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SPECIFICITY IN DEGRADATION OF ISOFLAVONOID PHYTOALEXINS

A. FUCHS and T. HIJWEGEN

Laboratorium voor Fytopathologie, Landbouwhogeschool, Wageningen*

Since the first observations by DE WIT-ELSHOVE (1969), that pea pathogens, contrary to non-pathogens, are able to degrade, and at least partially detoxify the pea phytoalexin pisatin, similar observations have been made for almost all isoflavonoid phytoalexins. This led to the idea, that breakdown of phytoalexins to products with a lower fungitoxicity is a prerequisite for pathogenic fungi to successfully colonize their host. There are, indeed, striking examples of closely related fungal species (e.g. *Ascochyta pisi* versus *A. fabae*; VAN 'T LAND et al. 1975a), or even *formae speciales* within one species (e.g. *Fusarium oxysporum* f. sp. *pisi* versus *F. oxysporum* f. sp. *lycopersici*; FUCHS et al. 1976, 1979), in which one, the pea pathogen, readily converts pisatin to less or even non-fungitoxic compounds, whereas the other, the non-pea pathogen, is not able to do so. In the case of *A. pisi* this led VAN 'T LAND et al. (1975b) to attach a significant importance to the ability of pisatin degradation in eliminating feed back inhibition of ascochitine biosynthesis by pisatin. Race specificity within *A. pisi* (cf. VAN 'T LAND & FUCHS 1973), on the other hand, could not be explained in terms of differential elicitation or degradation of phytoalexin (unpublished results).

Many examples have been found now which contradict the above-mentioned view, that all – and only – pathogens should be capable of metabolizing host phytoalexins. For instance, *Fusarium oxysporum* f. sp. *phaseoli* and *Thielaviopsis basicola*, both pathogenic to bean, did not metabolize phaseollin in shake cultures (VAN DEN HEUVEL & GLAZENER 1975). Further, *Aphanomyces euteiches*, although being a notorious pea pathogen, was found to be unable to degrade pisatin (PUEPPKE & VAN ETTEN 1976). On the other hand, metabolism of phytoalexins appeared not to be restricted to legume pathogens: for instance, *Cladosporium herbarum* detoxifies phaseollin to la-hydroxyphaseollone (VAN DEN HEUVEL & GLAZENER 1975), *Colletotrichum coffeanum* hydroxylates medicarpin to 6a,7-dihydroxymedicarpin (INGHAM 1976), *Septoria nodorum* converts phaseollin to 12,13-dihydrodihydroxyphaseollin (BAILEY et al. 1977), and *Fusarium anguioides* and *F. avenaceum* metabolize pisatin to 6a-hydroxyinermin (= 3,6a-dihydroxy-8,9-methylenedioxypterocarpan; LAPPE & BARZ 1978).

The alfalfa pathogen *Stemphylium botryosum* not only degrades the host phytoalexin medicarpin by reductive opening of the dihydrofuran ring to the corresponding isoflavan (vestitol; STEINER & MILLAR 1974), but can also, though less readily, convert the non-host phytoalexins maackiain (= inermin; HIGGINS

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1975) and phaseollin (HIGGINS et al. 1974) in a comparable way. The latter example poses the problem of substrate specificity: various substitutions of the pterocarpan skeleton do not seem to interfere with the fungus' ability to carry out the indicated degradation step. Other fungi seem to be more specific in this respect: *F. oxysporum* f. sp. *pisi* (FUCHS et al. 1976, 1979) as well as *Mycosphaerella pinodes* (FUCHS & DE VRIES, to be published) can degrade pisatin only after preceding demethylation at the C-3 position; the resulting 6a-hydroxyinermin is then readily metabolized to the corresponding 6a-hydroxyinermin-isoflavan (= 2',3,7-trihydroxy-4',5'-methylenedioxyisoflavan). Of 15 *Fusarium* species able to demethylate isoflavones, methylated at the C-7 position (BARZ et al. 1976), only the above-mentioned *F. anguioides* and *F. avenaceum* were able to also demethylate the corresponding methoxyl group (at the C-3 position) in pisatin, however, without further breakdown taking place. Although some non-pathogens thus clearly metabolize phytoalexins, in most cases the products formed retained part of their toxicity.

It should be realized that metabolism of phytoalexins has especially been studied in *in vitro* experiments. Since, as has been shown by DE WIT-ELSHOVE & FUCHS (1971), *in vitro* breakdown is affected by environmental conditions, such as sufficiently low carbohydrate concentrations in the culture medium, the results might not apply in an *in vivo* situation. In fact, results of *in vitro* phytoalexin degradation often do not reflect those of *in vivo* breakdown (INGHAM 1976). Critical consideration of experimental results is also justified as far as fungal sensitivity and fungitoxicity measurements are concerned. Not only very different techniques and media are used to assay for sensitivity, which makes these assays rather questionable (cf. VAN ETTEN 1973), but – in spite of widely varying phytoalexin sensitivity among fungi – fungitoxicity is often taken as fungitoxicity *per sé* and test organisms (e.g. *Cladosporium cucumerinum*) are used which do not allow conclusions as to specific fungal toxicity.

With these limitations in mind, the available data, however, seem to justify the contention, that pathogens, contrary to non-pathogens, are able to degrade host phytoalexins "beyond the fungitoxic level", a generalization to be understood in a quantitative as well as qualitative sense. However, it seems difficult to reconcile even this modified concept with the potential simultaneous occurrence of large numbers of phytoalexins in one plant species (e.g. 9 isoflavonoid compounds in green bean; 6 in soybean; cf. KEEN & BRUEGGER 1977), especially if it would necessitate the concurrent activation of very different degradative pathways in host pathogens. On the other hand, the widespread occurrence of, for instance, medicarpin in genera such as *Canavalia*, *Cicer*, *Medicago*, *Trifolium*, *Vicia*, *Vigna*, etc. (cf. GROSS 1977) renders a marked "discriminatory" function for medicarpin towards potential pathogens rather unlikely. Data on the distribution of isoflavonoid phytoalexins over the various genera within the *Leguminosae*, in relation to that of their pathogens as well as available data on degradative abilities among the latter (HIJWEGEN & FUCHS, to be published) leaves little reason to assign a really significant role to phytoalexins in legume plant diseases.

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