Repression of Virulence in Rhizoctonia solani by Glucose and 3-O-Methyl Glucose

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ABSTRACT

An exogenous source of glucose resulted in reduced virulence of *Rhizoctonia solani* on 5-day-old cotton seedlings. Lesion areas were 32.5, 7.8, and 4.1 mm^2 after treatment with solutions of 14, 28, and 56 mM glucose, respectively, compared with 82.0 mm^2 for water controls. A solution of 28 mM glucose prevented the production of pectinase by *R. solani* in vitro. Other sugars were tested and, with the exception of arabinose, there was a direct relationship between utilization by *R. solani* as a source of carbon, inhibition of disease development, and repression of pectinase. Arabinose was a relatively poor carbon source for growth, and was only partially effective in reducing disease development. It did, however, repress pectinase production in the in vitro system. Disease development was also prevented by a solution of 5.2 mM 3-*O*-methyl glucose (MeG). In contrast to glucose MeG was active at lower concns and enhanced pectinase production in vitro. Growth of *R. solani* was reduced by MeG but this effect was overcome by the addition of a small quantity of glucose. In the presence of glucose infection cushions formed but the typical *R. solani* lesion did not develop. With MeG, however, the pathogen grew on the hypocotyl, but infection cushions were not formed. Combinations of glucose and MeG increased mycelial growth on the hypocotyl, but did not result in infection cushion formation. Glucose appears to interfere with disease development through prevention of tissue maceration by pectinolytic enzymes, whereas MeG appears to interfere with processes that lead to infection cushion formation.

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Rhizoctonia solani Kuhn is an important pathogen with a large host range and a world-wide distribution. Many isolates of *R. solani* produce infection cushions on the surface of the host plant (5, 7). Recent studies have shown that extensive host damage occurs beneath these cushions before penetration (11). Numerous investigations have clearly shown the importance of pectolytic enzymes in host damage by *R. solani* (2, 3,9). These enzymes are produced early in pathogenesis and are largely responsible for the sunken cortical lesions characteristic of this pathogen (9, 11).

Although pectolytic enzymes have an important role in disease caused by *R. solani*, it is not clear whether their production is the determining factor in pathogenesis. During an investigation of the influence of pathogen nutrition on virulence, it was observed that an exogenous supply of glucose reduced lesion development. A study of this phenomenon was initiated because of the potential value of a method to block pathogenesis in furthering our understanding of the pathogenic mechanisms of *R. solani*.

MATERIALS AND METHODS.—Five-day-old seedlings of cotton (*Gossypium hirsutum* L. 'Acala 4-42'), grown in the greenhouse, were placed on glass plates as previously described (10). Washed sand was used to support the seedlings and to cover the roots. Most of this work was done with an isolate of *R. solani* (R-21) obtained from diseased cotton growing in the Central Valley of California. This isolate was also pathogenic to bean, potato, and sugar beet. On cotton, numerous dome-shaped infection cushions were formed, followed by tissue maceration.

Inoculum was prepared by growing the pathogen on a medium containing 20.0 g glucose, 2.0 g L-asparagine, 1.75 g KH_2PO_4, 0.75 g $MgSO_4 \cdot 7H_2O$, 37.0 mg $CaCl_2 \cdot 2H_2O$, 0.8 mg $CuSO_4 \cdot 5H_2O$, 1.0 mg $FeCl_3 \cdot 6H_2O$, 0.5 mg $NaMnO \cdot 2H_2O$, 0.9 mg $ZnSO_4 \cdot 7H_2O$, and 0.3 mg $MnSO_4 \cdot H_2O$ in 1 liter of distilled water. Asparagine was added to the medium before autoclaving and glucose was autoclaved separately and added aseptically. The fungus was grown in still culture on 20 ml liquid medium in 9-cm diam petri dishes for 5 days at 28 C. Disks of mycelium 2 mm in diam were cut from the center of the mycelial mat, washed, and placed on the sand approximately 1 mm from the hypocotyls. Seedlings were incubated in a growth chamber under conditions of continuous light, at a temp of 28 C. Disease severity was evaluated by measuring the area of macerated tissue 48 h after inoculation. Exogenous nutrients were supplied by moistening the sand with 40 ml of a solution of the chemical.

RESULTS.—*Lesion reduction by glucose and 3-O-methyl glucose.*—Solutions of glucose and 3-*O*-methyl glucose (MeG) were used to moisten the sand supporting the cotton seedlings. Inoculum disks were placed on the same in contact with the liquid. The presence of an external supply of glucose of 3-*O*-methyl glucose resulted in a definite reduction in lesion development on cotton hypocotyls (Table 1).

The optimum concn of glucose to give this effect was 56.0 mM (10 g/liter). Concentrations below 14.0 mM were not effective in reducing disease severity. The percentage reduction in lesion area in the presence of 14, 28, and 56 mM glucose was 38.5, 64.0, and 74.0, respectively. The reduction in lesion development was accompanied by an increase in vegetative growth of the fungus.

Methyl glucose was active at lower concns than glucose. Lesion formation was completely prevented by a solution of 12.9 mM MeG and the effect was pronounced at 2.5 mM MeG.

With both glucose and MeG the effect was persistent

TABLE 1. Influence of exogenous glucose and 3-*O*-methyl glucose on *Rhizoctonia* lesion severity on cotton

Concn of chemical in solution used to moisten sand (mM)	Mean lesion area (mm^2)[a] Time after inoculation (h) 48	72[b]
Glucose (G)		
5.6	40.8 ± 10.0	66.0 ± 7.6
14.0	32.2 ± 3.5	
28.0	7.8 ± 2.3	1.6 ± 1.0
56.0	4.1 ± 1.8	0.2 ± 0.2
112.0	3.6 ± 1.4	0.1 ± 0.1
224.0	6.0 ± 3.3	
3-*O*-methyl glucose (MeG)		
0.31	66.5 ± 1.0	
0.62	83.4 ± 7.0	
0.13	51.8 ± 17.0	
2.6	7.0 ± 4.2	11.4 ± 5.0
5.2	0.2 ± 0.2	0
12.8	0	0
25.7	0	0
MeG 25.7 + G 5.6	0	0
MeG 25.7 + G 14.0	0	0
MeG 5.2 + G 5.6	0	
Water check	82.0 ± 5.0	101.1 ± 6.7

[a]Each value is based on at least three experiments with three 10-plant replications/experiment.

[b]The data on lesion development after 72 h was based on a separate set of inoculations, done at the same time, and therefore are not directly comparable to the 48-h results.

and little change was observed from 48 to 72 h after inoculation, whereas the lesions on control plants increased markedly during this period. In this regard, MeG was again more effective than glucose, completely preventing lesion development. With glucose the lesions present 72 h after inoculation were very characteristic. They consisted of extensive pathogen growth on the surface of the hypocotyl and some tissue collapse immediately beneath the fungus. There was little necrosis or tissue maceration extending beyond the area of pathogen development.

Two additional isolates were tested for their response to exogenous glucose with cotton and bean as the host plants. One (R-3) was isolated from potato in California and the other (V-7) from bean in Venezuela. The reaction of these isolates was similar to that of the primary isolate (R-21) on both plants. The average lesion area resulting from inoculating cotton with R-21, R-3, and V-7, without an exogenous carbon source, was 64.7, 26.1, and 39.7 mm^2, respectively, after 48 h and 120.9, 98.2, and 105.3 after 72 h. When the sand supporting the hypocotyls was moistened with a solution containing 56.0 mM glucose, the average lesion area 48 h after inoculation with the above isolates was 0.05, 1.8, and 0.0 mm^2. In 72 h the lesion areas were 13.7, 15.2, and 0.0 mm^2. Inoculation of bean (*Phaseolus vulgaris* L. 'Great Northern') with R-21, R-3, and V-7, without an exogenous carbon source, resulted in an average lesion area of 24.9, 26.9, and 31.8 mm^2, respectively, after 48 h, and 40.2, 60.7, and 27.3 mm^2 after 72 h. In the presence of 56.0 mM glucose the lesion areas were 0.5, 2.6, and 0.0 mm^2 48 h after inoculation, and 10.4, 13.7, and 0.25 mm^2 after 72 h.

The effect of 12.9 mM MeG on isolates R-3 and V-7 on cotton and R-21, R-3, and V-7 on bean was determined. In all cases, there was complete inhibition of lesion development.

Lesion reduction by sugars other than glucose and by methyl glucopyranosides.—Several sugars were tested for their influence on *R. solani* damage to cotton. Solutions of the sugars (56.0 mM) were used to moisten the sand supporting cotton hypocotyls. Seedlings were inoculated with mycelium grown on the basic medium plus 20 g/liter glucose and 2.0 g/liter asparagine. The value after each sugar is the average lesion area (mm^2) of 60 seedlings (two experiments, three 10-plant replications per experiment) 48 h after inoculation: glucose, 0.3; sucrose, 0.1; galactose, 1.9; arabinose, 54; maltose, 3.3; xylose, 0.2; fructose, 0.5; water control, 86.8.

The above sugars were used as sources of carbon for growth of *R. solani*. The sugars were added, after separate autoclaving, to the basic medium plus 2 g/liter potassium nitrate. Each sugar was used at a concn to give 0.66 g atoms/liter. The value following each sugar is the average mg mycelial dry weight/petri plate after 5 days, based on 10 plates: glucose, 260; sucrose, 290; galactose, 238; arabinose, 73; maltose, 278; xylose, 236; fructose, 262.

Methyl α- and methyl β-D-glucopyranoside were tested for their effect on lesion development and as sources of carbon for *R. solani*. When solutions containing 51.5 mM of these materials were used to moisten the supporting sand, there was no influence on disease severity. Neither compound was utilized as a source of carbon by the pathogen. When these compounds were present in the medium, as the sole source of carbon, at a concn of 0.7 g atoms/liter, the mean dry weight of mycelium after 5 days was 5 and 33 mg/petri plate for the methyl α- and methyl β-D-glucopyranoside, respectively.

Effect of 3-O-methyl glucose on growth of R. solani.—The marked effect of MeG on disease severity suggested that pathogen growth might be affected. To investigate this point the linear growth of *R. solani* was determined on 1.5% Difco Noble agar containing various concns of MeG. After 72 h incubation at 28 C, the colony radius on agar containing 0, 5.2, 12.8, 25.7, and 51.5 mM MeG was 40, 23, 20, 21, and 19 mm, respectively. When the agar contained 5.6 mM glucose, the 72-h colony radius in the presence of the above concns of MeG was 39, 39, 39, 39, and 33 mm, respectively. A concn of 5.2 mM MeG reduced linear growth by 43% but this inhibition was eliminated when glucose was present at 5.6 mM. The maximum ratio of MeG to glucose that allowed optimum growth was approximately 5:1.

The ability of *R. solani* to utilize MeG as a source of carbon was determined. The compound did not support growth of the pathogen (Table 2). It was also shown that when added to a complete medium, MeG did not reduce growth of *R. solani* (Table 2).

Effect of glucose and MeG on development of R. solani on cotton hypocotyls.—Observations of the effect of glucose and MeG on the development and growth of *R. solani* on cotton hypocotyls were made at 10 h intervals from 20 to 60 h after inoculation. The development was rated on a scale of 1-4 as follows: 1 = mycelium had contacted hypocotyl and exhibited linear growth; 2 =

extensive lateral branching; 3 = infection cushions had formed; and 4 = lesions were present. Growth was determined by measuring the distance that the pathogen extended along the hypocotyl.

When the sand supporting the hypocotyls was moistened with water, infection cushions formed between 20 and 30 h after inoculation and lesions were present by 40 h (Fig. 1-A). In the presence of a solution containing 56.0 mM glucose, infection cushion formation was slightly delayed but they were present 40 h after inoculation. Although cushions were formed, there was no lesion development (Fig. 1-A). With 25.8 mM MeG there was growth of the pathogen along the hypocotyl with some lateral branching. However, 60 h after inoculation there were no infection cushions. The presence of 5.6 mM glucose in the MeG solution used to moisten the sand increased slightly the development of *R. solani* on the hypocotyls. It did not, however, result in infection cushion formation (Fig. 1-A).

The effect of the various exogenous materials on mycelial growth along the hypocotyls was similar to their effect on pathogen development (Fig. 1-B). The greatest growth occured when the sand was moistened with water, reaching a distance of 23 mm, 60 h after inoculation. Linear growth was somewhat reduced by glucose but the total amount of mycelium formed was greater than with water alone.

Moistening the sand with a solution containing 25.8 mM MeG resulted in reduced mycelial growth along the hypocotyl. The addition of 5.6 mM glucose increased growth, but it remained less than with glucose alone or water (Fig. 1-B). Washing the plates to remove the sand revealed that although the MeG + glucose resulted in less growth on the hypocotyl than the water control, the total growth was greater than the control. This is consistent with the fact that in culture the addition of glucose overcame the growth inhibition by MeG. An evaluation of total growth was accomplished by determining the dry weight of mycelium and adhering sand. The sand contributed most of the weight but it was held by the mycelium and thus was an indirect indication of growth. The mean dry weight of mycelium and sand/inoculation

TABLE 2. Growth of *Rhizoctonia solani* with glucose (G) and 3-*O*-methyl glucose (MeG) as sources of carbon

Concn of chemicals (mM)	Mean dry wt/plate (mg)
KNO_3 as nitrogen source[a]	
Glucose - 112.0	316.0
Glucose - 56.0	153.0
3-*O*-methyl glucose - 25.7	0.5
3-*O*-methyl glucose - 51.5	1.2
Asparagine as nitrogen source[b]	
Glucose - 112.0	215
G - 112.0 + MeG - 12.8	239
G - 112.0 + MeG - 25.7	253
G - 112.0 + MeG - 51.5	291

[a]Mean of 10 petri plates.
[b]Mean of 20 petri plates.

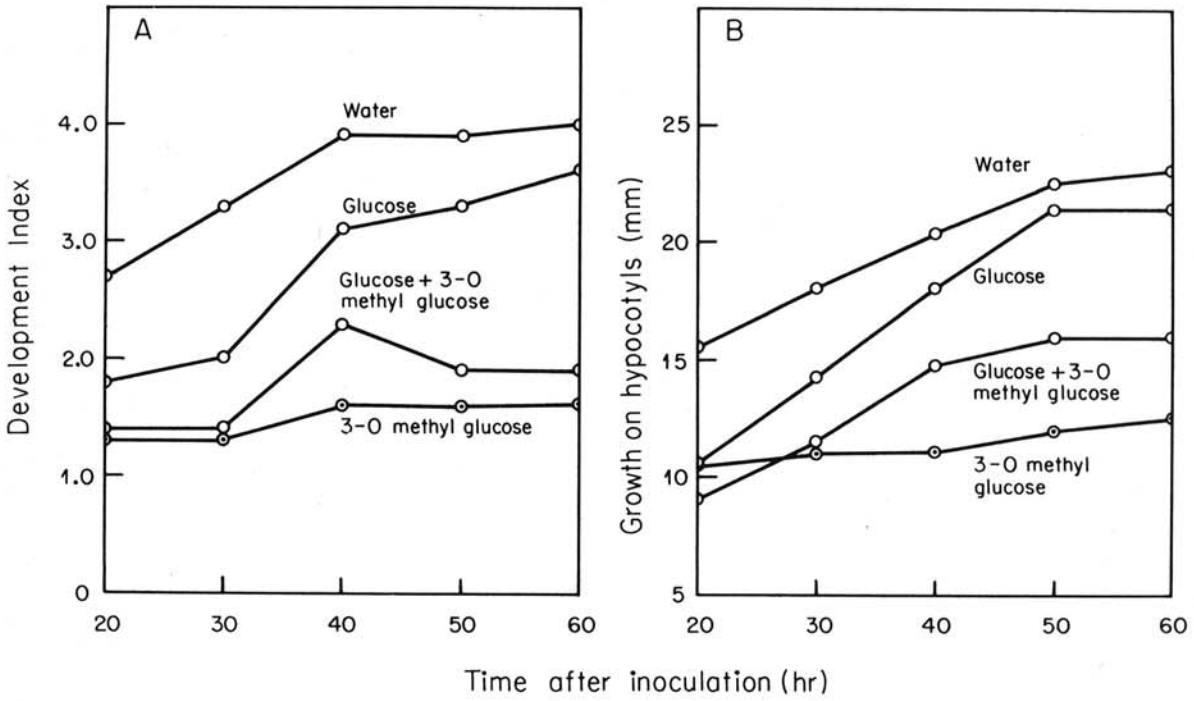

Fig. 1. Influence of glucose and 3-*O*-methyl glucose on growth and development of *Rhizoctonia solani* on cotton hypocotyls. Glucose at a concn of 56.0 mM was used to moisten the sand supporting the seedlings. Solutions of 3-*O*-methyl glucose were at a concn of 25.8 mM. The concn of glucose used in combination with 3-*O*-methyl glucose was 5.6 mM. Development index was as follows: 1 = mycelium contacted hypocotyl and exhibited linear growth; 2 = extensive lateral branching; 3 = infection cushions formed; 4 = lesions present. Each value is based on three experiments with a total of six 10-plant replications.

disk, 48 h after inoculation, was 77 and 488 mg when the sand was moistened with water or a solution containing 25.8 mM MeG + 5.6 mM glucose, respectively.

Influence of glucose and 3-O-methyl glucose on production of polygalacturonase by R. solani in culture.—The production of pectolytic enzymes by *R. solani* was studied using 2-mm diam disks of mycelium cut from 5-day-old cultures grown on the basic medium containing 20 g/liter glucose and 2.0 g/liter asparagine. These disks were comparable to those used for inoculum in the infection studies. Ten disks were placed in 5 ml of distilled water contained in 25 ml Erlenmeyer flasks and shaken at 28 C. After 12 h the water was replaced by various substrates and polygalacturonase (PG) activity was periodically assayed using viscometric techniques. The 12-h period in water corresponds to the time required for the fungus to grow from the inoculum disk and contact the host in the infection studies. Both glucose and MeG inhibited lesion development when water in the sand was replaced by either chemical 12 h after inoculation. One ml of the solution to be assayed was mixed with 6 ml of 1.0 g/liter sodium polypectate in 0.1 M acetate buffer at pH 5.0. Relative activity (RA) was expressed as the time in minutes (T) required for a 50% reduction in viscosity ($RA = 1/T \times 1{,}000$).

For enzymes to be produced, it was necessary to replace the water with a solution containing sodium polypectate (NaPP). When glucose at concns above 2.8 mM (0.5 g/liter) was mixed with the NaPP, enzyme production was repressed. Adding MeG enhanced rather than reduced enzyme production (Table 3). Enzymes could be detected at 4 h after addition of the inducer. Activity reached a maximum 6 h after induction with 1.0 g/liter NaPP, remained relatively constant for the next 6 h and then declined. When lower concns of NaPP were used, the time required for production was increased. Concentrations of NaPP below 0.25 g/liter did not induce production of PG.

The sugars that were tested for their capacity to serve as a carbon source for *R. solani* and to inhibit lesion development were evaluated as repressors of pectinase production. All of the sugars that were comparable to glucose as a carbon source for growth (sucrose, galactose, maltose, xylose and fructose) inhibited disease development and when available to the pathogen at a concn of 28 mM (5.0 g/liter) completely repressed PG production. Ribose was not used as a carbon source ($\bar{x}$ mg dry wt-petri dish = 3.0), did not inhibit disease development ($\bar{x}$ lesion area = 74), and did not repress PG (RA after 12 h on substrate = 34.2). Arabinose, however, did not conform to the pattern described above. It did not inhibit disease development, but it did repress PG production in the culture system used in this study.

The enzymes produced in culture were tested to determine whether they would macerate sections of cotton seedling hypocotyls. Sections 0.5-mm thick from 4-day-old seedlings were damaged after 5 h in preparations containing enzymes produced in response to NaPP and NaPP + MeG and maceration was complete in 7 h. The relative activity of the preparations, determined viscometrically, was 20. Solutions in which *R. solani* had been incubated with NaPP + glucose had no cotton tissue macerating activity.

We have not extensively examined the pectolytic enzyme produced by *R. solani* in the system described above, but believe it to be an endopolygalacturonase. No activity was detected when assays were conducted at pH 8.5, with or without Ca^{++}. Reducing group liberation was detected using dinitrosalicylic acid reagent and thin-layer chromatography of the reaction mixture revealed the presence of oligomers in 2 h and free galacturonic acid in 24 h.

Assay of hypocotyls for polygalacturonase activity at various times after inoculation.—Hypocotyl segments from seedlings on glass plates in the laboratory were extracted at 24, 48, and 72 h after inoculation with *R. solani* and the extracts assayed viscometrically for PG activity. Segments 1.8 cm long were cut at the site of inoculation from 15 hypocotyls and homogenized at 4 C for 1 min in 15 ml 0.5 N NaCl with a Virtis homogenizer at medium speed. The suspension was strained through cheesecloth and centrifuged at 6,000 *g* for 20 min at 4 C. The supernatant liquid was dialyzed against distilled water for 12 h at 4 C and assayed for enzyme activity. Each treatment was replicated three times.

No enzyme activity was detected in extracts from plants, up to 72 h after inoculation, when the supporting sand was moistened with either 56 mM glucose or 12.8 mM MeG. When the sand was moistened with water, PG activity was detected from plants extracted 24 h after inoculation. The mean relative PG activity from plants supported by sand moistened with water and extracted

TABLE 3. Effect of glucose and 3-*O*-methyl glucose (MeG) on in vitro production of pectinase by *Rhizoctonia solani*. Enzyme induced by 1.0 g/liter sodium polypectate (NaPP)

Time on substrate (h)	Relative activity (viscometric)			
	Composition of substrate[a]			
	water	Sodium polypectate (1.0 g/liter)	NaPP (1.0 g/liter) + glucose (5.0 g/liter)	NaPP (1.0 g/liter) + MeG (5.0 g/liter)
4	0	19 ± 9[b]	0	2 ± 2
6	0	66 ± 16	0	107 ± 25
8	0	52 ± 9	0	182 ± 70
12	0	44 ± 9	0	151 ± 20
24	0	19 ± 5	0	58 ± 13

[a]Substrate added after mycelial disks had shaken in water for 12 h.
[b]Values based on seven experiments; at least two replications/experiment; ± standard error is indicated.

24, 48, and 72 h after inoculation was 17.1, 64.6, and 78.0, respectively.

The extracts were also tested to determine whether they would macerate 0.5-mm-thick cross sections of cotton hypocotyls. Sections were placed in 1.0 ml extract, adjusted to pH 5.2 with 0.1 ml 0.01 M acetate buffer, and incubated at 28 C. There was no evidence of maceration when sections were placed in extracts from plants inoculated in the presence of glucose or MeG. Sections in extracts from plants sampled 24 h after inoculation in the presence of water showed obvious damage in 5 h. Extracts from similar plants sampled 48 and 72 h after inoculation completely macerated the sections in 5 h.

DISCUSSION.—The pathogenic mechanisms of most plant pathogens are largely unknown. The influence of pathogen nutrition on virulence of *R. solani* was investigated with the hope of developing a system whereby the manner in which this organism attacks and damages the host plant could be determined. During the course of these studies it was observed that an exogenous supply of glucose prevented the formation of hypocotyl lesions by *R. solani*. Data has been obtained that suggests this effect is due to catabolite repression of pectolytic enzyme production. A similar situation has been described for *Pyrenochaeta terrestris* (6), *Fusarium oxysporum* f. sp. *lycopersici* (8), and *Ceratocystis ulmi* (4).

The suggestion that the "glucose effect" is due to pectinase repression is supported by the fact that for six other sugars that were tested there was a direct relationship between utilization of the sugar as a carbon source by *R. solani*, inhibition of disease development and pectinase repression. The one exception encountered was with arabinose. This sugar was moderately effective as a carbon source and repressed pectinase production but it did not appreciably reduce disease development.

There are many studies emphasizing the role of pectolytic enzymes in host damage by *R. solani* (2, 3,9). The repression of virulence by exogenous glucose through inhibition of the production of these enzymes is consistent with the evidence for their involvement in disease development. The development of the pathogen on the hypocotyls, in the presence of glucose and the absence of enzyme activity in tissue extracts, was also consistent with repression of PG production. Mycelial growth was abundant; numerous infection cushions formed, but extensive tissue maceration did not occur.

The question of the relationship between PG produced in culture and during pathogenesis is the subject of a study to be published separately. We did, however, demonstrate in this work that PG from culture and infected tissue was active in macerating sections of cotton hypocotyl. There are reports that *R. solani* produces pectate lyase (1). Although we did not conduct a detailed evaluation of the enzymes produced in the culture system we used, the absence of activity at pH 8.5 and insensitivity to Ca^{++} indicated that this enzyme was not present. The spectrophotometric assay also indicated the absence of pectate lyase (L. W. Brookhouser, *unpublished*).

The effect of 3-*O*-methyl glucose was superficially similar to that of glucose. Closer examination, however, revealed that the mode of action was quite different. Although an exogenous supply of MeG reduced growth of the pathogen, this was overcome by the presence of a small quantity (5.6 mM) of glucose. In the presence of MeG alone or with MeG plus glucose the pathogen contacted the host hypocotyl, but did not continue normal development. Following host contact immediate branching leading to infection cushion formation was characteristic of a compatible host-parasite interaction. With MeG this process was inhibited and infection cushions did not form.

Growth of the pathogen along the cotton hypocotyl was reduced by MeG. Glucose (5.6 mM) eliminated any growth reduction in the sand, but did not appreciably increase the growth on the hypocotyl. A further difference between the action of glucose and MeG was that in culture glucose inhibited pectinase production whereas MeG enhanced the production of this enzyme.

An important question regarding the significance of pectolytic enzymes in pathogenesis is whether they are a determining factor in pathogenicity, or whether they are active only after a host-pathogen relationship has been established. Results of this study suggest that critical events determining the course of pathogenesis precede the formation of pectolytic enzymes. Glucose appears to interfere with disease development through the prevention of symptoms; i.e., tissue maceration. Glucose does not interfere with host-pathogen specificity expressed by infection cushion formation. The 3-*O*-methyl glucose appears to block a process involving initial host-pathogen interaction because although the pathogen grows on the host, the specific reactions leading to infection cushions do not occur.

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