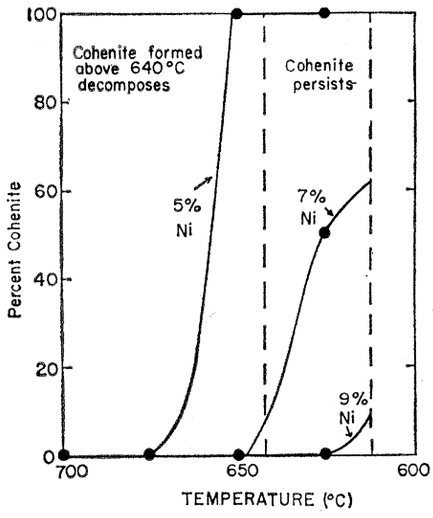

Fig. 4. Cumulative percentage of cohenite formed during cooling plotted against temperature for 3 meteorite compositions, each containing 0.3 percent carbon. Ordinate represents percentage of cohenite present with respect to the total cohenite which would form if all the carbon present were in the form of cohenite. The vertical dashed line at right is 610°, below which cohenite does not form.

in the alloy was plotted as a percentage. Amounts were obtained from the isothermal phase diagrams by the lever law. The diagram is plotted for a bulk carbon content of 0.3 percent and is little changed by alterations in carbon content within the meteoritic range.

The occurrence of cohenite in terrestrial basalts and in meteorites other than those containing 6 to 8 percent Ni may be explained by the fact that these bodies cooled rapidly (from textural evidence), thus preserving the cohenite, or by their very high C contents.

The presence or absence of cohenite in meteorites may be explained in terms of a low pressure origin. The lack of cohenite in iron meteorites other than those containing 6 to 8 percent Ni is strong evidence against a high-pressure origin for these meteorites, because at high pressures cohenite is stabilized. Such meteorites formed in bodies of asteroidal size, or near the surface of larger bodies.

ROBIN BRETT

U.S. Geological Survey, Washington, D.C. 20242

References and Notes

1. J. F. Lovering, *Geochim. Cosmochim. Acta* **28**, 1745 (1964); J. D. Brown and M. E. Lipschutz, *Icarus* **4**, 436 (1965).
2. All percentages mentioned are by weight.
3. A. E. Ringwood, *Geochim. Cosmochim. Acta* **20**, 155 (1960).
4. M. E. Lipschutz and E. Anders, *ibid.* **24**, 83 (1961); E. Olsen, *ibid.* **28**, 609 (1964); A. E. Ringwood, *ibid.* **29**, 573 (1965).
5. A. E. Ringwood and M. Seabrook, *Geochim. Cosmochim. Acta* **26**, 507 (1962).
6. M. E. Lipschutz and E. Anders, *ibid.* **28**, 699 (1964).
7. S. H. Perry, *U.S. Nat. Museum Bull.* **184** (1944); E. P. Henderson and S. H. Perry, *U.S. Nat. Museum Proc.* **107**, 339 (1958); B. Mason, *Meteorites* (Wiley, New York, 1962); E. P. Henderson, personal communication (1965).
8. A. A. Yavnel, *Int. Geol. Rev.* **2**, 298 (1960).
9. J. D. Buddhue, *Popular Astron.* **54**, 149 (1946).
10. K. W. Andrews, *J. Iron Steel Inst.* **184**, 414 (1956); J. I. Goldstein and R. E. Ogilvie, *Amer. Inst. Mining Met. Eng. Trans.* **233**, 2083 (1965); M. Hansen and K. Anderko, *Constitution of Binary Alloys* (McGraw-Hill, New York, ed. 2, 1958); W. Heller and J. Branner, *Arch. Eisenhuettenw.* **35**, 1105 (1964); L. Kaufman, in *Energetics in Metallurgical Phenomena*, W. Mueller, Ed. (Science Publ., New York, 1966); T. E. Kihlgren and J. T. Eash, *Metals Handbook*, T. Lyman, Ed. (American Society of Metals, Cleveland, 1948); J. J. Lander, H. E. Kern, A. L. Beach, *J. Appl. Phys.* **23**, 1305 (1952); R. F. Mehl and C. Wells, *Am. Inst. Min. Met. Eng. Trans.* **125**, 429 (1937); J. C. Meijering, *Proc. Nat. Phys. Lab. Symp.* No. 9, **2**, paper 5A, 2–20 (1959); A. E. Ringwood and L. Kaufman, *Geochim. Cosmochim. Acta* **26**, 999 (1962).
11. This stabilization causes kamacite nucleation at lower temperatures than in the pure system, which may partly explain the supercooled nucleation of kamacite demanded by recent authors (*15*).
12. R. H. Hickley and A. G. Quarrell, *J. Iron Steel Inst.* **178**, 337 (1954); G. T. Higgins and G. V. Jeminson, *ibid.* **203**, 146 (1965).
13. E. H. Klein, *Stahl Eisen* **54**, 827 (1934).
14. R. Brett, in preparation.
15. J. A. Wood, *Icarus* **3**, 429 (1965); J. M. Short and C. A. Anderson, *J. Geophys. Res.* **70**, 3745 (1965); J. I. Goldstein and R. E. Ogilvie, *Geochim. Cosmochim. Acta* **29**, 893 (1965).
16. I thank E. H. Roseboom, A. E. Ringwood, B. J. Skinner, E. P. Henderson, and H. R Shaw for suggestions. The kinetic study was done during the tenure of a postdoctoral fellowship at the Geophysical Laboratory, Carnegie Institution of Washington. Publication authorized by Director, U.S. Geological Survey.

11 February 1966

Peroxidase and Resistance to Ceratocystis in Sweet Potato Increased by Volatile Materials

Abstract. *Increased peroxidase activity and resistance to black rot was found in sweet potato roots incubated above infected roots in closed containers when compared with similar tissue incubated above uninfected roots. The peroxidase increases were detected in unpurified extracts and in extracts subjected to gel electrophoresis.*

Certain isolates of the fungus *Ceratocystis fimbriata* Ell. and Halst. are pathogens of sweet potato [*Ipomaea batatas* (L.) Lam.] and produce black rot in susceptible sweet potato roots. Activity of peroxidase and polyphenol oxidase is higher in sweet potato tissue inoculated with pathogenic isolates of *C. fimbriata* than it is in uninfected tissue; the increased activity extends into the host tissue beyond the area containing the pathogen (*1*). Recently similar changes in these enzymes were produced by inoculating resistant or susceptible sweet potatoe roots with nonpathogenic strains of *C. fimbriata* (*2, 3*). Normally susceptible tissue which had been inoculated in this way and which exhibited increased enzymic activity was resistant to subsequent infection by pathogenic isolates of *C. fimbriata* (*2, 4*). It appeared from these results that increased peroxidase and polyphenol oxidase activity was associated with resistance to black rot in sweet potato roots.

During the course of this work it was observed that the activity of these enzymes was unexpectedly high in uninoculated, susceptible tissue, used as experimental controls, which was incubated in containers together with tissue infected with pathogenic *C. fimbriata*. This finding suggested that some volatile material from the infected tissue had stimulated the changes in enzymic activity; the material may perhaps have also induced resistance in the uninfected tissue.

To investigate these possibilities, healthy sweet potato tissue was incubated together with either healthy or diseased tissue, and changes in the enzymic activity and susceptibility to black rot were evaluated. Four susceptible varieties (Jersey Orange, Julian, Maryland, and Nemagold) and one resistant variety (Sunnyside) of sweet potato were used (*5*). Roots were surface-sterilized with sodium hypochlorite solution and cut transversely into slices 2 cm thick. Healthy slices or slices infected with *C. fimbriata* (donors) were placed at the bottoms of sterilized glass containers. Healthy (receptor) slices were supported on sterile stainless-steel grids above the other slices in the containers. The containers were sealed and incubated at room temperature for 2 days. The groups of healthy receptor slices were then removed from the containers. Some slices from each container were inoculated with hyphal suspensions of *C.*

Table 1. Peroxidase activity in extracts of uninfected sweet potato root tissue incubated for 2 days above either uninfected sweet potato root tissue or root tissue infected with a pathogenic isolate of *Ceratocystis fimbriata*.

Variety	Peroxidase activity*	
	Uninfected tissue	Infected tissue
Jersey Orange†	0.8	7.2
Julian†	2.8	9.6
Maryland†	4.4	19.0
Sunnyside‡	6.2	22.0

* Increase in optical density, per minute, per milliliter of extract, at 420 mμ. † Susceptible to *C. fimbriata*. ‡ Resistant to *C. fimbriata*.

fimbriata and incubated in steril containers at room temperature and 90 percent humidity. Other slices from each container were cut into cylinders (18 mm in diameter). Discs 1 mm thick were cut transversely from the cylinders and were extracted under nitrogen in an equal volume of 0.1*M* tris-hydrochloride buffer (*p*H 7.4) containing 1 percent ascorbic acid and 12.5 percent sucrose (*6*). Extractions were carried out at 4°C with a modified sodium press (*7*). Extracts were centrifuged at 100,000 *g* for 15 minutes, and the supernatants were collected and stored at −15°C. Peroxidase activity in the extracts was detected after electrophoresis in polyacrylamide gels (*8*). Guaiacol ($2 \times 10^{-2}M$) and 0.3 percent hydrogen peroxide were used to detect peroxidases (*9*). Peroxidase activity in the extracts was measured by a modification (*10*) of the method of Mudd *et al.* (*11*). The reaction mixture contained 100 μmole of pyrogallol, 8 μmole of hydrogen peroxide, 100 μmole of acetate buffer (*p*H 5.0), sweet potato extract, and water in a total volume of 4 ml. Increases in optical density at 420 mμ and 25°C were measured at 15-second intervals.

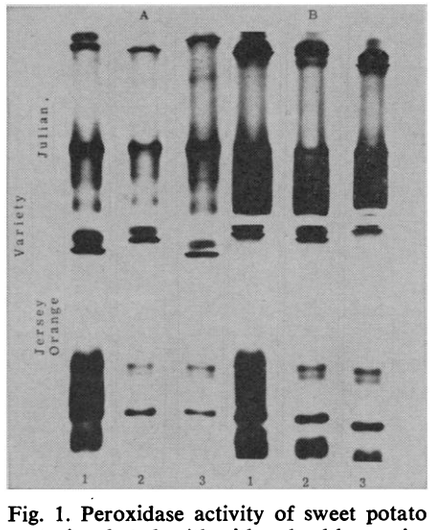

Fig. 1. Peroxidase activity of sweet potato roots incubated with either healthy or infected sweet potato root tissues. *A*, Root slices incubated together with uninfected root tissue. *B*, Root slices incubated together with root tissue infected with either *Ceratocystis fimbriata* (Julian), or *Rhizopus arrhizus* (Jersey Orange). Extracts from root slices were subjected to electrophoresis in polyacrylamide gels which were then stained for peroxidase activity. The numerals 1, 2, and 3 refer to the depth, in millimeters, below the surface of the root slice from which the samples were taken. The slices previously incubated with infected tissue (*B*) had increased peroxidase activity.

Much greater peroxidase activity was found in extracts from healthy tissue incubated with infected tissue than in extracts from tissue incubated with healthy tissue (Fig. 1, Table 1). On subsequent inoculation or challenge with the pathogen, uninfected tissues of normally susceptible varieties incubated above infected itssues were resistant to infection by *C. fimbriata* (Fig. 2). Tissue incubated with uninfected tissue remained susceptible (Fig. 2).

Uninfected tissue of the susceptible variety Jersey Orange was incubated together with similar tissue infected with *Rhizopus arrhizus* Fischer. This fungus rots sweet potato roots. Uninfected tissue inbubated in this way also had increased peroxidase activity (Fig. 1) and was resistant to *Ceratocystis fimbriata* infection (Fig. 2).

These results indicate that volatile materials from infected tissue can induce resistance to *C. fimbriata* infection in sweet potato roots which are normally susceptible to infection by this organism. Natural resistance in sweet potato tissue appears to be associated with a rapid increase in peroxidase and polyphenol oxidase activity following infection with *C. fimbriata* (*3*). Similar enzyme changes occur in susceptible tissue in which resistance has been induced by volatile substances from infected tissue. It therefore appears possible that these volatile substances may stimulate the defense mechanism in naturally resistant tissue.

The volatile materials from infected sweet potato responsible for the changes in uninfected tissue have not, as yet, been identified. A number of volatile compounds are produced by *C. fimbriata* cultures (*12*), and these may be involved. Ethylene is a common product of infected or injured plants (*13*, *14*) and can produce increases in respiratory activity such as

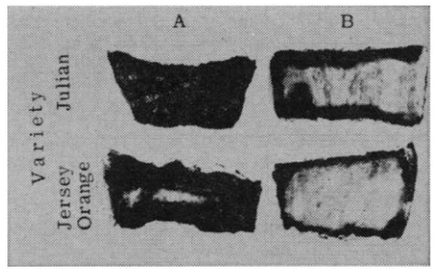

Fig. 2. Effect of volatile materials from infected tissue on the resistance of two varieties of sweet potato (Julian and Jersey Orange) which are normally susceptible to infection by *Ceratocystis fimbriata*. *A*, Root slices incubated for 2 days together with uninfected root tissue. *B*, Root slices incubated for 2 days together with sweet potato roots infected with either *C. fimbriata* (Julian) or *Rhizopus arrhizus* (Jersey Orange). All slices were inoculated with *Ceratocystis fimbriata*. The slices previously incubated with infected tissue (*B*) had acquired increased resistance to infection by *C. fimbriata*.

are found in infected plants (*13*). Increased peroxidase activity and resistance to *C. fimbriata* can be induced in susceptible sweet potato tissues by incubating them with apples, a known source of ethylene.

BRUCE CLARE*
DARRELL J. WEBER†
MARK A. STAHMANN
Department of Biochemistry, University of Wisconsin, Madison

References and Notes

1. I. Uritani and M. A. Stahmann, *Plant Physiol.* **36**, 770 (1961); N. Kawashima and I. Uritani, *Agr. Biol. Chem. Tokyo* **27**, 409 (1963); N. Kawashima, H. Hyodo, I. Uritani, *Phytopathology* **54**, 1086 (1964).
2. D. J. Weber and M. A. Stahmann, *Science* **146**, 929 (1964).
3. D. J. Weber, B. G. Clare, M. A. Stahmann, in preparation.
4. D. J. Weber and M. A. Stahmann, in preparation.
5. We thank C. E. Steinbauer, U.S. Department of Agriculture, Beltsville, Md., for supplying the sweet potatoes used in these experiments.
6. R. Heitefuss, D. J. Buchanan-Davidson, M. A. Stahmann, *Arch. Biochem. Biophys.* **85**, 200 (1959).
7. W. Woodbury and M. A. Stahmann, unpublished.
8. L. Ornstein and B. J. Davis, "Disc electrophoresis," preprint, Distillation Products Industries (1960).
9. K. Rudolph and M. A. Stahmann, *Nature* **204**, 474 (1964).
10. N. Kawashima and I. Uritani, *Plant and Cell Physiol.* **6**, 247 (1965).
11. J. B. Mudd, B. G. Johnson, R. H. Burris, K. P. Buchholz, *Plant Physiol.* **34**, 144 (1959).
12. R. P. Collins and M. E. Morgan, *Phytopathology* **52**, 407 (1962).
13. S. P. Burg, *Ann. Rev. Plant Physiol.* **13**, 265 (1962).
14. W. H. Smith, D. F. Meigh, J. C. Parker, *Nature* **204**, 92 (1964).

15. Supported by NIH grant AI 101 and by the Herman Frasch Foundation for Agricultural Research.

* Present address: Waite Agricultural Institute, University of Adelaide, Adelaide, Australia.
† Present address: Department of Biology, University of Houston, Houston, Tex.

24 March 1966