PATHOHISTOLOGY OF SUNFLOWER STEMS ATTACKED BY DIAPORTHE HELIANTHI

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AIMS OF THE STUDY

The disease of sunflower plants caused by the holomorph Diaporthe/Phomopsis helianthi Munt.-Cvet. et al. was first noticed in Yugoslavia in July 1980 (Mihaljčević et al., 1980), when cankers on the stems had already dramatically damaged the plants. The stem lesions were then considered to be the first disease symptoms. Petrov et al.(1981) soon refuted this assertion and indicated that the cankers were the expression of an advanced step of pathogenesis: the fungus entered through the leaves, progressed towards the petioles along the foliar veins, and finally gained entrance to the stems. The leaf-petiole-stem infection pathway has now been recognized by several French investigators (Bertrand & Tourvieille, 1987); some authors, however, still consider that the fungus enters directly from outside into the plant stems, and that this is the common ingress route of D.helianthi.

The aims of the present study were:

1. To follow the invasion of the host tissues by the parasite and the pathological alterations which the fungus causes in the host stem tissues;
2. To determine when and where in vivo the fungus forms its asexual fructifications (the Phomopsis state);
3. To determine when and where differentiation of the fungus sexual reproductive structures begins in the host tissues (the Diaporthe state);
4. To follow the development of fungal perithecia in the host tissues under field conditions.

Since the foliar blades constitute a particular context, histological studies on this subject will be the matter of a separate publication.

RESULTS

Through nearly 7000 histological slides of sunflower plant parts attacked by the holomorph Diaporthe/Phomopsis helianthi, which have been examined since 1980, the leaf-petiole-stem infection pathway has been corroborated for Yugoslav conditions.

After foliar infection the hyphae progress through the vascular system and then spread to invade other tissues. Hyphal masses form in the host cortex which represent pycnidial primordia. When fully differentiated the pycnidia expand towards the host periphery and rupture the epidermis. The stem cankers with the asexual fructifications of the fungus represent an advanced stage of pathogenesis.

The fungus nuclear phase change occurs in the host pericycle, beneath the endodermis. Ascogonia can be observed beginning from the autumn; their development into protoperithecia and perithecia takes place slowly and unevenly during the subsequent months. Perithecial maturation in spring, when ascospores are abundantly released, must coincide with the onset of the host vegetation to satisfy the nutritional needs of the anamorphic, parasitic phase of the fungus.

Exceptions to this general scheme have been observed, with ascospore maturation occurring during the winter months.

LITERATURE

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