necrotic local lesions induced by AMV on primary bean leaves, and reduced their final number. The delay in formation of necrotic lesions was correlated with a similar delay in the appearance of induced b-proteins.

Band p1, and bands p1, p2 and p3 were also observed when necrotic lesions were induced by treatment with mercuric chloride, or triphenylphosphite, respectively. There was a strong correlation between cell injury and induction of new proteins in bean leaves.

Ethylene-induced chitinase: is it a pathogenesis-related protein?

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Ethylene or an ethylene-releasing compound, 2-chloroethylphosphonic acid (ethephon) have been found to induce pathogenesis-related proteins (PRs) in tobacco leaves (1). Ethylene has also been found to induce a highly active endochitinase in many plants; for example, in bean leaves, chitinase activity increased 30-fold within 24 h of an ethylene treatment (2). In fully induced leaves, more than 1% of the total protein, or more than 5% of the protein in a crude extract obtained at pH 5, consisted of chitinase. The enzyme was purified to homogeneity; its apparent mol.wt obtained by SDS-polyacrylamide gel electrophoresis was 30 000. While no endogenous substrate for purified chitinase was found in the plant, the enzyme readily attacked cell walls of a potential pathogenic fungus, *Fusarium solani*, and acted as a lysozyme on bacterial cell walls. This led us to the hypothesis that chitinase functions as a defense against pathogens (2).

We were interested to know whether ethylene-induced chitinase has any relation to PRs. We found that purified bean chitinase, due to its high isoelectric point, did not enter a non-denaturing polyacrylamide gel upon electrophoresis at pH 8.3, which is generally used for the separation of PRs. Thus, chitinase, at least from bean leaves, is different from the PRs currently investigated.

On the other hand, experiments with pea pods infected with *Fusarium solani* and with tobacco leaves reacting hypersensitively to tobacco mosaic virus showed that chitinase is also induced in the course of a pathogen attack. Although an enhanced synthesis of ethylene accompanied the plant's response in both cases, stress ethylene formation was not a necessary condition for chitinase induction, at least not in pea pods: a treatment of pea pods with aminoethoxyvinylglycine (AVG), a specific inhibitor of ethylene biosynthesis, prevented stress ethylene formation but did not affect the induction of chitinase. Thus, chitinase is induced by pathogenesis independently of ethylene.

We conclude that chitinase, while being different from the PRs presently studied, is a pathogenesis-related protein in a broader sense.

(1) Loon, L.C. van, 1977. Induction by 2-chloroethylphosphonic acid of viral-like lesions, associated proteins, and systemic resistance in tobacco. Virology 80: 417-420.
(2) Boller, T., Gehri, A., Mauch, F. & Vögeli, U., 1983. Chitinase in bean leaves: induction by ethylene, purification, properties, and possible function. Planta 157: 22-31.