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Global Regulation of Pectinases and Other Degradative Enzymes in *Erwinia carotovora* subsp. *carotovora*, The Incitant of Postharvest Decay in Vegetables

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Among the myriad bacteria present in our environment, only a small minority can cause diseases in plants, animals, or humans. It seems plausible that such bacteria have co-evolved with their hosts, acquiring traits that allow them to colonize host tissues and produce symptoms by triggering deleterious physiological responses or by destroying preformed structural components. An example of the latter is the elicitation of soft rot in a variety of plant tissues by a microbial consortium containing several *Erwinia* species as the primary component (Perombelon, 1987; Perombelon and Kelman, 1980). These

bacteria produce an array of degradative enzymes that act on middle lamella and plant cell wall polysaccharides and proteins, weakening or solubilizing them, and ultimately causing cell separation and death.

Most commonly found enzymes in cultures of soft-rot bacteria or in rotted (i.e., macerated) tissues are pectinases, cellulases, proteases, and phospholipases (Bateman and Millar, 1966; Chatterjee and Vidaver, 1986; Collmer and Keen, 1986). Theoretical considerations alone imply that these enzymes, by acting on such structural components as pectin, cellulose, wall proteins (including hydroxyproline-rich glycoproteins), and membrane phospholipids, could inflict physiological and physical stress to which host tissues may ultimately succumb. Indeed, genetic and biochemical evidence indicates a crucial role for some of the pectinases in the elicitation of soft rot (Barras et al., 1987; Boccara et al., 1988; Collmer and Keen, 1986; Payne et al., 1987; Ried and Collmer, 1988; Roberts et al., 1986; Thum et al., 1987). In contrast, there is no convincing evidence for cellulase, protease, or phospholipase activities in the elicitation of tissue maceration by *Erwinia* spp., with the possible exception of *E. carotovora* subsp.

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carotovora (Jones) Bergey et al. (Ecc) (see "Note added in proof"). Therefore, at this juncture, it would be premature to summarily dismiss ancillary roles of these enzymes for the following reasons. 1) Since these enzymes have the potential to damage the cell wall and cell membranes, one could argue that they may augment the action of pectinases. 2) The degradative actions of cellulases and proteases may generate oligomers, dimers, or monomeric residues that the pathogens could assimilate. Thus, these depolymerizing enzymes, as components of catabolic pathways for assimilating polymeric substances, may contribute to pathogen fitness. 3) These enzymes, along with an assortment of pectinases, are consistently detected in macerated tissue, although the temporal sequence of their appearance is not known. Thus, unless proven otherwise, we have to entertain the possibility that deployment of these enzymes at some stage during pathogenesis is of consequence.

The degradative nature of these enzymes, their presence in macerated tissues, and their purported role in pathogenicity also raise the issue of coregulation of their production. The notion that the production of these enzymes may be subject to a common (i.e., global) regulatory control was first prompted by the pleiotropic nature of *E. carotovora* mutants isolated by Beraha and Garber (1971). By nitrosoguanidine mutagenesis, they isolated mutants deficient in extracellular pectate lyase (Pel), polygalacturonase (Peh), cellulase (Ccl), protease (Prt), and phospholipase activities; subsequently, they obtained a one-step revertant that regained the ability to produce all of these enzymes. In Ecc strain 71, we also have isolated pleiotropic mutants using various transposons as well as the chemical mutagen, ethyl methanesulfonate (EMS). Properties of the mutants and the complementing DNA, discussed below, indicate the existence of a global regulatory network controlling extracellular enzyme production in the subspecies of *E. carotovora*.

Pectin lyase (Pnl), like Pel and endo-Peh, causes tissue maceration (Ishii, 1976; Tanabe and Kobayashi, 1987). Moreover, studies with fractionated Pels and Pnls indicate that a mixture of these enzymes elicits a more rapid substrate depolymerization than either enzyme alone (Tsuyumu et al., 1991). Thus, it is reasonable to suppose that Pnl may augment the tissue-macerating potential of Pel. This notion is supported by the observation that a Pnl mutant was severely attenuated in its ability to rot tobacco (*Nicotiana tabacum* L.) (Pirhonen et al., 1991).

Pnls produced by *Erwinia* spp. differ from the Pels in structural features and kinetic properties (Chatterjee et al., 1991a). A particularly remarkable difference lies in the mode of regulation. In most *Erwinia* spp., Pnl production, but not Pel, is induced after exposing bacteria to DNA-damaging agents such as UV light, mitomycin C, or nalidixic acid (Itoh et al., 1982; McEvoy et al., 1990; Tsuyumu and Chatterjee, 1984; Zink et al., 1985). Other traits that are generally coincided with Pnl include bacteriocin (i.e., carotovoricin, Ctv) production, temperate bacteriophage induction, or cellular lysis (Lss) induction. Coregulation of these traits by DNA-damaging agents is reminiscent of SOS gene expression in *E. coli* (Little and Mount, 1982; Walker, 1984, 1987). (SOS gene expression is activated by agents and conditions that either damage DNA or interfere with DNA replication.) Indeed, we have determined that the activation of Pnl and Ctv production and Lss induction, like the activation of SOS responses, requires a RecA function (Zink et al., 1985). However, our observations, summarized below, also indicate that the stimulation of *pnlA* expression, the structural gene for pectin lyase, in response to DNA-damaging agents requires a transcriptional activator (RdgB), which is in sharp contrast to the negative regulation of SOS genes. Since Rdg function is also required in Ctv production and Lss induction, we postulate that *pnlA*, *ctv*, and *lss* represent a subset of damage-inducible genes of Ecc. Gene expression is controlled by RecA and Rdg.

PLANT METABOLITE-ACTIVATED EXTRACELLULAR ENZYME PRODUCTION

Previous studies had indicated that pectate, which markedly stimulated pectate lyase production in *E. chrysanthemi* Burkholder et al. strain EC16 (Chatterjee et al., 1979, 1985, 1991c), was only marginally effective in Ecc (Chatterjee et al., 1991b). In *E. chrysanthemi*,

genetic and physiological evidence has also indicated that intermediates of the pectate catabolic pathway (viz., keto-deoxyuronate, ketodeoxygluconate, or both) serve as inducers (Chatterjee et al., 1985; Collmer and Bateman, 1981, 1982; Condemine et al., 1986; Condemine and Robert-Boudouy, 1987; Hugouvieux-Cotte-Pattat and Robert-Boudouy, 1987). The lack of a significant effect of pectate in Ecc, therefore, raised the following possibilities. 1) The intracellular pool of ketodeoxyuronate remains low, and hence the *pel* genes are never fully induced when pectate is provided as the inducer source. 2) Conversely, the pool sizes of catabolic intermediates are high when pectate is the inducer source, and, as Tsuyumu (1977) postulated, the high levels cause catabolite repression" of Pel production. 3) It also is conceivable that a metabolite other than one derived from the pectate catabolic pathway is the "true" inducer for the Ecc enzymes. The findings discussed below support the last hypothesis.

In addition to sustaining a low Pel production level in Ecc, pectate did not significantly stimulate the production of other extracellular enzymes such as protease and cellulase, yet high levels of all these activities are consistently present in rotted tissue. These findings prompted the notion that plant tissues provide a signal(s) that simultaneously activates expression of genes specifying extracellular enzymes. To test this idea, we determined the levels of extracellular enzymes in AC5006, a LacZ derivative of the Ecc strain 71, grown in salts-yeast extract-glycerol (SYG) medium supplemented with pectate or extracts from celery (*Apium graveolens* L.) petioles, potato (*Solanum tuberosum* L.) tubers, or carrot (*Daucus carota* L.) roots. Specific activity of Pel was highest in SYG + celery extract, followed by SYG + potato extract, SYG + carrot extract, SYG + pectate, and SYG. Ccl and Prt levels also were much higher in SYG + celery extract than in SYG or SYG + pectate. Stimulating extracellular enzyme production in Ecc with crude celery extract could be a response to separate signals, each activating a specific enzyme-producing system (i.e., Pel, Ccl, or Prt), or a response to a common signal that activates a global regulatory system specifically activating extracellular enzyme production.

The dogma that pectate is the universal inducer source for Pel production also is not supported by recent observations in *E. chrysanthemi*. For example, *pelA* expression is not stimulated to any significant extent by pectate in a derivative of *E. chrysanthemi* strain EC 16 (Chatterjee et al., 1991 c). In addition, Beaulieu and Van Gijsegem (1990) have provided genetic evidence for plant metabolite-activated *pelA* expression in *E. chrysanthemi* strain 3937. Kelemu and Collmer (1990) made a remarkable observation: in derivatives of *E. chrysanthemi* strain EC 16, wherein all the known *pel* genes (i.e., *pelA*, *pelB*, *pelC*, and *pelE*) had been inactivated (Ried and Collmer, 1988), a new set of Pel species was produced when bacteria were grown in the presence of plant cell wall. This phenotype apparently resulted from the activation of expression of normally silent *pel* genes.

Some of our recent observations with *E. rapontici* (Millard) Burkholder (Choi et al., 1991) are also similar to those of Kelemu and Collmer (1990). *Erwinia rapontici* strains do not produce Pel, Peh, or Prt in artificial media supplemented with pectate or pectin, yet they can macerate plant tissue, albeit at a slow rate. In extracts of rots caused by *E. rapontici* strains, we detected Pel activity that caused electrolyte loss and cell death in potato tuber tissue. Multiple Pel species were detected in rot extracts, and the enzyme profile was variable, depending on the plant tissue and bacterial strain. Plant cell wall preparations also activated Pel production in *E. rapontici*; however, the inducer activity was lost upon autoclaving. Since plant extracts that activated extracellular enzyme production in Ecc (see above) did not stimulate Pel production in *E. rapontici*, it is reasonable to assume that these bacterial systems respond to different plant signals. Further investigations should help identify signal(s) activating enzyme production in *E. rapontici* and the regulatory gene(s) responding to plant signal(s).

GLOBAL REGULATION OF EXTRACELLULAR ENZYME PRODUCTION

Genetic and physiological studies have unraveled two global systems controlling extracellular pectate lyase production and protease and cellulase production in Ecc: one requires the functions of the genes designated as *aep* (activator of extracellular protein production)

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Integrating Biological Control into Postharvest Disease Management Strategies

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Technological advances in regular (cold, ambient atmosphere; RA) and controlled-atmosphere (CA) storage, computerized sorting and grading, and mechanical processing and packing of apple (*Malus domestica* Borkh.) and other tree fruits allow the industry to harvest, store, and market large crops efficiently. Unfortunately, the same postharvest diseases that occurred 60 years ago still cause economic losses (Baker and Heald, 1932; Heald and Ruehle, 1931), although perhaps not to the same extent. Blue mold (caused by *Penicillium expansum* Link), gray mold (caused by *Botrytis cinerea* Pers.:Fries), and mucor rot (caused by *Mucor piriformis* E. Fisch.) cause economic losses for growers and packers of apples and pears (*Pyriscommunis* L.) every year. Bull's eye rot of apple and pear{ caused by *Cryptosporiopsis malicorticis* (Cordl.) Nann. [*Peziclla malicorticis* (H. Jacks.) Nann.] } causes episodic losses when environmental conditions at petal fall and harvest are conducive to infection. Losses caused by these diseases historically have been managed primarily by applying fungicides, although interest in integrated postharvest disease control has increased recently (Willett et al., 1989). Several biological and regulatory factors have spurred development of alternative disease management approaches. Development of resistance in pathogens to fungicides, particularly to benzimidazoles (Rosenberger and Mayer, 1979; Spotts and Cervantes, 1986), has limited or eliminated the effectiveness of these chemicals for postharvest use in some areas of the United States. Some countries that import fruit from the United States do not

allow residues of some fungicides, such as N-[(trichloromethyl)thio]-4-cyclohexene- 1,2-dicarboximide (captan). Other countries, such as Japan, allow no residues of any postharvest fungicide on fruit imported from the United States. Additionally, emotional debates over chemical residues in food products (National Research Council, 1987), highlighted by the Alar (n-dimethylamino succinamic acid) controversy, have made some chemical companies reluctant to pursue re-registration of effective materials. Developing non- or reduced-chemical postharvest disease management strategies has become an economic imperative rather than an option.

Biological control of plant disease is not anew concept, but efforts to apply biological control theory to postharvest diseases of fruit are recent, as indicated by the scarcity of references to postharvest biocontrol in general texts and in reviews of biocontrol of plant pathogens (Baker and Cook, 1982; Cook and Baker, 1983; Lewis and Papavizas, 1991). Promising postharvest biocontrol agents have been identified, and significant efforts are being made to develop them for commercial use. Pioneering work by Tronsmo and Raa (1977) investigated the use of the filamentous fungus *Trichodermapseudokoningii* Rifai as an antagonist against gray mold (caused by *B. cinerea*) of strawberry (*Fragaria xananassa* Duch.). Biocontrol yeasts, including *Acremonium breve* (Sukapure & Thirumalachar) W. Gains (Janisiewicz, 1988), *Candida guilliermondii* (Castellani) Langeron & Guerra [as *Debaryomyces hansenii* (Zopf) Lodder & Krieger-van Rij] (Chalutz and Wilson, 1990; McLaughlin et al., 1990), *Hanseniaspora uvarum* (Niehaus) Shehata, Mrak & Phaff (McLaughlin et al., 1989), *Cryptococcus laurentii* (Kuff.) C.E. Skinner, *C. flavus* (Saito) Phaff & Fell, and *C. albidus* var. *aerius* (Saito) Phaff & Fell (Roberts, 1990a, 1990b, 1991), are effective against various postharvest decays in several fruits

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