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Reduced Lysine Uptake by Bean Rust Haustoria in a Resistant Reaction

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Host-cell necrosis, which commonly occurs when pathogens infect incompatible host varieties, has suggested a causal relationship between abnormal, rapid cell death and resistance (see [1]). It is not known, however, whether the 'hypersensitive' [2] cell death inhibits fungal growth by formation of substances toxic to the pathogen and by depriving the biotrophic pathogen of its food base, the living cell; or whether the hypersensitive reaction is only a consequence of an unsuccessful interaction between the host and pathogen and thus, is not primarily responsible for resistance. Our results are consistent with the latter possibility.

The nutrient uptake (^{3}H-lysine) by *Uromyces phaseoli*, bean rust, was studied by autoradiographic electron microscopy in compatible and incompatible cultivars of bean, *Phaseolus vulgaris*. The cultivar 'Favorit' is compatible, i.e., about 18 h after inoculation with the uredospores, the infection hypha forms the first haustorium in the leaf mesophyll. Within 3 to 5 h later, the haustorium is fully developed and running hyphae differentiate haustoria every 8 h. Eight days later, uredia appear. The bean cultivar 'Golden Gate Wax' is incompatible, i.e., the formation of the first haustorium also begins 18 h after inoculation, but 3–5 h later, disintegration of the plant cell content begins and extends to most infected cells. Haustoria also deteriorate and fungal growth is retarded. Only very small uredia form two weeks later. To study the function of the haustoria, the leaf was fed with lysine by dipping the petiole in 1 ml water with 330-μCi ^{3}H-lysine. One day later, unlabeled uredospores were allowed to infect the labeled leaf under high-humidity conditions. After 24 h, samples were fixed for electron microscopy. In the compatible combination, normal haustoria had developed [3]. In the incompatible host, haustoria of some infection hyphae were in cells with normal fine structure, others were in cells whose membranes were disrupted and whose mitochondria and plastids appeared swollen or disintegrated. After application of an autoradiographic emulsion (Ilford L 4 Gel), it could be seen that haustoria in the compatible cultiver had taken up the labeled

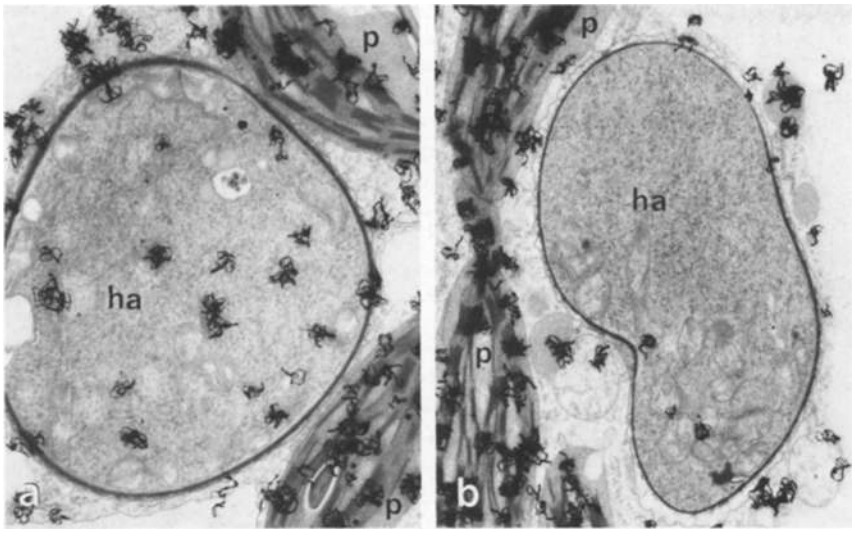

Fig. 1. (a) Haustorium of the bean rust in a cell of the compatible cultivar Favorit. Silver grains over the haustorium indicate transport of ^{3}H-lysine or its metabolites from host to parasite. (b) Haustorium in the incompatible cultivar Golden Gate Wax. It is only slightly labeled (*ha* haustorium, *p* plastid, ×8800)

material (Fig. 1a). In the incompatible bean, all haustoria in cells with intact fine structure were covered with only a few silver grains (Fig. 1b). This indicates that the uptake of lysine or its metabolites by haustoria in the incompatible cultivar is reduced. Obviously, a reduction in nutrient uptake by the haustorium occurs before the hypersensitive death of the host cell begins.

These results cannot yet be generalized. Previous morphologic studies have shown that, as in other host-parasite combinations (e.g. [4]), every combination of host and rust seems to be unique [5]. Different mechanisms might induce hypersensitive cell death [6]. This may explain why there is still much controversy about the significance of premature host cell necrosis in disease resistance [7]. Therefore, a study of other incompatible bean rust-bean combinations is in progress to see whether starvation of haustoria occurs generally.

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