

Stahmann M A, Clare B G & Woodbury W. Increased disease resistance and enzyme activity induced by ethylene and ethylene production by black rot infected sweet potato tissue. *Plant Physiol.* 41:1505-12, 1966.
[Dept. Biochemistry, Univ. Wisconsin, Madison, WI]

Exposure of susceptible sweet potato root tissue to ethylene induced a resistance to infection by *Ceratocystis fimbriata* and an increase in peroxidase and polyphenol oxidase. Susceptible tissue inoculated with pathogenic or nonpathogenic strains of *C. fimbriata* that induced resistance formed more ethylene than tissue inoculated with strains that did not induce resistance. Ethylene may diffuse from infected areas into adjoining tissue to initiate metabolic changes that lead to disease resistance. [The SC[®] indicates that this paper has been cited in over 115 publications since 1966.]

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"I wanted to learn how genes for disease resistance protected plants and, since genes control protein synthesis, I began a study of the proteins from resistant and susceptible plants both before and after inoculation with viral, bacterial, or fungal pathogens. With Ikuzo Uritani,¹ I inoculated cut surfaces of sweet potato tissues with the fungus *Ceratocystis fimbriata*. Typical black rot symptoms developed in only susceptible tissues; fungal growth was confined to the surface of resistant tissues. Immunoelectrophoresis showed two new antigens in resistant tissues, but not in healthy tissue or inoculated susceptible tissue. These new antigens were identified as a peroxidase and a polyphenol oxidase which formed in the underlying cells of resistant tissue.

"With Darell Weber,² I showed by chromatography and gel electrophoresis changes in isozyme patterns of tissue below the surface of sweet potato roots infected by *C. fimbriata*. Peroxidase

activity increased and new isozymes of peroxidase appeared.

"Inoculation of susceptible sweet potato tissue with nonpathogenic isolates of *C. fimbriata* induced a resistance to subsequent challenges by pathogenic isolates.³ This resistance was limited to cells adjacent to the surface inoculated with the nonpathogen and protected against some other pathogens.

"It was observed that the activity of peroxidase and polyphenol oxidase was unexpectedly high in uninoculated tissue incubated in closed containers with infected tissue.⁴ This suggested a volatile substance. I wondered if this could be ethylene and could ethylene induce resistance to the spread of the fungus and the synthesis of peroxidase and polyphenol oxidase. However, we had no ethylene. So a piece of susceptible sweet potato tissue was incubated above an apple which would liberate ethylene. The piece was inoculated two days later; fungal growth was confined to the surface layer as with resistant tissue.

"Gas chromatography showed that ethylene was produced by sweet potato tissue infected with *C. fimbriata* and that more ethylene was produced by tissues inoculated with those isolates that induced resistance. Exposure of susceptible tissue to 8 ppm ethylene prior to inoculation with the pathogenic isolate induced a resistance response and the formation of new isozymes of peroxidase and polyphenol oxidase. Ethylene induced a ten- to 1,000-fold increase in oxidase activity. These results indicated that ethylene was the stimulus or hormone that diffused from infected sites into adjoining tissue to induce oxidase synthesis and blocking of further penetration by the fungus. Our studies⁵ of protein polymerization by quinones generated by plant oxidative enzymes may provide a biochemical basis for this induced resistance.

"I think this paper has been highly cited because it clearly indicated that ethylene could increase the resistance of sweet potato tissue to infection by *C. fimbriata* and that associated with this induced resistance was a large increase in the activity and isozymes of peroxidase and polyphenol oxidase. At the time our paper was published, neither this hormonal activity of ethylene nor the induction of resistance in plants by non-pathogens was widely recognized."

1. Uritani I & Stahmann M A. Changes in nitrogen metabolism in sweet potato with black rot. *Plant Physiol.* 36:770-82, 1961.
2. Weber D J & Stahmann M A. Ceratocystis infection in sweet potato: its effect on proteins, isozymes, and acquired immunity. *Science* 146:929-31, 1964.
3. _____, Induced immunity to ceratocystis infection in sweet potato root tissue. *Phytopathology* 56:1066-70, 1966.
4. Clare B, Weber D J & Stahmann M A. Peroxidase and resistance to ceratocystis in sweet potato increased by volatile materials. *Science* 153:62-3, 1966.
5. Leatham G F & Stahmann M A. In vitro polymerization by quinones of free radicals generated by plant or fungal oxidative enzymes. *Phytopathology* 70:1135-40, 1980.