonstrated the formation of encasements around haustoria in these plants as a host reaction to metalaxyl (12). Several workers also have investigated differential interactions between *Phytophthora* spp. and host plants with specific resistance genes (11, 12, 22, 28). In the *P. megasperma*-soybean system (28), host cells at and around infection sites died rapidly and wall appositions occurred with a high frequency in the incompatible interaction. Host cell necrosis and callose-like encasements were also found in the resistant potato tissues infected with *P. infestans* (11, 22).

Formation of wall appositions in plant cells has been known to be induced by various stresses such as pathogens, wound, and chemical substances (1, 13). The wall apposition may function as chemical or physical barriers against pathogen attack. Detailed studies of wall appositions have been carried out concerning the sequence of events in fungal penetration and host response to *Phytophthora* spp. (6, 11~14, 28).

The present study was carried out to examine the cytological and ultrastructural changes that occur in tomato stems and *P. capsici* in both compatible and incompatible reactions. In particular, we investigated the early stage in the infection of tomato stems by *P. capsici*, including entry through the epidermis and cortical tissues and penetration and colonization of stem tissue.

MATERIALS AND METHODS

Plant, fungus, and inoculation. The tomato cultivar 'Kwangyang' was selected for this study to compare both a compatible and incompatible response of *P. capsici* on the same cultivar. Six seeds of tomato cultivar Kwangyang were sown in a plastic pot (5×15×10 cm) containing a mixture of steam-sterilized loam soil, sand, and peat (4:4:2, v/v/v). Tomato plants were raised in a growth room under 16 h/day illumination at 25± 2℃.

Two isolates of *P. capsici* were used to observe ultrastructural differences between compatible and incompatible tomato-*P. capsici* interactions. The isolate S 197 which caused compatible response to the tomato cultivar Kwangyang (17) was provided by E. Pochard (Plant Pathology Station, Institut National de la Recherche Agronomique at Montfavet, France). The isolate CBS 178.26 incompatible to the cultivar Kwangyang was also obtained from the Centraal bureau voor Schimmelcultures at Baarn, the Netherlands. The two isolates were cultured on oatmeal agar at 28℃ for 7 days and then induced to sporulate under fluorescent light. Sterilized tap water was poured into the plates to harvest sporangia. Sporangial suspension was chilled at 4℃ for 30~60 min to release zoospores and then decanted through four layers of cheesecloth. To use as an inoculum, zoospore suspension was adjusted to 1×10^5/ml with sterilized tap water.

At the two-leaf stage, tomato seedlings were inoculated by zoospore suspension (1×10^5/ml) of each of *P. capsici* isolates. After prickling stems of the tomato plants by using several needles, sterile cotton soaked in zoospore suspension was placed on the wounded sites of the stems. The inoculated sites were then covered with plastic tape to maintain a moist condition.

Electron microscopy. One-mm pieces of infected stems, 1 mm apart from the wound-inoculated sites, were excised in 0.1 M cacodylate buffer (pH 7.2) 24 h after inoculation with *P. capsici*. The stem tissues were transferred immediately to the fixative of 2.5% glutaraldehyde and 2% paraformaldehyde in 0.1 M cacodylate buffer, and infiltrated under vacuum for 4 h at 4℃. After washing in four changes (30 min) of the buffer, the tissues were postfixed in 1 % osmium tetroxide in the same buffer for 2.5 h at room temperature. After rinsing twice with buffer and distilled water, the tissues were dehydrated in an acetone series, embedded in 'Spurr' medium in flat rubber moulds, and then cured at 70℃ for 24 h.

Ultrathin sections were cut with glass knives on a Sorvall MT-2 ultramicrotome, and placed on Formvar-coated nickel and uncoated copper grids. Penetration sites and the invaded area in the thick sections were also examined under an Olympus light microscope and then photographed by using Fuji minicopy II microfilm (ASA 25). The ultrathin sections were stained for 2 h in 4% uranyl acetate, washed in distilled water, and poststained for 5 min in Reynold's (25) lead citrate before examining with a Jeol JEM 100 CX-II transmission electron microscope.

RESULTS

Twenty four hours after inoculation of pepper stems with zoospores of *P. capsici*, fungal hyphae grew intercellularly and intracellularly in the stem tissue. In compatible or incompatible interactions, fungal invasion induced various host cell reactions. The compatible isolate S 197 colonized the epidermis, cortex, and xylem vessels of stem tissue (Fig. 1 a). Severely damaged cortical layer became shrunken and darkly stained. Along with extended fungal colonization, a large number of fungal cells were observed in the transverse section of the compatible interactions. In the incompatible interaction, isolate CBS 178.26 rarely invaded vascular steles. The fungal colonization usually was restricted to the cortex (Fig. 1b). There was much less damage to epidermal cells in the incompatible interaction. In some incompatible responses, epidermal layers of the stem tissue became necrotic and dark-stained (Fig. 1c). Only few fungal cells colonized the stem tissue inoculated with the incompatible isolate CBS 178.26.

Ultrastructure of fungal hyphae and infection sites in compatible interactions. Disorganization of host cell cytoplasm and the destruction of plasma membranes and cell organelles in the compatible interaction were clearly observed at the ultrastructural level (Fig. 2). Penetrating fungal cells grew intercellularly and intracellularly in the cortical parenchyma cells of tomato stems. Host plasma membrane separated from the host cell wall was considerably disrupted and fragmented (Fig. 2a). The parenchyma cell wall was partially degraded or thinned. Distorted chloroplasts, mitochondria, and remnants of host cell cytoplasm were scattered within the infected cortical parenchyma cells (Fig. 2b).

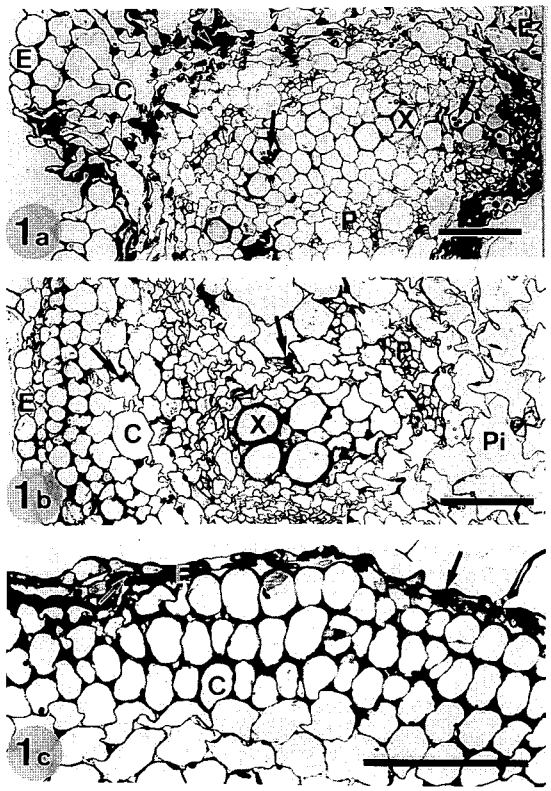


Fig. 1. Light micrographs of transverse sections of stems from tomato seedlings 24 h after inoculation with compatible (S 197) or incompatible (CBS 178.26) isolates of Phytophthora capsici. (1a) compatible interaction. The epidermis, cortex, and xylem of stem tissue are severely and evenly colonized by the fungus (arrows). Some parts of the tissue are also severely collapsed due to the fungal invasion (arrow heads). (1b) Incompatible interaction. Stem tissue is slightly infected by the fungus (arrows) and the vascular stele is almost free of fungal colonization. (1c) Incompatible interaction. Note the necrotic dark-stained epidermis of stem tissue (arrows). Bars = 50 μm. E, epidermis; C, cortex; X, xylem element; Pi, pith.

In the compatible reactions, no cell wall appositions were formed on the side opposite to points of contact between the fungus and the host (Fig. 3). However, host cell wall was greatly dissolved by the fungal cell in the intercellular space, thus resulting in the plasmolysis of host cell (Fig. 3a). Normal cell organelles such as mitochondria, endoplasmic reticulum, vacuoles, ribosomes, and lipid-like bodies were visible in the hyphal cell. In the reaction of xylem parenchyma, numerous electron-dense, os-

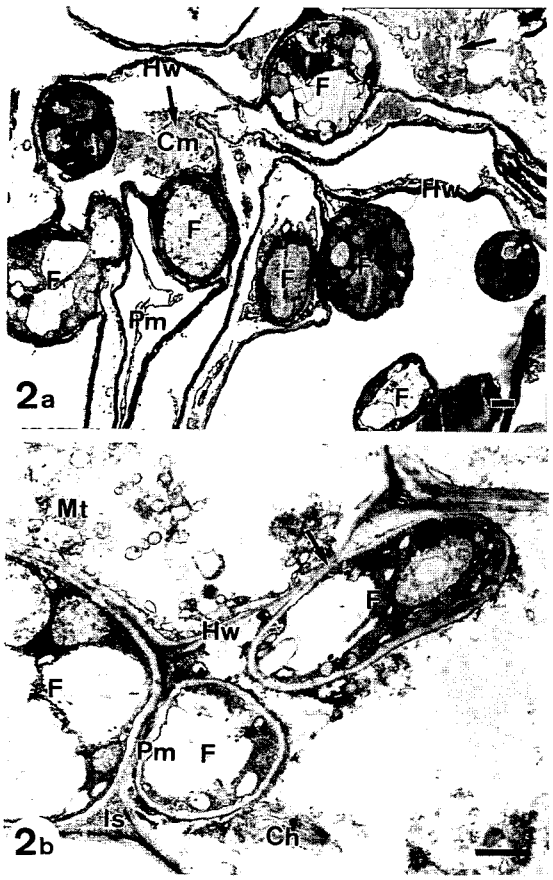


Fig. 2. Electron micrographs of transverse sections of cortical parenchyma cells of stems from tomato seedlings 24 h after inoculation with compatible (S 197) isolate of *Phytophthora capsici*. (2a) Host cells degraded by intercellular and intracellular colonizations of fungal cells. Host plasma membrane is separated from host cell wall. Note the disrupted cytoplasmic material (arrows). (2b) Host cell wall (arrow) is dissolved and becomes thin at the contact site of the fungal cell accompanying by distorted chloroplast and remnants of host cytoplasm. Bars = 1 μm. Cm, cytoplasmic material; F, fungus; Hw, host cell wall; Pm, plasma membrane; Ch, chloroplast; Mt, mitochondria.

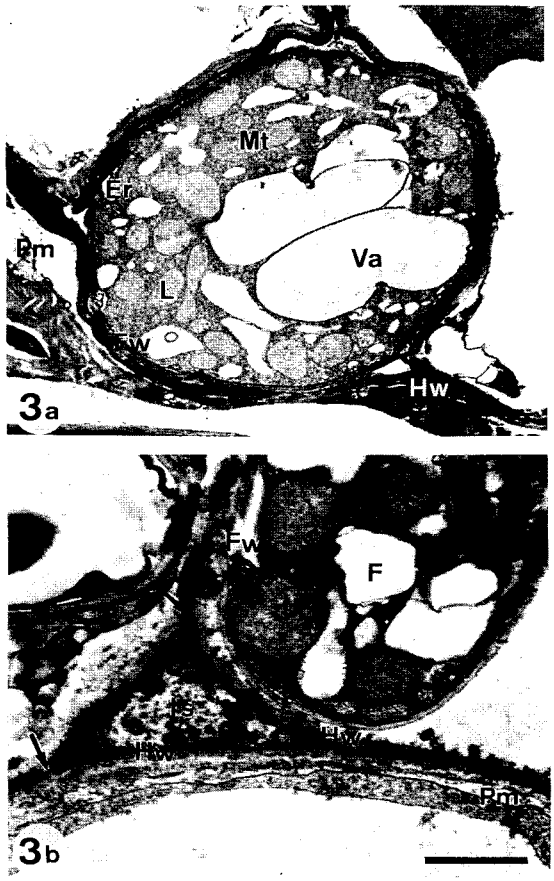


Fig. 3. Electron micrographs of host cell wall reactions in stem tissues from tomato seedlings 24 h after inoculation with compatible (S 197) isolate of *Phytophthora capsici*. (3a) Host cell wall degraded by the fungal cell in the intercellular space and host-cell plasma membrane fragmented. The fungal cell contains numerous intact mitochodria, endoplasmic reticulum, and vacuoles. Remaining spaces of the fungal cell are filled with ribosomes and lipid-like bodies. (3b) Host cell wall dissolved by the invading fungal cell (arrows). Numerous electron-dense, osmiophilic particles are scattered on or around the host cell wall and in the intercellular space. Bars = 1 μm. Er, endoplasmic reticulum; Fw, fungal cell wall; Is, intercellular space; Hw, host cell wall; L, lipid-like body; Va, vacuole.

miophilic particles were distributed in the intercellular space, and on or around the host cell wall dissolved by the invading fungal cell (Fig. 3b).

In the interaction with the compatible isolate S 197 in the cortical parenchyma cells, hyphae frequently grew within host cell walls or through the middle lamella between adjoining cells (Fig. 4a), or in the intercellular space (Fig. 4b). The fungal hyphae which penetrated the cortical parenchyma cells formed haustoria, highly vacuolated or devoids of contents. The haustorium perforated the host cell wall, but was not encased by wall apposition (Fig. 4 b). The host cell cytoplasm was completely disorganized by the infecting fungus.

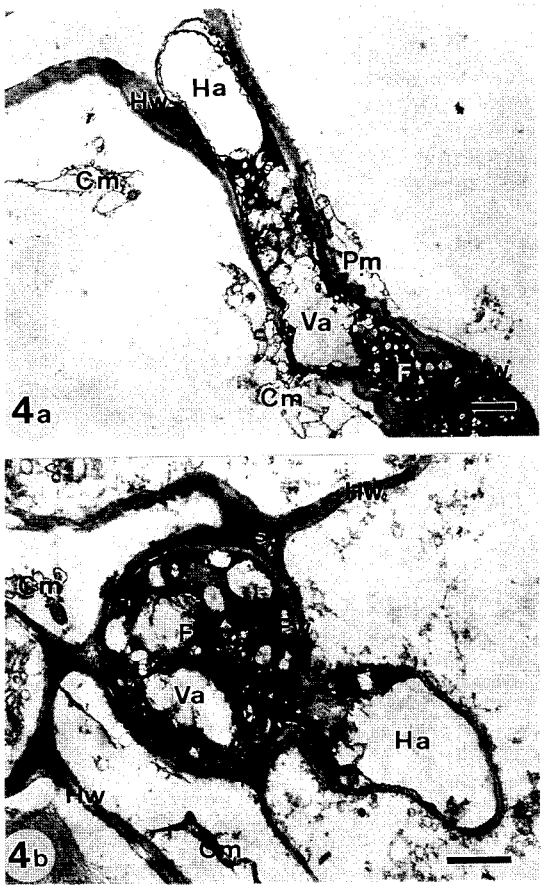


Fig. 4. Electron micrographs of haustorium formation in stem tissues from tomato seedlings 24 h after inoculation with compatible (S 197) isolate of *Phytophthora capsici*. (4a) Host cell wall swollen and host plasma membrane fragmented by hyphal growth through the middle lamella. (4b) Host parenchyma cell wall is perforated by the well-developed haustorium. Distinguishable wall apposition is not observable. Bars=1 μm. Cm, cytoplasmic material; Ha, haustorium; Hw, host cell wall; Pm, plasma membrane; Va, vacuole; Is, intercellular space.

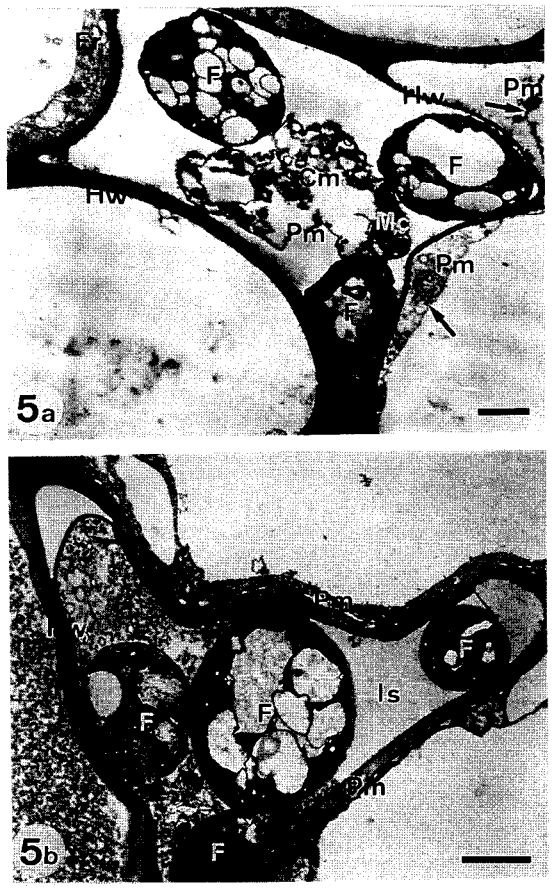
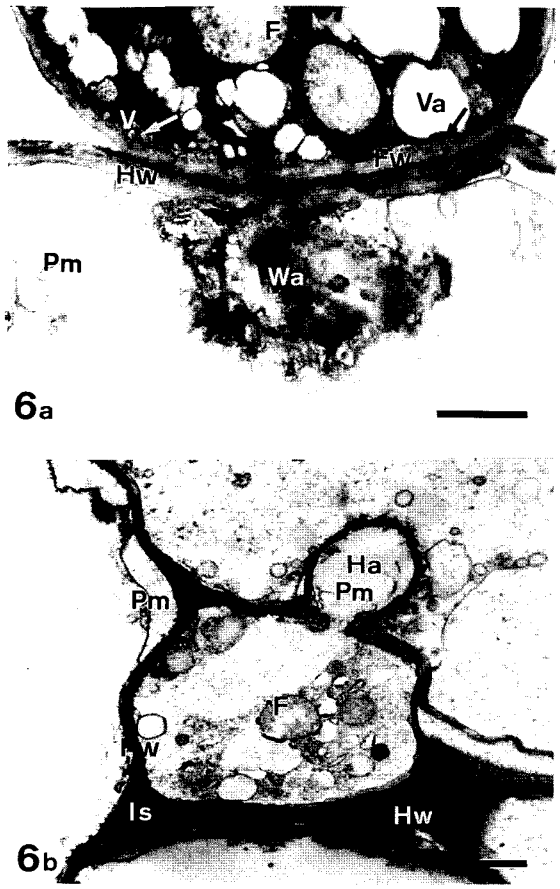


Fig. 5. Electron micrographs of transverse sections of cortical parenchyma cells of stems from tomato seedlings 24 h after inoculation with incompatible (CBS 178.26) isolate of *Phytophthora capsici*. (5a) Host cell colonized intracellularly by fungal cells. Note the remnants of host cytoplasm and plasma membranes separated from the intense dark-staining host cell wall (arrows). (5b) Intercellular space heavily colonized by fungal cells. Electron-dense particles are seen in the intercellular space and host cell lumen. The intense dark-staining cytoplasm and numerous vacuoles are filled in the fungal cell. Bars=1 μm. Abbreviations used were same as those in Fig. 4.

Ultrastructure of fungal hyphae and infection sites in incompatible interactions. In the incompatible interactions, ultrastructural changes in fungal hyphae and infection sites were distinct 24 h after inoculation with the incompatible isolate CBS 178.26. The fungal hyphae growing in the cell lumen or in the intercellular space became dark-stained with numerous vacuoles (Fig. 5a, b). The host cell walls were not heavily damaged by fungal infection. The remnants of host cytoplasm with no recognizable organelles were scattered in the cell lumen and the plasma membranes were separated and fragmented (Fig. 5a). Electron-dense material was seen in the intercellular space and host cell lumen.

At the host-parasite interface of incompatible isolate CBS 178.26 in tomato stems, cell wall apposition or cytoplasmic aggregation ocurred distinctly below the host cell walls in close contact with the



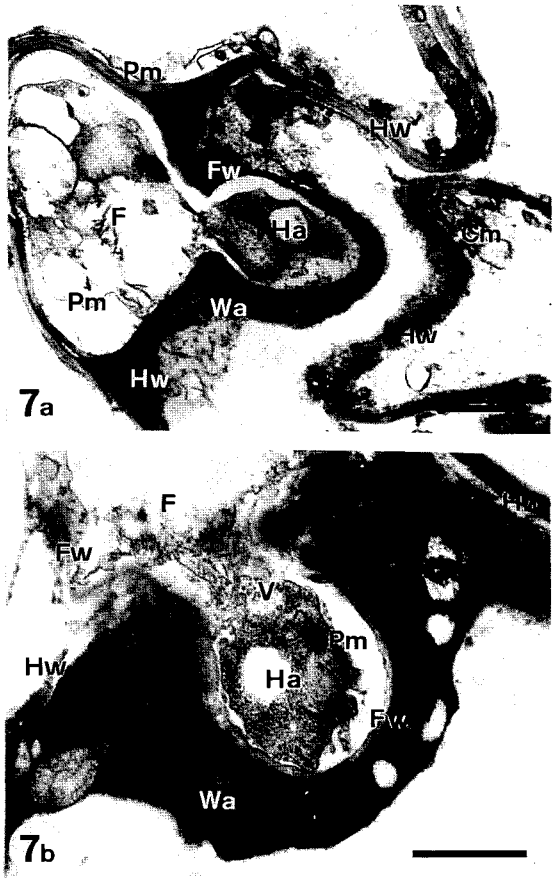


Fig. 6. Electron micrographs of host cell wall reactions in stem tissues from tomato seedlings 24 h after inoculation with incompatible (CBS 178.26) isolate of *Phytophthora capsici*. (6a) Host cell wall dissolved by the fungal contact. Osmiophilic, amorphous substances are dispersed below the host cell wall intimately in contact with the fungal cell. The fungal plasma membrane is invaginated. The space between fungal cell wall and plasma membrane is filled with small vesicles (arrows). (6b) The cell wall apposition on the opposite side of the host cell wall intimately contacted by the fungal cell. Fungal cell lumen is palely stained. Bars=1 μm. F, fungus; Fw, fungal cell wall; Hw, host cell wall; Pm, plasma membrane; Wa, wall apposition; Va, vacuole; Is, intercellular space.

Fig. 7. Electron micrographs of haustorium formation in cortical parenchyma cells of stems from tomato seedlings 24 h after inoculation with incompatible (CBS 178.26) isolate of *Phytophthora capsici*. (7a) Haustorium encased by densely stained host cell wall apposition. Fungal cell organelles are disorganized. Neighboring host cells not directly contacted by the fungus are also plasmolyzed and host plasma membranes are fragmented. (7b) Haustorium is heavily surrounded by osmiophilic wall apposition with electron translucent areas. Fungal plasma membrane is fully separated from the fungal cell wall. Note numerous vesicles in the fungal cytoplasm. Bars=1 μm. Abbreviations used were same as those in Fig. 6.

fungal cells (Fig. 6a, b). The space between fungal cell wall and plasma membrane was filled with numerous small vesicles (Fig. 6a). Palely stained fungal cell lumen contained some disrupted cell organelles of an indistinct structure (Fig. 6b).

Wall appositions ocurred frequently in the cortical parenchyma cells invaded by the incompatible isolate CBS 178.26 (Fig. 7a, b). The fungal cells enclosed by the wall appositions were completely disorganized. The fungal plasma membranes fully separated from the fungal wall were broken and fragmented (Fig. 7a). Numerous vesicles were present in the damaged cytoplasm of fungal cells (Fig. 7b). In

the cortical cells, the heavily disorganized haustorium did not grow through the host wall apposition and cytoplasmic aggregation.

DISCUSSION

The compatible (S 197) and incompatible (CBS 178.26) isolates of *P. capsici* successfully colonized the epidermal layer, cortex, and xylem vessels of tomato stems. In the stem tissues, most of the fungal hyphae grew intercellularly, but intracellular hyphae were less frequent. The finding that the invading hyphae preferred to grow intercellularly might be due partly to physical weakness of the middle lamella, as previously observed by Hinch *et al.* (10) in maize roots inoculated with *P. cinnamomi*. Host cells damaged due to fungal invasions were intensely stained with toluidine blue, as in pepper tissues inoculated with *P. capsici* (21). Early cytological differences between the compatible and incompatible interactions were found at the light microscopic level. The incompatible response to *P. capsici* of tomato cells was characterized by reduction in the extent of fungal colonization. This might be due to the limited nutrients available for the fungal cells or the formation of chemical barriers to the fungal growth. In comparison with the compatible isolate S 197, colonization of the incompatible isolate CBS 178.26 was distinctly reduced in the cortex layer and vascular steles of tomato stems.

Our observations at the electron microscopic level clearly demonstrated that ultrastructural changes occurred in the stem tissues of tomato plants inoculated with either the compatible or the incompatible isolate. Degraded host cell wall, fragmented host plasma membrane, distorted chloroplast, and remnants of host cytoplasm were early ultrastructural features of the damaged host cell organelles observed commonly in the compatible and incompatible interactions, although the damage of host cell organelles were less severe in the incompatible interaction. Damages of host cell organelles, such as host cell wall, and host plasma membrane also have similarly been demonstrated in various plant tissues infected with each of several *Phytophthora* spp. (6, 11~14, 18, 19, 27).

In the incompatible interaction, a number of vesicles, probably derived from dictyosome or endoplasmic reticulum (7), were distributed in the spaces between fungal cell walls and plasma membrane. This suggests the possible involvement of vesicle formation in the incompatible response of fungal cells to the host cells. In our previous studies (13, 15),it was demonstrated that the systemic fungicide, metalaxyl, altered the morphology and location of vesicles in fungal cells in the pepper-*P. capsici* combination.

The host cell walls of stem tissue were degraded and became thin at the point in contact with the fungal cells, suggesting that some enzymatic process may be participated in penetrating host cell wall. The possibility of enzymatic degradation of host cell walls are well supported by the finding of Yoshikawa *et al.* (29) that P. capsici produced plant cell wall-macerating enzymes. The degradation of host cell walls by *P. capsici* was more pronounced in the compatible than the incompatible interactions. Numerous electron-dense particles were scattered on or around the host cell wall and the fungal cell was somewhat degraded (Fig. 3b). These particles were considered to be products of enzyme-substrate reaction. Plant hydrolases such as 1, 3, -β- glucanases and chitinases were identified from tomato tissues inoculated with *Fusarium oxysporum* or *Cladosporium fulvum* (5, 20). Benhamou *et al.* (4) also reported that plant hydrolases accumulated predominantly in xylem cells of tomato plants.

Along with confinement of fungus to specific plant tissue, rapid host cell necrosis is a commom feature of the incompatible interaction. However, we could not find the clear evidence of host cell necrosis in the incompatible interaction. Host cells not directly contacted with the fungal cell were often unaffected (Fig. 5a). Although host cells neighboring the pathogen were degenerated in the incompatible interaction. Their degeneration was not considered to be the host cell necrosis, but rather due to diffusion of fungal metabolites harmful to the plant cell. Contrary to our results, rapid necrosis of host cells neighboring the pathogen was observed in other incompatible interactions of *P. megasperma*-soybean and *P. infestans*-potato systems (22, 23, 28). In our previous study, no hypersensitive, necrotic reaction was observed in tomato stems inoculated with the incompatible isolate CBS 178.26 at the macroscopic level (17). In some sections of incompatible interactions, the epidermal layer of stem tissue was rapidly collapsed, but the inner part of the stem was rarely

invaded by the fungal cell.

The incompatible interactions of tomato cells with *P. capsici* were characterized by formation of wall apposition in the cortical parenchyma cells (Fig. 6 and 7). The fungal cells encased by the wall apposition had abnormal cytoplasm and separated plasma membranes. The haustorium which formed from the fungal hyphae in the cortical parenchyma cells did not further penetrate through the host wall apposition and cytoplasmic aggregation in the incompatible reactions. However, the haustorium of the compatible isolate S 197 was not encased by wall apposition. Host cell wall apposition has been known to be a nonspecific reaction to the fungal invasion. It also occurs in the compatible interactions or fungicide-treated plants (13, 14). Wall appositions also have been observed in some host-Phytophthora combinations, more frequently in the incompatible than the compatible interactions (3, 8, 28). The functions and compositions of the host wall appositions remain still unknown (1, 2). Our results suggest that they may function as barriers against host cell damage by the fungus, i.e., possessing low permeability to small nutrients, or restricting the progress of the fungus by other factors such as phytoalexins (16).

요 약

토마토와 *P. capsici*의 친화적, 불친화적 상호작용의 조직학적 차이를 비교하기 위해 *P. capsici*를 접종한 토마토(품종 : 광양)의 줄기조직을 광학현미경과 전자현미경을 사용하여 조사하였다. 접종 24시간 후, 친화적 균주인 S 197은 줄기의 표피, 피층 및 목부에 심한 피해를 초래하였으며, 불친화적 균주인 CBS 178.26은 소수만이 줄기조직에 정착하였다. 세포막의 절단, 엽록체의 변형, 세포벽의 분해 및 기주 세포질의 소실 등과 같은 기주세포의 미세구조적 변화는 친화적 상호작용과 불친화적 상호작용에서 공통적으로 관찰되었지만, 불친화적 상호작용에서는 기주세포의 손상이 적었다. 불친화적 상호작용에서 소수의 소포가 병원균 세포벽과 세포막 사이의 공간에서 관찰되었다. *P. capsici*에 의한 기주세포벽의 분해는 불친화적 상호작용에서 보다 친화적 상호작용에서 더 뚜렷하였다. 토마토 세포와 *P. capsici*간의 불친화적 상호작용의 특징은 피층유세포에 wall apposition이 형성되는 것으로, 이는 기주세포벽에 형성되는 물질이 병원균의 침입에 대해 방어역할을 하고 있음을 뜻한다. Wall apposition으로 둘러싸인 병원균 세포의 세포질은 비정상적이었으며 세포막은 세포벽으로부터 분리되었다. 불친화적 상호작용의 경우, 병원균에서 형성된 흡기는 wall apposition과 세포질응집물 (cytoplasmic aggregation) 을 뚫고 생장하지 못하였다. 이와 반대로, 친화적 균주인 CBS 178.26의 흡기는 wall apposition에 둘러싸이지 않았다.

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