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Biological Cycle of Helminthosporium solani: An Overview using Microscopy

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Silver scurf of potato (*Solanum tuberosum* L.) is a surface-blemishing disease spoiling the appearance of potato tubers [1]. The disease is caused by the fungus *Helminthosporium solani* Durieu & Mont. [2]. Silver scurf-infected tubers are characterized by metallic discoloration of the periderm in irregular patterns [3]. Long recognized as a disease of minor importance, silver scurf, which is present in most potato-growing areas [2], has become during the 1990s a leading cause of rejection of commercial potatoes [4,5]. Development of *H. solani* resistance to thiabendazole, which formerly provided control as post-harvest treatment [5,6,7,8], is one of the reasons explaining the dramatic increase in economic importance of silver scurf. The present study was carried out to further elucidate infection processes using transmission and scanning electron microscopy.

The first steps of *H. solani* penetration were visible 6 hours after inoculation of potato tubers with a suspension of the fungus. Scanning and transmission electron microscopy showed that the fungus penetrates potato tubers mainly via hyphae (Fig. 2). Germ tubes, which appeared at different levels of the conidia (Fig. 1), were also able to penetrate the periderm. In this study, no appressoria were observed. Transmission electron microscopy showed the presence of an extracellular sheath around hyphae on the periderm surface (Fig. 3). Dissolution of the host cell wall material was observed during the penetration by hyphae (Fig. 2). Host cell wall and membrane were totally destroyed by the penetration of the fungus. The presence of the fungus inside the host cell was observed 9 hours after potato inoculation. After this period, the fungus was present principally in the periderm but was also observed in some cells of the cortex. Invasion of host cells appeared mainly during the 2 days following the penetration leading to host cell death (Fig. 4). Conidiophores were observed on periderm surface 2 days after inoculation of potato tuber (Figs. 5-7).

This cytological study which allowed to describe key steps of potato tuber infection by *H. solani* is a part of an ongoing study on relationships between the pathogen and newly selected antagonist microorganisms for the biocontrol of potato silver scurf [9].

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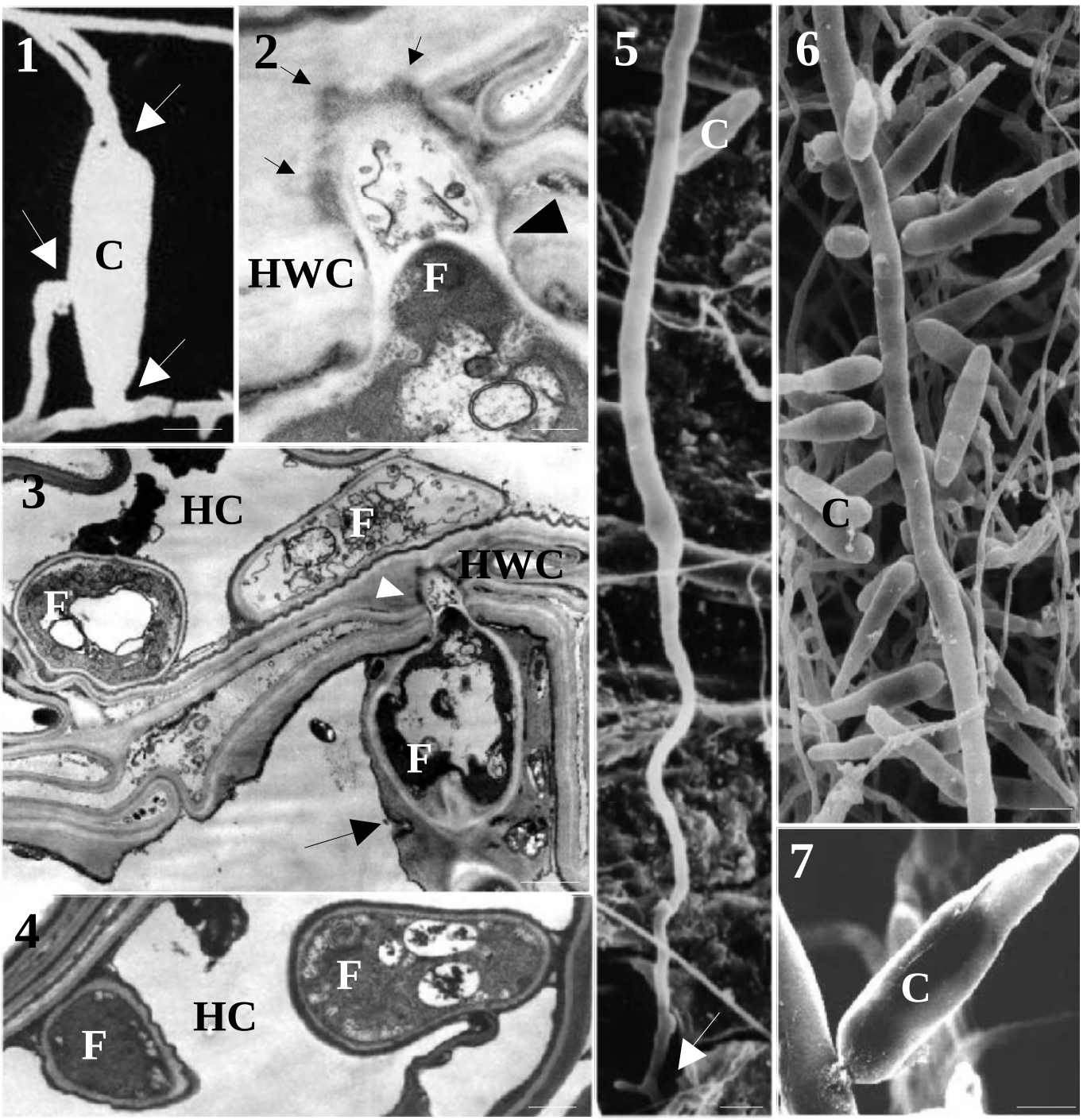

FIG. 1. Germination of conidia (C) at different level of conidial cells (arrow). Bar ≡ 5µm
FIG. 2. Penetration of hyphae. The host cell wall is broken (arrowhead) by the fungus (F). Note deposit (arrows) at the point of contact between fungus and the host cell wall. Bar ≡ 200nm
FIG. 3. Hyphae inside and outside host cell. Note the presence of a sheath (arrow) around the fungus outside the host cell. Bar ≡ 1µm
FIG. 4. Fungus cells inside the host cell in the cotex zone of the potato tuber. Note that the host cell is totally destroyed. Bar ≡ 500nm
FIG. 5. Development of a conidiophore. Note the point of emergence (arrow) of the conidiophore from periderm cell of the potato tuber. Bar ≡ 10µm
FIGS. 6-7. Conidiophore and conidia (C). Fig. 6: Bar ≡ 10µm, fig 7: Bar ≡ 5µm

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