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Ultrastructure and molecular cytology of interaction between wheat and *Puccinia striiformis* Westend

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Wheat stripe rust, caused by *Puccinia striiformis* Westend f. sp. *tritici*, occurs worldwide and is considered a major disease in temperate regions, particularly in China. To reveal the resistance mechanism of wheat to stripe rust, we recently examined the compatible and incompatible combinations between wheat and *P. striiformis* by means of electron microscopic and immuno-gold labeling techniques. The infection process of *P. striiformis* is similar to that of other cereals rust. After penetrating the stoma of wheat leaves, it forms substomatal vesicle, infection hypha, haustorial mother cell and haustorium within the host tissue, respectively. The multinucleat condition, *e.g.* more than two nuclei, is usually found in the intercellular hypha cells, haustorial mother cells and haustoria. In the infected wheat leaves of the susceptible cultivar a higher hyphae number was usually detected as compared to the corresponding tissues in the resistant cultivar, indicating that the fungal development was restricted in wheat leaves of the resistant cultivar. The structural defense reactions such as formation of cell wall apposition, collar or papillae, and encasement of haustorium were essentially more pronounced in the infected wheat leaves of the resistant cultivar than in the susceptible one. Sometimes, in the wheat leaves of the incompatible combination the typical papillae of large size, detected in the host cell subjacent to the penetration site of the haustorial mother cell stopped the pathogen's further development. Immunogold studies demonstrated the presence of callose in the collars or papillae, cell wall appositions and encasements formed in *P. striiformis*-infected wheat leaves. Immunogold localization of lignin revealed a markedly higher labelling density in host cell walls of the infected wheat leaves of the resistant cultivar than in the cell walls of the infected wheat leaves of the susceptible wheat cultivar. These findings indicated that lignin accumulation in the infected wheat leaves may play an important role in resistance to the spreading of the pathogen in the host tissues.

Two antisera raised against acidic chitinase and acidic ß-1, 3-glucanase were used to investigate the subcellular localization of the two enzymes in the compatible and incompatible interactions between

wheat and *P. striiformis*. The studies demonstrated that the labelling patterns for both enzymes were very similar in the uninoculated healthy and infected wheat leaves. The enzymes were localized mainly in the host cell walls, while no labeling was observed in cytoplasm and organelles of the host cells. However, the accumulation of two enzymes in the infected wheat leaves differed markedly between resistant and susceptible wheat cultivars. The labelling densities for the two enzymes in the infected leaves of the susceptible cultivar increased slightly as compared to the uninoculated healthy leaves, whereas significantly higher labelling densities of chitinase and ß-1, 3-glucanase were found in the infected leaves of the resistant cultivar compared to the uninoculated healthy leaves. Furthermore, the labelling of chitinase and ß-1, 3-glucanase also occurred over the extrahaustorial matrix and the fungal cell walls in the infected wheat leaves. The extrahaustorial matrix and the hyphal cell walls in the infected leaves of the resistant cultivar usually showed a higher density of the labelling than those in the susceptible cultivar. These finding indicated that chitinase and ß-1, 3-glucanase accumulation have potential role in the defense reactions in the incompatible interaction between wheat and *P. striiformins*.