Role of Host-Selective Toxin in Colonization of Corn Leaves by Helminthosporium carbonum

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Supported in part by NSF Grant No. GB-24962.

Journal Article No. 5892 of the Michigan Agricultural Experiment Station.

Accepted for publication 5 July 1972.

ABSTRACT

Conidia of *Helminthosporium carbonum* race 1 germinated, formed appressoria, and penetrated epidermal cells of susceptible, tolerant (intermediate), and resistant corn cultivars. Further fungal growth was rapid in susceptible, restricted in tolerant, and confined to one or two cells in resistant leaves; resistance was evident by 16 hr after inoculation. *H. victoriae* (an oat pathogen) and a nonpathogenic *H. carbonum* isolate penetrated corn leaves but were confined to one or two cells. *H. carbonum*-susceptible but not resistant corn leaves were colonized by *H. victoriae* and nonpathogenic *H. carbonum* in the presence of *H. carbonum* (HC) toxin (2.0 μg/ml). When corn leaves were inoculated with *H. victoriae*, and HC-toxin was added after fungal growth had stopped, tissue colonization was successful. Prior inoculation with *H. victoriae* did not affect development of *H. carbonum* in susceptible corn leaves. The data support three conclusions: (i) HC-toxin is required for colonization of susceptible corn tissue by *H. carbonum*; (ii) dead or seriously damaged cells are not required for successful colonization (disruptive effects were not evident for more than 20 hr after inoculation); and (iii) inhibitory compounds produced by corn cells do not account for resistance to *H. carbonum* or to homologous pathogens.

Phytopathology 63:24-29

Additional key words: phytoalexin, disease resistance.

Helminthosporium carbonum Ullstrup race 1, the causal fungus of leaf spot of corn (*Zea mays* L.), produces a host-selective toxin (HC toxin) which is required for pathogenicity to certain corn genotypes (9, 11, 12). However, the role of toxin in initial colonization or infection of corn leaves is not established. It is sometimes implied that colonization of tissues by toxin-producing fungi is not typical of infection by foliage-invading fungi in general.

Experiments described below were designed to provide better understanding of pathogenesis by *H. carbonum* and disease resistance by corn. Included are: (i) a histological study of penetration and colonization, to determine whether or not colonization of host tissue by *H. carbonum* is typical of most fungal pathogens; (ii) an evaluation of the role of HC-toxin in colonization, which is defined as continued development of the fungus beyond initial penetration of the epidermal cell wall; (iii) an evaluation of hypothetical host-produced inhibitors as factors which limit fungal growth in resistant tissue; and (iv) experiments on electrolyte leakage after inoculation, because increased leakage is indicative of disruption of the host cell (10, 13). An abstract describing part of this work was published (1).

MATERIALS AND METHODS.—Two near-isogenic corn hybrids were used in most experiments; Pr × K61 is susceptible and Pr 1 × K61 is resistant to *H. carbonum* race 1. Corn inbred P8, with an intermediate or tolerant reaction to *H. carbonum* race 1 and to its toxin (6), was used in some experiments. Seeds were supplied by A. J. Ullstrup, Purdue University, Lafayette, Ind. Plants were grown at ca. 23 C with an 18-hr photoperiod, in vermiculite plus White's nutrient solution. The second true leaves of 10- to 11-day-old plants were used in most experiments.

The *H. carbonum* race 1 isolate used in most experiments was a hybrid between wild-type *H. carbonum* and *H. victoriae* Meehan & Murphy. The race 1 isolate produced both HC and *H. victoriae* (HV) toxins (11), but had the same specificity to corn lines, and the same histological interactions (3) as did the wild-type isolates of *H. carbonum* race 1. A hybrid isolate which produced neither toxin was used also. The *H. victoriae* isolate produced HV toxin but not HC toxin, and was a parent of the HC toxin-producing and the nonpathogenic isolates. Thus, the essential difference between the *H. victoriae* and *H. carbonum* isolates was controlled by one gene pair, as shown in previous work (11). Conidia were produced on filter paper as described previously (15). Droplets of water or toxin solution containing conidia were placed on the upper surfaces of excised leaves (10 or 20 conidia/mm^2), which were held in a chamber at 100% relative humidity. At various times after inoculation, the leaves were examined for fungal development. In previous work (15), comparable results were obtained with inoculated excised leaves and with inoculated leaves on intact plants.

Leaves were sectioned with a Hooker (Lab Line) fresh tissue microtome (2). Cross-sections of leaves (20-60 μ thick) were collected in water and transferred to a drop of water on a glass slide. Sections were stained with 0.1% cotton blue in lactophenol, immediately rinsed with lactophenol to

remove excess stain, and mounted in lactophenol for microscopic examination. Photographs were taken of the sections, and drawings were made from the photographs. All histological data are based on the results of at least three experiments using different sets of leaves each time.

HC toxin was prepared by established procedures (4, 9) using chloroform extractions, ethanol-ether

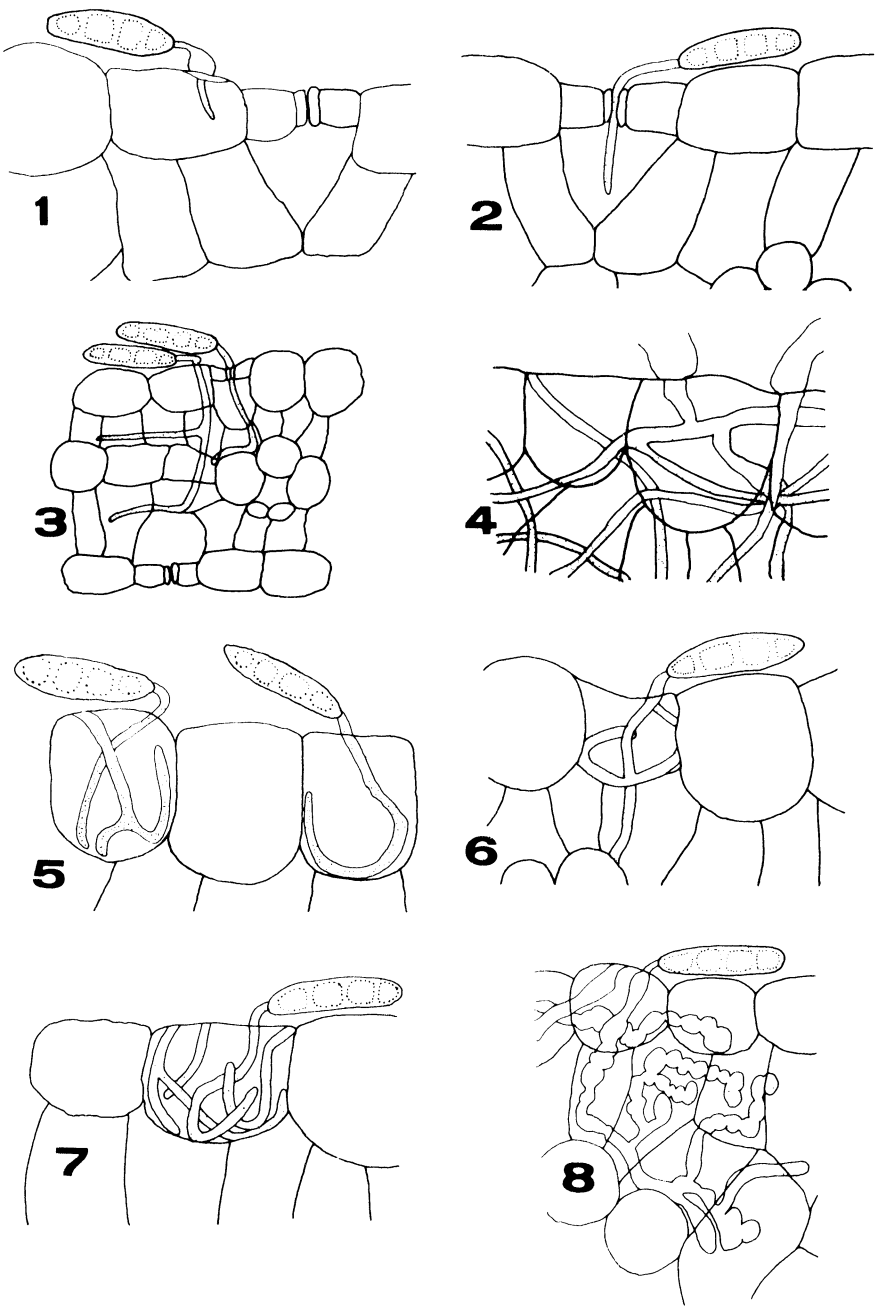

Fig. 1-8. 1) Direct penetration of susceptible corn leaf by *Helminthosporium carbonum*, 12 hr after inoculation. Conidium, appressorium, and hypha are shown. 2) Stomatal penetration of susceptible leaf, 12 hr after inoculation. 3) *H. carbonum* in susceptible leaf, 24 hr after inoculation. 4) *H. carbonum* in susceptible leaf, 48 hr after inoculation. The bases of two conidiophores are shown on the upper leaf surface. 5) *H. carbonum* in a resistant corn leaf, 20 hr after inoculation. 6, 7) *H. carbonum* in resistant leaves, 48 hr after inoculation. 8) *H. victoriae* in a corn leaf resistant to *H. carbonum*, 48 hr after inoculation with conidia plus HC toxin (40 μg/ml). Hyphae were bulbous, and 8 to 12 cells were invaded per infection site.

extractions, and gel filtration. Toxic fractions from Bio-Gel P-2 columns caused 50% inhibition of root growth by susceptible corn seedlings at 0.2 μg/ml. This activity is comparable to that of crystalline toxin (9).

Electrolyte leakage from inoculated and control leaves was determined with a Model RC 16B1 Industrial Instruments conductivity bridge, using dip type electrodes (K = 1.0 or 0.1). Replicate leaf samples were used for each treatment. At various times after inoculation, leaves were rinsed, cut into 1.0- to 2.0-cm^2 pieces, and placed in prewashed cheesecloth bags (each, 0.5 g fresh wt). Each bag was then placed in a 300-ml flask containing 50 ml glass-distilled water, and incubated on a reciprocal shaker at 100 strokes/min; water was changed 4 times during a 2-hr washing period. Conductivity measurements of the ambient solutions were then taken at intervals.

RESULTS.—*Development of H. carbonum in susceptible, tolerant, and resistant corn leaves.*—Conidia germinated on the leaf surfaces of susceptible, tolerant (intermediate), and resistant corn by 2 to 4 hr after inoculation. Appressoria were often small, but their formation appeared to be equal on all three corn types. The fungus usually penetrated the intact leaf surface, but an occasional hypha entered through a stomate (Fig. 1, 2). Most units had penetrated one or two epidermal cells by 12 hr; a few conidia did not complete the penetration process until 24 hr after inoculation.

TABLE 1. Relative colonization of susceptible, tolerant (intermediate), and resistant corn leaves by *Helminthosporium carbonum* race 1

	No. of cells invaded at[a]		
Leaf type	12 hr	24 hr	48 hr
Susceptible	1-2	20-30	>1,000
Tolerant	1-2	8-10	20-30
Resistant	1-2	2-4	2-4

[a] Cells invaded per infection site. Comparable results were obtained in three different experiments.

TABLE 2. Effect of HC toxin at several concentrations on colonization of corn leaves by *Helminthosporium victoriae*

	No. of cells invaded at 48 hr[a]	
Fungus and treatment	Susceptible	Resistant
H. carbonum (control)	>1,000	2-4
H. victoriae (control)	2-4	2-4
H. victoriae + HC toxin (0.2 μg/ml)	2-4	2-4
H. victoriae + HC toxin (2.0 μg/ml)	>1,000	2-4
H. victoriae + HC toxin (20.0 μg/ml)	>1,000	4-12
H. victoriae + HC toxin (40.0 μg/ml)	>1,000	4-12

[a] Cells invaded per infection site. Comparable results were obtained in three different experiments.

In susceptible corn, most fungal growth at 24 hr was in the half of the leaf adjacent to the inoculated surface (Fig. 3), but some hyphae had penetrated completely through the leaf. Approximately 25 susceptible host cells were invaded per average infection site in 24 hr. The fungus continued to grow inter- and intracellularly; by 48 hr there was dense fungal growth for some distance around each infection site (Fig. 4). Conidiophores were evident on the upper leaf surface and there was some tissue disintegration. However, most individual host cells in infected tissue did not differ visibly from healthy cells in control leaves. Inoculations with 10 and 20 conidia/mm^2 gave essentially the same results on susceptible leaves.

The initial stages of fungal development were the same in susceptible and tolerant (intermediate) corn leaves. Differences in fungal growth were evident by 24 hr after inoculation, when only 8 to 10 cells of tolerant corn were invaded. The amount of fungal growth in tolerant leaves at 48 hr was comparable to that in susceptible leaves at 24 hr (Table 1). No conidiophores were observed on tolerant corn leaves at 48 hr after inoculation.

In resistant corn, only one or two epidermal cells/infection site were invaded by 16-20 hr after inoculation (Fig. 5), whereas 10 to 20 susceptible cells were invaded. Usually there was little or no further development in resistant tissue (Fig. 6, 7). Thus, resistance was expressed as inhibition of hyphal growth as early as 16 hr after inoculation, which is somewhat later than is the case with *H. victoriae* in resistant oats (15). Occasionally, the fungus continued to grow within a single invaded host cell until the lumen was filled (Fig. 7). Otherwise, the appearance of invaded resistant cells and cells in noninoculated plants was similar at 48 hr after inoculation. Furthermore, the hyphae in resistant cells did not differ visibly at any time up to 48 hr from hyphae in cells of susceptible or tolerant corn. Inoculum concentration had a slight effect on invasion of resistant corn. With 10 conidia/mm^2, only 2 to 4 cells were invaded per infection site in 48 hr; with 20 conidia/mm^2, 4 to 10 cells were invaded.

Development of H. victoriae and nonpathogenic H. carbonum in corn leaves.—*H. victoriae* is pathogenic to oats but not to corn. Most conidia of *H. victoriae* and nonpathogenic *H. carbonum* germinated on the corn leaf within 4 to 5 hr after inoculation. Hyphae penetrated the epidermal cells by 12 hr, but further growth in tissues stopped less than 24 hr after inoculation. The fungus was thereafter confined to one or two cells, and no conidiophores developed. Similar limitation of both fungi occurred in corn that is susceptible, tolerant (intermediate), or resistant to *H. carbonum* race 1. The restriction of both fungi in all corn leaves was similar to the restriction of pathogenic *H. carbonum* in resistant corn (Fig. 5, 6, 7).

Effect of HC toxin on colonization of corn leaves by H. victoriae and nonpathogenic H. carbonum.—HC toxin at several concentrations (0.0, 0.2, 2.0, 20.0, and 40.0 μg/ml) was tested for its effect on

colonization of corn leaves by the homologous (7) pathogen, *H. victoriae*. Conidia were suspended in HC toxin solutions prior to droplet inoculation of *H. carbonum*-susceptible, tolerant, and resistant corn leaves. Leaf sections were examined at 12, 24, and 48 hr after inoculation. HC toxin at the lowest concentration (0.2 μg/ml) did not affect development of *H. victoriae* in any of the three corn lines (Table 2); the fungus was confined to a few cells. When HC toxin at 2.0 or more μg/ml was present with the inoculum, *H. victoriae* colonized *H. carbonum*-susceptible corn leaves within the first 48 hr, as readily and as extensively as did *H. carbonum* race 1 (Table 2). Conidiophores were evident by 48 hr after inoculation. Tolerant corn was also invaded by *H. victoriae* in the presence of HC toxin (2.0 μg/ml), to an extent comparable to that of *H. carbonum* in tolerant corn. There was a limited colonization of the leaf by 48 hr after inoculation. Other concentrations of HC toxin were not tested on tolerant corn.

The nonpathogenic hybrid isolate of *H. carbonum*, which produced neither toxin, was used in other experiments. When toxin was added with inoculum, this isolate colonized susceptible, but not resistant, tissue in the manner described for *H. victoriae*.

Colonization of resistant corn tissues by *H. victoriae* was not affected by HC toxin at 0.2 or 2.0 μg/ml. Toxin at 20 or 40 μg/ml allowed *H. victoriae* to invade a few more corn cells (Table 2). However, in the presence of these high levels of toxin, the hyphae of *H. victoriae* in resistant corn tissue were bulbous, varied greatly in diameter, and appeared as strings of spherical bodies (Fig. 8). This type of fungal growth was not observed under other conditions.

In another set of similar experiments, toxin was applied after fungal development had stopped. *H. carbonum*-susceptible and resistant corn leaves were inoculated with *H. victoriae* (10 conidia/mm^2). At 24 or 48 hr after inoculation, excess water in infection droplets was removed with a small pipet; droplets of HC toxin solution (2.0 or 20 μg/ml) were then added to the inoculation sites. Drops of water rather than the toxin-containing solution were added to control leaves. Fungal development was examined microscopically at 24 and 48 hr after addition of toxin. *H. victoriae* resumed growth in susceptible corn tissue when 20 μg HC toxin/ml were added to the infection site, regardless of the time (24 or 48 hr) between inoculation and toxin treatment (Table 3). Colonization and conidiophore development were evident 48 hr after toxin was applied. If inhibitory compounds were formed by corn cells, they were not effective in checking growth of the fungus in the presence of HC toxin. However, toxin concentration was critical, because *H. victoriae* failed to resume growth with HC toxin at 2.0 μg/ml. There may have been some dilution of toxin, perhaps to below a critical level, by the water remaining from the inoculation droplet. *H. victoriae* did not colonize *H. carbonum*-resistant corn tissue when HC toxin (20 μg/ml) was added at 0, 24, or 48 hr after inoculation.

TABLE 3. Effect of HC toxin (20 μg/ml) applied after inoculation on colonization of corn leaves by *Helminthosporium victoriae*

Time of toxin treatment[a]	No. of cells invaded at[b]		
	0 hr[c]	24 hr[c]	48 hr[c]
0	0	20-30	>1,000
24	2-4	20-30	>1,000
48	2-4	20-30	>1,000

[a] Hour after inoculation.
[b] Cells invaded per infection site. Corn is susceptible to *H. carbonum* but not to *H. victoriae*.
[c] Hour after toxin treatment. Comparable results were obtained in three different experiments.

Effect of inoculation with H. victoriae on colonization of corn leaves by H. carbonum.—Corn leaves inoculated with *H. victoriae* (10 conidia/mm^2) were challenge-inoculated 24 or 48 hr later with *H. carbonum* race 1. Some control leaves were inoculated (10 conidia/mm^2) with *H. carbonum* alone, and others were left without inoculation. *H. victoriae* had stopped growing, and was confined to one or two epidermal cells at the time of the challenge inoculation with *H. carbonum*. Leaf sections were examined 48 hr after challenge inoculation. *H. carbonum* developed equally in susceptible tissue that had been inoculated previously with *H. victoriae* and in susceptible tissue that was not previously inoculated with the homologous pathogen. Comparable results were obtained in each of three experiments. If phytoalexins or other hypothetical inhibitors were responsible for stopping growth of *H. victoriae*, they did not restrict subsequent development of *H. carbonum*.

Effect of infection on loss of electrolytes from corn leaves—Susceptible and resistant corn leaves were inoculated with *H. carbonum* or *H. victoriae* (10 or 20 conidia/mm^2). Water droplets without conidia were placed on control leaves. Inoculated and control leaves were held for various time periods in a moist chamber (100% relative humidity) prior to leakage assays. Two hr before leaching measurements were begun, leaves were cut into pieces, placed in cheesecloth bags, and rinsed with glass-distilled water. Conductivity determinations of fresh ambient solutions were started at 12, 18, 24, 30, and 36 hr after inoculation. When leaching of susceptible leaves was started 24 hr after inoculation with *H. carbonum* (10 conidia/mm^2), electrolyte losses were the same as losses from control leaves until 26 hr after inoculation; similarly treated, resistant leaves were only slightly affected at 30 hr after inoculation (Fig. 9). When leaching of susceptible leaves was started at 12 or 18 hr, there was no difference between inoculated and control leaves until the total time after inoculation was more than 20 hr. Susceptible leaves inoculated with 20 conidia/mm^2 lost more electrolytes than did control leaves by 20 hr.

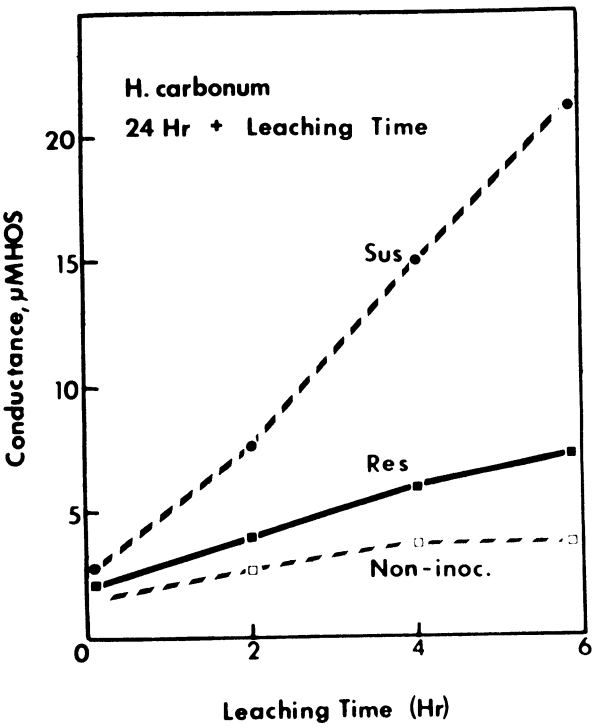

Fig. 9. Effects of *Helminthosporium carbonum* on leakage of electrolytes from susceptible and resistant corn leaves. All conductivity measurements began 24 hr after inoculation with 10 conidia/mm^2. Sus = susceptible inoculated leaves; Res = resistant inoculated leaves; non-inoc. = noninoculated resistant and susceptible control leaves. There were no differences in electrolyte losses by inoculated and control leaves at earlier times after inoculation.

Resistant leaves inoculated with 20 conidia/mm^2 lost slightly more electrolytes than did resistant leaves inoculated with 10 conidia/mm^2. *H. victoriae* (20 conidia/mm^2) had no effect on leakage of electrolytes from corn leaves of either type, even after 36 hr. The experiment was repeated 2 times with essentially the same results.

H. carbonum differs from *H. victoriae* in relative ability to disrupt the host cell. *H. victoriae* causes increased leakage from susceptible leaf tissue within 6 to 7 hr after inoculation (15); HV toxin causes drastic leakage immediately after leaves or other tissues are exposed (10). Apparently, *H. carbonum* and its toxin do not have a disruptive effect on the host cell for some time (14, 16). The notion that toxin-producing fungi only invade toxin-killed cells was not confirmed.

DISCUSSION.—These histological observations agree in general with those of Jennings & Ullstrup (3), and indicate that invasion of corn tissues by *H. carbonum* is typical of invasion by most leaf-infecting fungi. *H. carbonum* quickly colonizes and spreads in susceptible corn tissue, but usually is confined to one or two initially penetrated cells in resistant tissue. Nevertheless, the questions involved in our study differed from those of Jennings & Ullstrup (3), which account for some differences in procedures. For example, we had a special interest in determining the time of initial expression of disease resistance. A comparison of fungal growth in resistant and susceptible tissue indicates that resistance is expressed as early as 16 hr after inoculation.

The host-selective toxin produced by *H. victoriae* (HV toxin) was shown in a previous study to be required for colonization of susceptible oat leaves (15). Nonpathogenic mutants, which do not produce toxin, will colonize susceptible oats only when HV toxin is added at the site of penetration. These data, plus genetic and physiological evidence, show that HV toxin is required for pathogenicity of *H. victoriae* to certain genotypes of oats (12). However, HV toxin has rapid and drastic effects on many functions of the host cell which may not be typical of fungal infections in general. HC toxin has more subtle effects, including stimulation of growth and several metabolic processes (6, 14, 16). Therefore, many of the experiments with *H. victoriae* and its toxin were repeated, using *H. carbonum* and HC toxin.

These data presented here, plus the genetic (11) and physiological evidence (5), show that HC toxin is required for colonization of susceptible corn by *H. carbonum*. When HC toxin was present, *H. victoriae* (an oat pathogen) and a nonpathogenic isolate of *H. carbonum* invaded and colonized *H. carbonum*-susceptible leaves; without HC toxin, these fungi were not able to colonize. Leaves with an intermediate level of resistance to *H. carbonum* were invaded to a limited degree by *H. victoriae* when supplemented with HC toxin. Tissues resistant to *H. carbonum* and to HC toxin were not colonized by either *H. carbonum* or *H. victoriae* when supplemented with HC toxin. Therefore, resistance to the fungus appears to be based on insensitivity to toxin produced by the fungus.

Resistance of some plant tissues to pathogens has been induced by prior inoculation with homologous pathogens, or pathogens of other plants (7). However, resistance of corn to *H. carbonum* was not induced by prior inoculation with *H. victoriae*, which penetrated one or two epidermal cells in ca. 12 hr with little or no further development. After 24 or 48 hr, HC toxin was added at the site of penetration; *H. victoriae* then resumed growth in tissues susceptible to *H. carbonum*. These data show that hypothetical fungistatic compounds (phytoalexins), if produced at the site of penetration, did not prevent further development of the pathogen or the homologous pathogen. No other conclusion is possible with available evidence, unless we assume that toxin inactivates hypothetical inhibitors. This was shown not to be the case with HV toxin and the fungal inhibitor induced in oat leaves after fungal penetrations (8). Apparently, resistance is not an induced process in these cases. It is more logical to suggest that host-selective toxins induce susceptibility.

Inoculation of oat leaves with *H. victoriae* causes increased loss of electrolytes within 6-7 hr; the effect

was attributed to toxin released by the fungus (15). Such drastic effects of HV toxin have led to speculations that *H. victoriae* only invades dead cells. *H. carbonum* and HC toxin have much more subtle effects; there is little possibility that *H. carbonum* kills host cells in advance of initial colonization. The delay in induction of electrolyte loss from susceptible corn leaves by *H. carbonum* further supports a conclusion that dead or seriously damaged cells are not required for successful colonization. Furthermore, there is no evidence that *H. carbonum* (3, 5) or its toxin (6, 14) have deleterious effects on susceptible cells for some time after the fungus is established. In resistant cultivars, resistance is evident at an early time in relation to the time required for induction of electrolyte leakage, or any other known indications of damage to resistant or susceptible cells.

For the first 20 hr, HC toxin has a stimulatory rather than a disruptive effect on treated susceptible cells. The first effects detected to date were increases in uptake of NO_3^-, Na^+, Cl^-, 3-0-methylglucose, and leucine (16). Toxin at low concentrations stimulated growth of seedlings (6), and increased the incorporation of amino acids into alcohol-insoluble components of the cell (14). Such data indicate that HC toxin from the colonizing fungus does not have serious disruptive effects on host cells during the early stages of infection. However, it is evident from data presented here that toxin is required for colonization beyond simple penetration of the epidermal cell wall.

LITERATURE CITED

1. COMSTOCK, J. C., & R. P. SCHEFFER. 1970. The role of Helminthosporium carbonum toxin in corn leaf invasion. Phytopathology 60:1288 (Abstr.).
2. HOOKER, W. J. 1967. A microtome for rapid preparation of fresh sections of plant tissue. Phytopathology 57:1126-1129.
3. JENNINGS, P. R., & A. J. ULLSTRUP. 1957. A histological study of three Helminthosporium leaf blights of corn. Phytopathology 47:707-714.
4. KUO, M. S., & R. P. SCHEFFER. 1969. Factors affecting activity of Helminthosporium carbonum toxin on corn plants. Phytopathology 59:1779-1782.
5. KUO, M. S., & R. P. SCHEFFER. 1970. Comparative effects of host-specific toxins and Helminthosporium infections on respiration and carboxylation by host tissue. Phytopathology 60:1391-1394.
6. KUO, M. S., O. C. YODER, & R. P. SCHEFFER. 1970. Comparative specificity of the toxins of Helminthosporium carbonum and Helminthosporium victoriae. Phytopathology 60:365-368.
7. MATTA, A. 1971. Microbial penetration and immunization of uncongenial host plants. Annu. Rev. Phytopathol. 9:387-410.
8. NISHIMURA, S., & R. P. SCHEFFER. 1965. Interactions between Helminthosporium victoriae spores and oat tissue. Phytopathology 55:629-634.
9. PRINGLE, R. B., & R. P. SCHEFFER. 1967. Isolation of the host specific toxin and a related substance with nonspecific toxicity from Helminthosporium carbonum. Phytopathology 57:1169-1172.
10. SAMADDAR, K. R., & R. P. SCHEFFER. 1971. Early effects of Helminthosporium victoriae toxin on plasma membranes and counteraction by chemical treatments. Physiol. Plant Pathol. 1:319-328.
11. SCHEFFER, R. P., R. R. NELSON, & A. J. ULLSTRUP. 1967. Inheritance of toxin production and pathogenicity in Cochliobolus carbonum and Cochliobolus victoriae. Phytopathology 57:1288-1291.
12. SCHEFFER, R. P., & O. C. YODER. 1972. Host-specific toxins and selective toxicity, p. 251-272. *In* R. K. S. Wood, A. Ballio, & A. Graniti [ed.]. Phytotoxins in Plant Diseases. Academic Press, London.
13. WHEELER, H., & PENELOPE HANCHEY. 1968. Permeability phenomena in plant disease. Annu. Rev. Phytopathol. 6:331-350.
14. YODER, O. C., & R. P. SCHEFFER. 1969. Stimulatory effects of Helminthosporium carbonum toxin on corn seedlings. Phytopathology 59:1058-1059 (Abstr.).
15. YODER, O. C., & R. P. SCHEFFER. 1969. Role of toxin in early interactions of Helminthosporium victoriae with susceptible and resistant oat tissue. Phytopathology 59:1954-1959.
16. YODER, O. C., & R. P. SCHEFFER. 1971. Selective stimulation of ion uptake by Helminthosporium carbonum toxin. Phytopathology 61:918 (Abstr.).