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## Compared Anatomy of Young Leaves of *Prunus persica* (L.) Batsch with Different Degrees of Susceptibility to *Taphrina deformans* (Berk.) Tul.

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### Abstract

Anatomical observations of leaves infected by *Taphrina deformans* were studied in tolerant peach trees (TPT) and in very susceptible (VSPT) ones. Leaves from the first sampling (2nd April) showed hyphae penetrating through the stomata or into the cuticle of the host tissue; anatomical structures of leaf sections were similar for both TPT and VSPT. The ultrastructure of the leaves of TPT showed seemingly normal mesophyll cells. In contrast, mesophyll cells of the VSPT showed important signs of degradation. Cells were organelle-free and the middle lamella was expanded and invaded by hyphae of *T. deformans*. In some samples, the leaves of TPT showed deformed epidermal cells, loss of some spongy cells and increase of the intercellular spaces and division of the palisade cells. The pathogen proliferation in the leaves of the VSPT was considerably superior. In this case, stimulation of cell division occurred in the abaxial epidermis. Cells showed periclinal and oblique divisions, with an increased number of plasmodesmata; palisade or spongy cells were not differentiable. Leaves from TPT collected on 26th April showed hyphae with a non-cylindrical section and with a squashed aspect. The hyphae were very evident in the intercellular spaces, showing abundant endoplasmic reticulum of rough type (RER) in the cytoplasm. On the other hand, epidermis of the leaves of the VSPT had numerous hyphae under the cuticle, which were growing in a thick pectin matrix. Leaves from TPT and VSPT collected on 6th May showed relevant differences. The leaves of TPT had a palisade mesophyll with fewer cells but with active chloroplasts. In contrast, the leaves from VSPT showed empty mesophyll cells, the cytoplasm was collapsed and the adaxial epidermis was covered with the fungus fructification. The observed anatomical and ultrastructural differences of leaves from TPT and VSPT confirm a different behaviour in plant-host reaction at early stages of infection.

### Introduction

*Taphrina deformans* (Berk.) Tul. is a very harmful fungus that causes leaf curl on peach trees [*Prunus persica* (L.) Batsch] in most of the cultivation areas of this fruit species (Agrios 1988). It is responsible for early defoliation and crop loss on nearly all peach cultivars, with very important economic injuries.

Many authors (Ritchie and Werner 1981; Simeone and Corazza 1987; Corazza 1988; King et al. 1988; Scorza 1992; Roselli et al. 1997; Ivascu and Balan 1998; Trandafirescu et al. 2007) have studied the susceptibility of peaches and nectarines to leaf curl, reporting a wide range of variability among cultivars. Nectarine seems to be less susceptible than peach according to Ackerman (1953) or more susceptible according to others (Ritchie and Werner 1981; Padula 2010). Only a few cultivars are considered tolerant (Pscheidt 1995). Among 108 commercially available cultivars in Italy monitored for four years, no tolerant genotypes were identified by Simeone (1987); on the other hand, a three-year evaluation of 66 U.S. peach and nectarine introductions and six commercial cultivars showed 12 plants to be highly resistant, some of them showing no visible symptoms of infection (Scorza 1992). A study on the susceptibility of peach and nectarine cultivars to leaf curl was carried out in 2007–2009 (Padula 2010), analysing 241 different genotypes including international cultivars, Italian local varieties and new selections recently obtained by breeding; some of them resulted resistant, others showed different degrees of susceptibility.

The curled leaf caused by *T. deformans* (Berk.) Tul. was anatomically observed by Matuyama and Misawa (1961) and Syrop (1975a,b), and some of the ultrastructural alterations and cytochemical modifications observed during cell wall degradation were studied by Marte and Gargiulo (1972), Syrop and Beckett (1976) and Bassi et al. (1984). In addition, physiological effects promoted by the fungus, like the negative CO<sub>2</sub> fixation in curled leaf (Raggi 1995) and the promotion of cell

elongation and division (Sommer 1961), were investigated to elucidate the fungus–host cell interaction.

The aim of the present work was to identify and study anatomical differences between the infected leaves of *Prunus persica* (L.) Batsch on both types of reaction to the infection of *T. deformans* (Berk) Tul., that is to say using tolerant peach trees (TPT), represented by tolerant advanced selections obtained by breeding (Bellini et al. 1996, 2002), and very susceptible peach tree (VSPT), represented by commercial cultivars.

### Materials and methods

The leaves were collected from adult trees grown in the experimental field of the Montepaldi University Farm in San Casciano Val di Pesa (Florence Province - Italy) in the years 2008–2009, in the frame of a study on the susceptibility of peach to leaf curl (Padula 2010). The orchard is located at 43°40'N, 11°09'E and 230 m above sea level.

Once the 241 genotypes of the collection were scored using the 0–5 rating scale developed by Simeone and Conte (1994), two tolerant accessions (with no visible signs of leaf curl; score 0) and two very susceptible cultivars with more than 50% leaves with symptoms (score 5) were chosen for the study as TPT and VSPT, respectively. In further detail, the plant material consisted of young leaves of white-flesh peach advanced selections 'DOFI-84.364.004' and 'DOFI-84.364.017', tolerant to *T. deformans*, and of 'Baby Gold 6' and 'Mary Star', two very susceptible cultivars. Leaves were collected on three different dates during spring 2008: on 2nd April, when leaves started to open and no leaf curl symptoms were observed; on 26th April, when curled leaves were clearly visible; and on 6th May, when symptoms exhibited maximum intensity.

Twenty leaves were collected for each of the trees and date. Samples were observed using different techniques of optical and electron microscopy.

### Optical microscopy analysis

Leaf material fixed in FAA was embedded in paraffin wax. Five blades of each sample were cut in 7–10  $\mu$ m sections and stained with safranin and fast-green (D'Ambrogio de Argüeso 1986) to study the mesophyll and epidermis cell alteration.

### Environmental scanning electron microscopy (ESEM)

Leaves collected on 2nd April and fixed in FAA (D'Ambrogio de Argüeso 1986) were explored externally. The epidermis blade surfaces were observed through a Philips Electron Scan 2010. Five samples of each tree type/date of collection were employed.

### Transmission electron microscopy (TEM)

Leaves were fixed in a 3% (v/v) glutaraldehyde solution in phosphate buffer (pH 7.4) for 24 h and then postfixed in OsO<sub>4</sub> at 2°C in the same buffer for 3 h. After this procedure, they were dehydrated in an ethanol series and embedded in Spurr's resin. Thin sections (75–90 nm

thick) were stained with uranyl acetate and lead citrate. Five samples of each tree type/date of collection were observed with a Jeol-Jem 1200 EXII TEM.

### Results

Leaves collected on the first sampling date (2nd April) showed little anatomical differences in comparison to the uninfected leaves (Fig. 1). As a matter of fact, *T. deformans* mycelium was present on the lower surface of the sprouted young leaf blades of all the studied trees (Fig. 2a). No asci were visible on the lower surface. Hyphae penetrated through the stomata (Fig. 2b) or into the cuticle of the host tissue. Figure 2c shows a particular case, with the penetration on the principal vein zone. The anatomical structure of the leaf sections was similar for both the TPT and VSPT. The adaxial surface showed the epidermis constituted by one layer of isodiametric cells with the cuticle. Mesophyll was dorsiventral. Palisade parenchyma and spongy parenchyma were well distinguished (Fig. 2d). The palisade parenchyma was formed by two to three layers of elongated cells in cross-section, shorter towards the central mesophyll. Spongy cells were equal in diameter and the areas of contact between the cells were flatter and wider; as a consequence, the volume of intercellular spaces in this tissue was superior to that in the palisade tissue. Veins, composed by phloem and xylem, were present in the mesophyll and were surrounded by a layer of tightly packed parenchyma cells. Infection was observed in all the described tissues. The pathogen extended the mycelium following a tangential direction in the intercellular space and the cells surrounding the area of pathogen invasion were gradually influenced.

The ultrastructure of TPT leaves showed seemingly normal mesophyll cells. The cytoplasm of these cells contained chloroplasts with large starch grains and well-developed grana, numerous ribosomes, mitochondria and vacuoles (Fig. 3a, b). The middle lamella showed a slight thickening (Fig. 3a, b).

In contrast, mesophyll cells of VSPT showed important signs of degradation. Cells were free of organelle and the middle lamella was expanded; the hyphae of *T. deformans* grew through it (Fig. 3c,d).

More developed leaves of tolerant trees showed deformed epidermal cells, loss of some spongy cells, an increase in the intercellular spaces and division of the palisade cells (Fig. 4a, b). Proliferation of the pathogen in susceptible trees was considerably greater. Stimulation of cell division was observed in the abaxial epidermis. Cells were divided in periclinal and oblique sense and the number of plasmodesmata increased (Fig. 4d). These cells showed an anatomical structure typical of a condition of intense metabolic activity (Fig. 4d). Mesophyll cells were dehydrated and cytoplasmic contents were lacking (Fig. 4c). It was not possible to differentiate palisade or spongy cells and the intercellular spaces were reduced. Cells were enlarged noticeably and many of them were very large. In addition, there was an important proliferation of

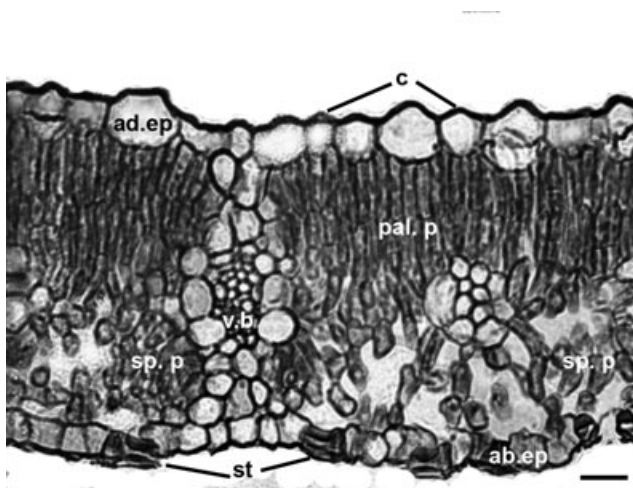


Fig. 1 Micrograph of portion of a cross-section of the uninfected leaf blade of *Prunus persica* (L.); **ad.ep**, adaxial epidermis; **ab.ep**, abaxial epidermis; **c**, cuticle; **pal.p**, palisade parenchyma; **sp.p**, spongy parenchyma; **v.b**, vein bundle. Bar: 50  $\mu$ m

the mycelium under the cuticle of the adaxial epidermis (Fig. 4c, d).

Leaves collected on the second sampling date (26th April) from TPT showed abnormal hyphae, with a non-cylindrical shape in section and with a squashed aspect (Fig. 5a, b). The hyphae were very evident in the intercellular spaces and they showed abundant endoplasmic reticulum of rough type (RER) in the cytoplasm (Fig. 5c). At this stage of infection development, some mesophyll cells of the leaves of the TPT had active cytoplasm and walls with ingrowths (Fig. 5b). Plasmodesmata increased between mesophyll

cells (Fig. 5d). On the other hand, epidermis of the very susceptible trees had numerous hyphae under the cuticle, which were growing in a thick pectin matrix (Fig. 5e, f). The hyphae cytoplasm contained mitochondria with well-developed cristae, abundant free ribosomes and lipidic globules (Fig. 5f).

Finally, leaves collected on the last sampling date (6th May) from tolerant and susceptible trees showed important differences. The former showed palisade mesophyll with a lower number of cells but with active chloroplasts (Fig. 5a). In contrast, 'Baby Gold 6' and 'Mary Star', the very susceptible specimens, showed empty mesophyll cells; furthermore, the cytoplasm was collapsed and the adaxial epidermis was completely covered with the fungus fructification (Fig. 6b).

Asci development was observed only on the very susceptible trees. Each ascus contained more than eight ascospores (Fig. 6c). Remnants of cytoplasm between the ascospores were also observed (Fig. 6c). Ascospores contained numerous lipidic globules and high electron-dense corpuscles. Cell walls showed some invaginations into the cytoplasm (Fig. 6d).

## Discussion

*Taphrina deformans* (Berk.) Tul. is the causal agent of leaf curl and the disease is evident in spring when new leaves are growing. Fungus incites neoplastic growth of leaves of *P. persica* (L.) Batsch. This effect is accomplished by cell division and cell enlargement which in turn promote a tumour-like structure. Infection occurs when the mycelia developed on the lower leaf surface penetrate into the tissues of the young leaves. In agreement with Bassi et al. (1984), no asci

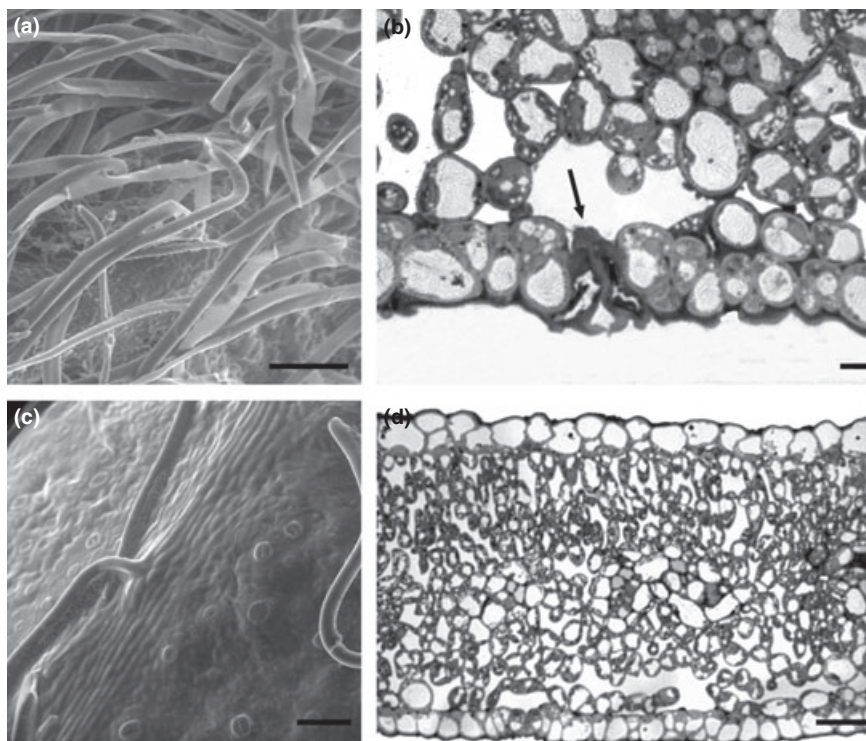


Fig. 2 Leaves of *Prunus persica* (L.) Batsch. at the start of infection by *Taphrina deformans* (Berk.) Tul. (a, c) Hyphae of *T. deformans* on abaxial surface observed by ESEM. (b, d) light microscopy micrographs; (b) Detail of infected abaxial epidermis with a hypha going through stomata (arrow); (d) Transversal section of the leaf at the beginning of the infection. Bars: (a, b, d) 50  $\mu$ m; (c), 100  $\mu$ m



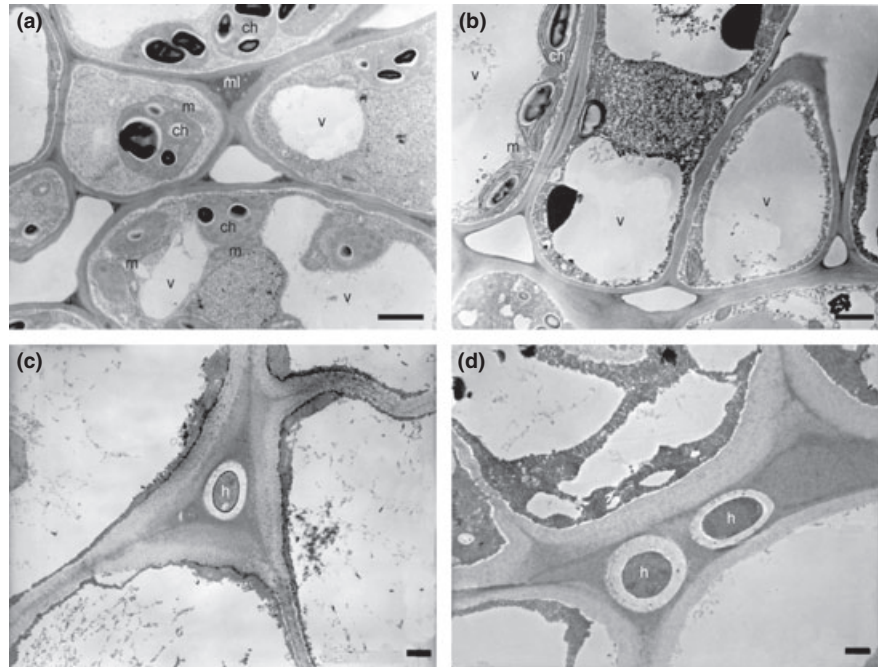


Fig. 3 Transmission electron microscopy micrographs of infected leaves of *Prunus persica* (L.) Batsch. by *Taphrina deformans* (Berk.) Tul. (a, b) Detail of palisade parenchyma cells of tolerant trees, chloroplasts (ch), mitochondria (m), middle lamella (ml), vacuole (v). (c, d) Hyphae (h) of *T. deformans* growing through middle lamella of mesophyll cells of susceptible trees. Bars: (a, b) 2  $\mu$ m, (c, d) 500 nm

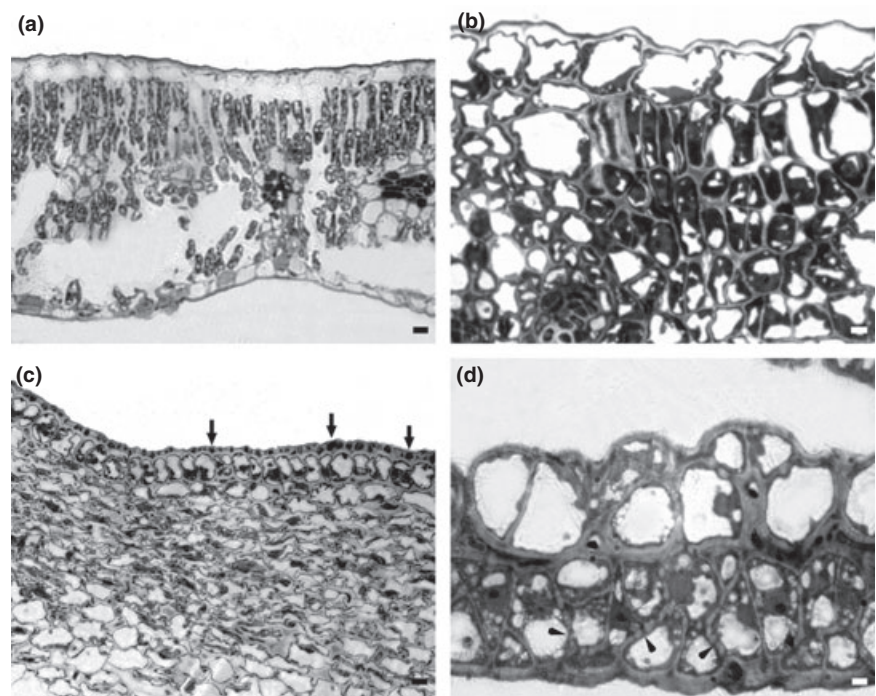


Fig. 4 Light microscopy micrographs of transverse section of *Prunus persica* (L.) Batsch. leaf on advanced infection by *Taphrina deformans*. (a, b) Leaf of tolerant trees; (c, d) Leaf of susceptible trees. (c) Hyphae under the cuticle of the adaxial epidermis (arrows); (d) Abaxial epidermal cells with plasmodesmata (arrow head). Bars: (a, c) 10  $\mu$ m; (b, d) 50  $\mu$ m

were observed on this surface of the leaves from tolerant trees in the present study.

*Taphrina deformans* (Berk.) Tul. has an intercellular habit and it can grow below both the cuticle and epidermis, mainly in the upper surface of leaf blade and deeply in the mesophyll. Infection of the leaf tissue is possible because of the alteration of the interface fungus-leaf cell wall. Bassi et al. (1984) noted a dissolution of the middle lamella, loosening of the cell wall structure and an alteration of the plasma membrane in infected leaves. This effect of the fungus infection was

well manifested when the leaf ultrastructure of very susceptible trees was studied. On tolerant trees, this process occurs more slowly and apparently a resistance response is activated, producing a deformation of the cell aspect.

The host reaction shows that the presence of the fungus causes immediate cell division followed by cell enlargement and cell differentiation, according to the observations of Matuyama and Misawa (1961) and Sommer (1961). The former authors showed that stainability is greater when the tissue is damaged.

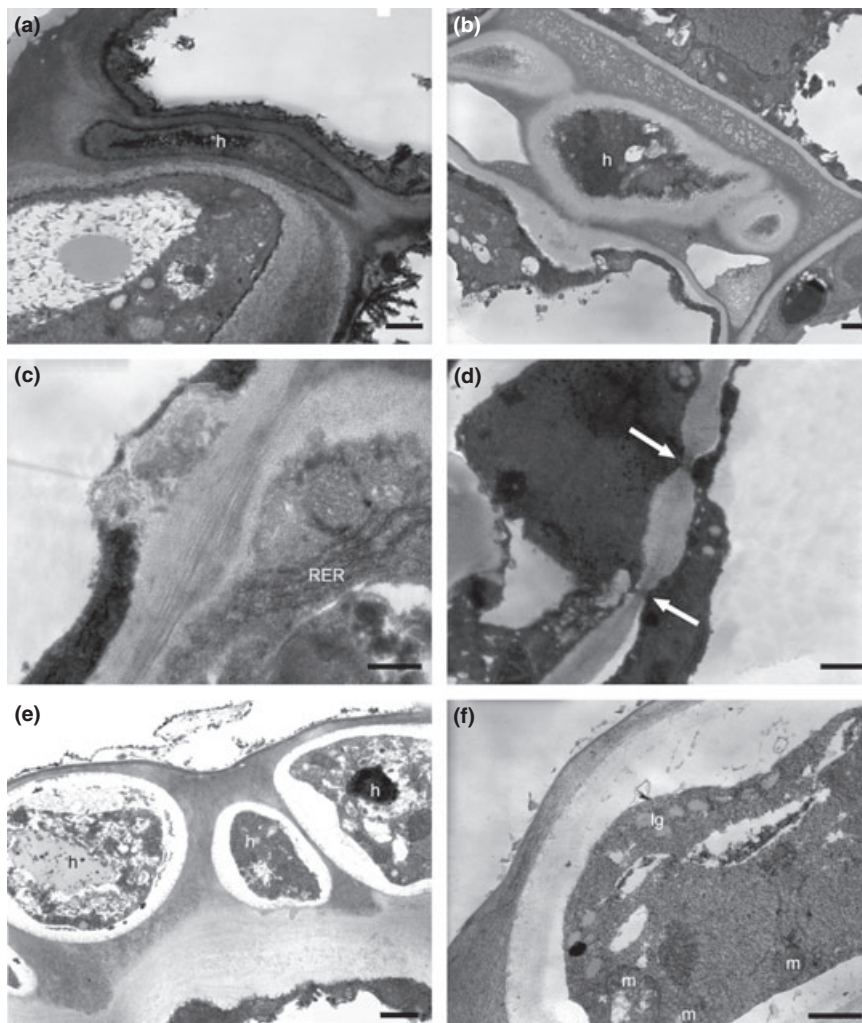


Fig. 5 Transmission electron microscopy micrographs of infected leaf of *Prunus persica* (L.) Batsch. by *Taphrina deformans* (Berk.) Tul. (a-d) Leaf of tolerant trees. (a, b) Detail of hyphae (*h*) growing in the intercellular matrix. (c) Detail of hypha cytoplasm, endoplasmic reticulum of rough type (*RER*). (d) Detail of plasmodesmata (*arrows*). (e, f) Leaf of susceptible trees. (e) Details of hyphae (*h*) growing under the cuticle. (f) Cytoplasm of hypha with mitochondria (*m*), lipidic globules (*lg*) and free ribosomes. Bars: (a-f) 500 nm

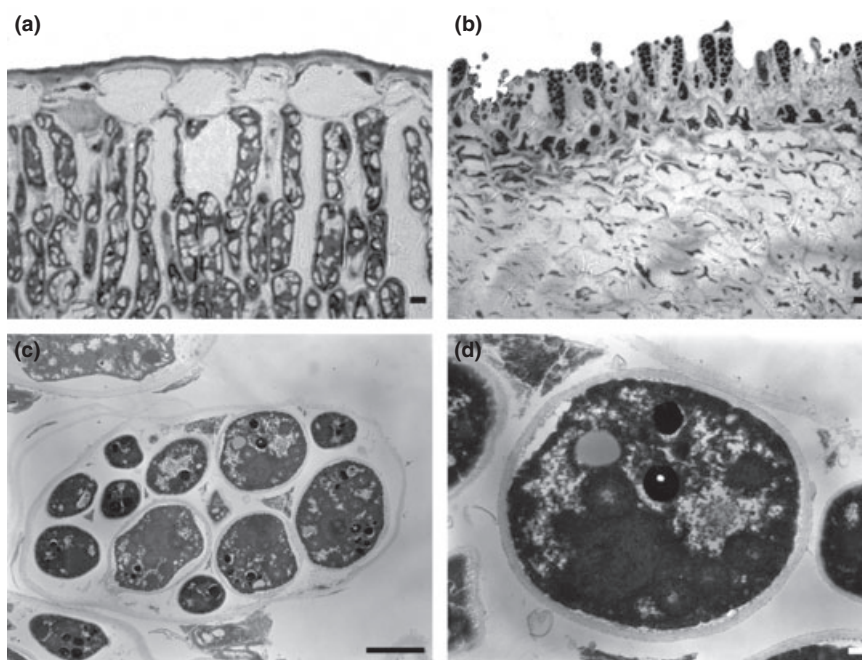


Fig. 6 Light microscopy micrographs of leaves with advanced infection. (a) Adaxial epidermis and mesophyll of tolerant trees, 100 $\times$ ; (b-d) Leaf of susceptible trees. (b) General aspect of asci on adaxial epidermis; (c) Detail of an ascus. (d) Detail of an ascospore. Bars: (a) 50  $\mu$ m; (b) 10  $\mu$ m; (c) 5  $\mu$ m; (d) 500 nm



Comparison of healthy and infected tissue showed that leaves infected with *T. deformans* (Berk.) Tul. have an increased cytokinin activity, with a high indole 3-acetic acid (IAA) and tryptophane content (Sziràki et al. 1975). In addition, ethanolic extract of *T. deformans* (Berk.) Tul. contains a cytokinin-like substance that promotes cell division in the presence of exogenous IAA (Sommer 1961). Cytokinin and auxin may play a role in producing abnormal growth of the host plant (Sommer 1961).

*Taphrina deformans* (Berk.) Tul. secretes polygalacturonide-degrading enzymes and probably cellulose-degrading enzymes as well (Bassi et al. 1984). This secretion of lytic enzymes allows penetration of fungus along the intercellular spaces and, furthermore, the digestion of wall polysaccharides offers nutrients to the parasite.

Asci were observed only on the leaves of VSPT, four weeks after the initial infection, as observed by Rossi et al. (2007). As a matter of fact, the leaves of very susceptible trees suffer a strong inhibition of CO<sub>2</sub> fixation and consequently a reduction of photosynthetic activity (Raggi 1995). In parallel, the hyphae on these types of leaves show evident mitochondria, which indicate an intense respiration activity. On the contrary, abundant RER was observed on the hyphae growing in leaves of TPT. In this regard, it is known that *T. deformans* may continue the parasitic cycle when nutritious substrate is available (Rossi and Languasco 2007; Rossi et al. 2007). In fact, this would be the case of hyphae observed on leaves of TPT, where photosynthesis is not lessened by infection. According to the results obtained and in agreement with the investigations of other authors, the resistance demonstrated by TPT (the advanced peach selections) is presumably due to a different behaviour of the host cells. It is possible that the leaf cells of 'DOFI-84.364.004' and 'DOFI-84.364.017' react against enzymatic secretions of *T. deformans* (Berk.) Tul., thus reducing the damage caused by the fungus in the cell wall. Hence, the fungus would find some impediment to formation of hyphal strand aggregates in the subcuticular regions and it would be unable to satisfy its reproductive cycle. It is worth recalling that recent studies have demonstrated that the leaves of TPT display a twofold greater concentration of phenolic compounds with respect to that observed in leaves of the susceptible trees (Padula 2010). A similar trend was observed in epidermis and mesocarp of peaches produced by trees resistant to *Monilia fructigena* (Gradziel et al. 1998). This finding is in line with the reports on the role of polyphenols in defence of plants against fungal and bacterial diseases observed in several species (Mayer 2006; Lattanzio et al. 2006). For instance, Treutter and Feucht (1990) reported higher levels of polyphenols in apple leaf tissues in cultivars resistant to *Venturia inaequalis* (bacterial scab) compared with the susceptible ones; the polyphenolic pattern of leaves has been correlated with scab resistance and different levels and types of preformed polyphenols have been

observed in leaves in relation to apple plant resistance (Picinelli et al. 1995). Hence, the different resistance levels found in peach resistance to leaf curl and the hypersensitive necrotic reaction observed in the resistant trees (Bellini et al. 1996, 2002) may be explained by both preformed and induced antifungal phenolics (Lattanzio et al. 2006).

In conclusion, the anatomical and ultrastructural differences observed in leaves of tolerant and very susceptible peach trees confirm a divergent host-parasite reaction from early stages of infection up to the reproductive phase of *T. deformans* (Berk.) Tul.

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