Lectins: A Possible Basis for Specific Recognition in the Interaction of *Trichoderma* and *Sclerotium rolfsii*

Rina Barak, Y. Elad, D. Mirelman, and I. Chet

First and second authors, research assistants; fourth author, professor, Department of Plant Pathology and Microbiology, Faculty of Agriculture, The Hebrew University of Jerusalem, Rehovot 76100, Israel; third author, professor, Department of Biophysics, Weizmann Institute of Science, Rehovot, Israel.

This research was supported by a grant from the National Council for Research and Development, Israel, and the G.S.F. München, Germany.

We gratefully acknowledge D. Zamir and M. Pines for helpful discussions and Y. Nuchamowitz for excellent technical assistance.

Accepted for publication 24 September 1984.

ABSTRACT

Barak, R., Elad, Y., Mirelman, D., and Chet, I. 1985. Lectins: A possible basis for specific recognition in the interaction of *Trichoderma* and *Sclerotium rolfsii*. Phytopathology 75:458-462.

The plant pathogen *Sclerotium rolfsii* produced a lectin in solid and liquid media. Extracts of the fungus, as well as a culture filtrate, agglutinated certain Gram-negative bacteria and yeasts, but not human red blood cells. D-Glucose, D-mannose (20 mM), and several of their derivatives specifically inhibited the agglutination of cells of *Escherichia coli*. Agglutination activity was also blocked by 1% trypsin or 1 mM Na_2-EDTA. The effect of the latter was reversed by the addition of Mn^{++} and CA^{++}. Agglutinin activity was associated with the extracellular polysaccharide of *S. rolfsii*. The agglutinin was purified by ammonium sulfate precipitation, followed by gel filtration on a column of Sepharose 6B. SDS/polyacrylamide gel electrophoresis of the column-purified agglutinin showed two protein bands with molecular weights of 60 and 55 kdaltons. The ability of different isolates of the mycoparasite *Trichoderma* spp. to attack *S. rolfsii* was correlated with the agglutination of conidia of *Trichoderma* by *S. rolfsii*. The possible role of agglutinin in specific recognition in fungus-fungus interactions is discussed.

Lectins are sugar-binding proteins or glycoproteins of nonimmune origin, which agglutinate cells and/or precipitate glycoconjugates (15). Discovered first in plants and later in other categories of living things, lectins are involved in the interactions between the cell's surface components and its extracellular environment (3). Cells of *Saccharomyces* have been known to be agglutinated by concanavalin A (34). The same lectin also agglutinated blastospores of *Candida albicans* (5). Plant lectins specifically bind to hyphal tips and septa of *Trichoderma viride*, penicillia, and aspergilli and inhibit fungal growth as well as spore germination (2,26). Lectins have been shown to mediate in the firm attachment of predatory fungi to nematodes (27).

Fungal lectins are also involved in the relationship between fungi and algae in lichens. In slime molds, lectin activity is closely correlated with the development of cohesiveness between the cells (23,33). Furthermore, hemagglutinins have been reported in the fruiting bodies of various mushrooms (e.g., *Agaricus campestris, A. bisporus, Flamulinia velutipes*, and *Aleuria aurantia* [14,19,31,32,36]) and in extracts of seven different mushrooms (1). Hemagglutinating activity was also present in cell wall and culture filtrates of *Conidiobolus lampranges* (18). Yet, the physiological role of lectins in these fungi was not discussed. Indeed, the comprehensive review by Lis and Sharon (22) includes only a few reports on the role of lectins in fungal growth.

Bacteria are known to display great variations in the composition and structure of their cell surface carbohydrates. Certain bacteria that react with lectins can serve as a useful assay for agglutination activity (24,28,30).

The mycoparasite *Trichoderma harzianum* Rifai, a natural enemy of plant pathogenic fungi (e.g., *Rhizoctonia solani* and *Sclerotium rolfsii*) was successfully applied as a biocontrol agent against these fungi in field crops (13). *T. harzianum* coils around the hyphae of its host and then penetrates them by enzymatically digesting the cell wall (6,12).

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

We recently reported the presence of a hemagglutinin in hyphae of *R. solani* and their extract (9). The agglutinin had a high specific activity in agglutination of type O erythrocytes, and it probably binds to galactose residues on cell walls of *Trichoderma* (9). This may explain how the antagonist recognizes its host, but not the specificity of this mechanism. In this paper, we report the properties of the agglutinin of *S. rolfsii* and its possible role in the specific recognition between *Trichoderma* spp. and its host, *S. rolfsii*.

MATERIALS AND METHODS

Strains and growth conditions. *Sclerotium rolfsii* Sacc. type A ATCC 26325, *Trichoderma harzianum* and *T. hamatum* (Bon.) Bain, were maintained on a synthetic medium (SM) (29) at 28 ± 1 C. *S. rolfsii* was grown either in liquid SM in 250-ml Erlenmeyer flasks, each containing 75 ml of SM, on a rotary shaker (New Brunswick Scientific Co., New Brunswick, NJ) at 180 rpm and 28 C or on SM agar in plates. Isolates of *Trichoderma* were grown on SM agar plates for 6 days at 28 C. Bacterial cells used throught this study were: *Escherichia coli* B, *Aerobacter aerogenes, Serratia* sp., *Azotobacter chroococcum*, and *Azospirrilum brasilense* (Department of Microbiology, Faculty of Agriculture), *E. coli* 0111, *E. coli* 0128, *E. coli* 0124, *E. coli* 09K, and *E. coli* 068-61K (Department of Biophysics, Weizmann Institute of Science). They were grown in nutrient broth (Difco Laboratories, Detroit, MI) for 24 hr at 37 C. *Saccharomyces cerevisiae* was grown in liquid SM for 24 hr at 37 C.

Agglutinin source. Mycelium of *S. rolfsii* from 5-day-old cultures was collected by centrifugation at 27,000 *g* for 20 min at 4 C. The supernatant solution was dialyzed against 0.1 M phosphate-buffered saline (PBS), pH 7, for 24 hr. This material served as crude agglutinin (CA) in most experiments. A mycelial extract was also tested for agglutination activity. The mycelium was washed three times in PBS, homogenized (Heidolph, Electro, K G, D-842), Kelheim, W. Germany) for 3 min, sonicated (ultrasonic disintegrator; M.S.E. London, England) for 3 min, and centrifuged at 27,000 *g* for 30 min. The supernatant solution was dialyzed against PBS (4 × 2 L) for 24 hr and tested for activity.

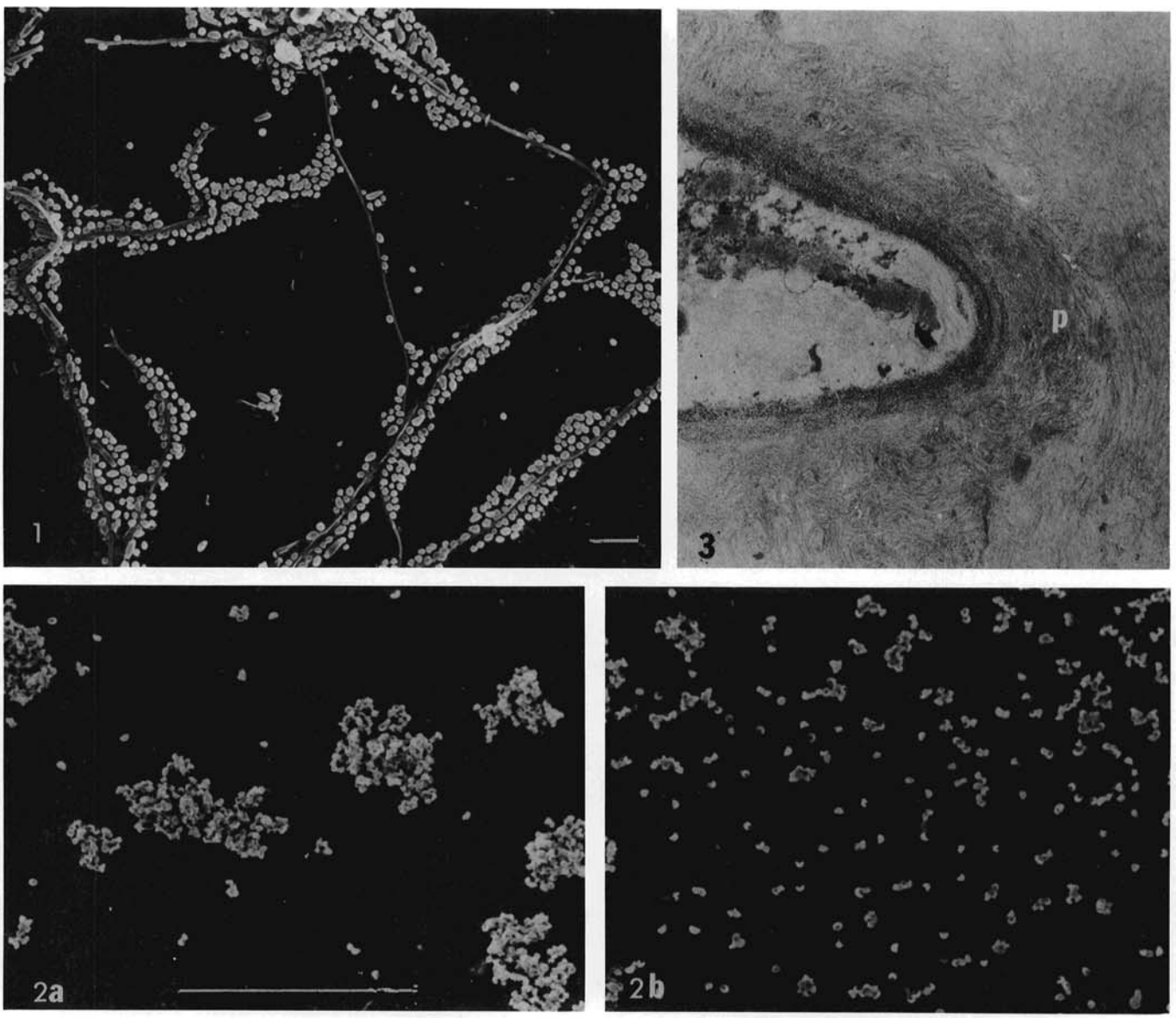
Agglutination assay. Bacterial cells were collected by centrifugation at 20,000 *g* for 15 min at 4 C, washed twice in PBS, and fixed with 0.25% glutaraldehyde (Sigma Chemical Co., St. Louis, MO) in PBS for 30 min at 4 C. Fixed cells were washed once in PBS, suspended in 0.1 M glycine in PBS for 2 hr, then washed four times in PBS and kept at 4 C. Fixed and untreated cells were washed twice and brought to a final concentration of 5×10^9 per milliliter of PBS before the assay. Preliminary agglutination tests were carried out with all bacterial strains. Five microliters of CA were mixed with 5 μl of a suspension of either untreated or fixed cells on a microscope slide. Agglutination after 10 min at room temperature was determined with a phase-contrast microscope. Quantitative agglutination assays with *E. coli* B were performed by mixing 5-μl serial twofold dilutions of Ca with 5 μl of a suspension of *E. coli* B. The titer was defined as the reciprocal of the highest agglutinin dilution that agglutinated cells. Protein concentration was determined by the folin phenol reagent according to Lowry et al (25). Specific activity was defined as the titer per milligram of protein per milliliter of CA. Inhibition of agglutination by various saccharides (100 or 20 mM) was estimated by microscopic examination of the number of aggregates caused by the agglutinin and sugar solution compared to the agglutinin in PBS.

The effect on agglutination of trypsin (BDH Chemicals, Ltd., Poole, England), Na_2-ethylenediamine tetraacetate (Na_2-EDTA) (Sigma), or cations was determined by preincubating each compound with CA for 30 min, and microscopically examining aggregation compared to CA without the chemical.

For observation of cell adherence to the hyphae of *S. rolfsii*, the fungus was grown on water agar plates for 4 days and a suspension of bacteria was poured over the culture. Attachment of cells to the mycelium was observed under light and scanning electron microscopes. All experiments were repeated at least three times.

Spore agglutination. *T. harzianum* isolates 233 and 110 and *T. hamatum* isolate 244 (6) were isolated from the soil. They all attack *R. solani*. Parasitism was tested on SM agar plates by incubating mycelial disks of *T. harzianum* and *S. rolfsii* opposite each other.

Conidia of *T. harzianum* were collected from SM agar plates by gently shaking 6-day-old cultures in PBS. The conidia were filtered through gauze cloth and washed several times during 24 hr in PBS. The filtration and the washing prevented spontaneous agglutination. Conidia were then brought to a concentration of 10^9/ml, and agglutination tests were carried out as previously described for bacterial cells. All experiments were repeated at least three times.



Figs. 1–3. 1, Adherence of cells of *Escherichia coli* to young hyphae of *Sclerotium rolfsii* (SEM micrograph ×500) (bar = 10 μm). **2, a,** Agglutination of conidia of *Trichoderma hamatum* isolate 244 by crude agglutinin of *S. rolfsii* and **b,** nonagglutinated conidia of *T. harzianum* isolate 233. (SEM micrographs ×360) (bar = 10 μm). **3,** Thin-section (TEM) of a hypha of *Sclerotium rolfsii* sheathed by multilayered polysaccharide (P) (×2,000).

Cell wall analysis. Cells of *E. coli* were isolated (7) and hydrolyzed in 1 N H_2SO_4 for 3 hr. The hydrolysate was neutralized with $CaCO_3$, and its saccharide components were separated by descending paper chromatography according to Taylor and Cameron (35).

Purification of agglutinin. Purification of agglutinin of *S. rolfsii* was obtained by adding solid ammonium sulfate to 100 ml of CA with continuous stirring for 1 hr at 4 C to give 60% saturation. The precipitate obtained after centrifugation at 27,000 *g* for 20 min was dissolved in 10 ml of PBS and dialyzed against PBS (4 × 2 L) for 48 hr. The solution was sonicated for 3 min to decrease its viscosity. One milliliter of this solution was then applied to a column (1.5 × 60 cm; Pharmacia, Uppsala, Sweden) of Sepharose 6B (Sigma) and eluted with 200 ml of PBS at a flow rate of 0.5 ml/min. The void volume, as determined with 0.1% blue dextran (Sigma) was 30 ml. Fractions (2 ml) that showed agglutinin activity were pooled, dialyzed against distilled water, lyophilized, and kept at −20 C. Polysaccharide contents after each separation step were calculated as 80% of the total dry weight according to the findings of Kritzman et al (20).

SDS-PAGE was performed in 10% polyacrylamide vertical slab gels, according to Laemmli and Favre (21). Agglutinating fractions from the Sepharose column were lyophilized, redissolved in a small volume of distilled water so that their protein contents were 1 mg/ml, and were prepared for electrophoresis under reducing conditions as described by Laemmli and Favre (21). Standard solutions of bovine serum albumin, ovalbumin, and lysozyme were used as molecular weight markers. After electrophoresis, the gels were stained for protein with Coomassie brilliant blue (Sigma) and scanned with an LKB Ultrascan densitometer (Bromma, Sweden) at 550 nm.

Electron microscopy. The SEM and TEM procedures used were according to Elad et al (10,11).

RESULTS

Crude agglutinin properties. Crude agglutinin of *S. rolfsii* was tested for agglutination activity with several bacteria and yeast strains. *E. coli* B, *E. coli* 086 61K, and *Aerobacter aerogenes* agglutinated in a few seconds, forming many large aggregates. *E. coli* 09K, *Azotobacter chroococcum, Azospirillum brasilense*, and *S. cerevisiae* gave a few small aggregates after 5–10 min. *E. coli* 0111, *E. coli* 0128, *E. coli* 0124, *Serratia* sp., and human red blood cells (group ABO) did not agglutinate. There was no difference in the reaction to CA of either fixed or untreated cells.

Mycelium extracts from liquid or solid medium reacted with the cells in the same manner as Ca. An adherence of *E. coli* B, *E. coli* 086 61K, and *Aerobacter aerogenes* to the hyphae of *S. rolfsii* was observed (Fig. 1), while *E. coli* 0111, *E. coli* 0124, and red blood cells (group ABO) did not attach to the mycelium. Other bacterial cells were not examined. *E. coli* B was the only strain we examined that reacted with the agglutinin of *R. solani* (8; R. Barak, D. Mirelman, and I. Chet, *unpublished*). To compare the properties of the two agglutinins, we used *E. coli* B in further agglutination tests in this study.

Specific activity of the agglutination of *E. coli* by *S. rolfsii* was much higher in culture filtrates than in extracts of the mycelium. These results were a function of the low protein contents of the culture filtrates rather than differences in their titers (Table 1). Culture filtrates also had lower viscosity than the mycelial extracts.

Agglutination activity in the culture filtrates depended on the age of the culture (Table 1). It was rather low during the first 3 days of incubation, reached its maximum at 5 days, and had disappeared at 7 days.

Growth medium containing 1.5% glucose showed almost the same titer, protein content, and specific activity as medium containing 0.5% glucose. However, culture filtrate viscosity caused by the extracellular polysaccharide of *S. rolfsii* was lower in the medium containing 0.5% glucose. Therefore, dialyzed culture filtrates (pH 7) from 5-day-old cultures, grown in low-glucose medium, served as crude agglutinin (CA) in further experiments.

The sugar specificity of agglutination by *E. coli* was studied by preincubating CA with different sugars for 30 min. D-Mannose, D-glucose, α-methyl D-glucoside, α-methyl D-mannoside, and maltose were the most effective agglutination inhibitors (Table 2). Sucrose, lactose, and DL-fucose were less effective, and the other sugars that were tested did not inhibit agglutination. Glucose, mannose, galactose, and fucose were detected in analyses of cell walls of *E. coli*.

Agglutinin activity was completely inhibited by preincubation of CA with 1% trypsin at 37 C or with Na_2-EDTA (1 mM) for 15 min at room temperature. Reversion of activity was obtained by applying 1 mM of either Mn^{++} or Ca^{++}, but not Cu^{++} or Fe^{++}, to the solution containing Na_2-EDTA. *S. rolfsii* CA was stable within the pH range 2.5–10.5. Activity was lost after incubation at room temperature for 24 hr, at 4 C for 7 days, at 60 C for 2 hr, or 5 min of boiling. Lyophilization did not affect CA activity.

Spore agglutination. Three isolates of *Trichoderma* were tested for ability to attack the mycelium of *S. rolfsii* cultured on SM agar plates. *T. hamatum* isolate 244 grew over the colony of *S. rolfsii* and degraded its mycelium, whereas *T. harzianum* isolate 110 reached the colony of *S. rolfsii* but did not invade it. *T. harzianum* isolate 233 stopped growing before reaching the colony of *S. rolfsii* and formed a 2-mm clear zone. Untreated conidia of the three isolates of *Trichoderma*, suspended in PBS, did not react with CA. Conidia of *T. hamatum* isolate 244 that had been washed five times and

TABLE 1. Agglutination activity by *Sclerotium rolfsii* agglutinin

Source of agglutinin	Age of culture (days)	Glucose concentration in growth medium (%)	Titer[a]	Protein (μg/ml)	Specific[b] activity
Culture filtrate	3	0.5	4	5	800
Culture filtrate	5	0.5	32	7	4,600
Culture filtrate	7	0.5	0	ND[c]	···
Extract of mycelium from liquid medium	5	0.5	128	575	220
Extract of mycelium from solid medium	5	1.5	8	850	9
Culture filtrate	5	1.5	64	15	4,200

[a] Reciprocal of the highest agglutinin dilution that agglutinated cells of *Escherichia coli* B.
[b] Titer · mg^{-1} protein · ml.
[c] Not determined. All experiments were repeated at least three times.

TABLE 2. Sugar specificity of *Sclerotium rolfsii* agglutinin[a]

Sugar	Concentration (mM)	Inhibition[b] (%)
D-glucose	20	90
D-mannose	20	90
α-methyl-D-glucoside	20	90
α-methyl-D-mannoside	20	90
Maltose	20	90
Sucrose	100	50
Lactose	100	50
Fructose	100	50
L-arabinose	100	0
D- or L-fucose	100	0
D- or L-galactose	100	0
L-fucosylamine	100	0
D-glucosamine	100	0
N-acetyl D-glucosamine	100	0
Rhamnose	100	0
Cellobiose	100	0
Sialic acid	100	0

[a] Crude agglutinin was preincubated with each sugar for 30 min and then tested for its ability to agglutinate cells of *Escherichia coli* B.
[b] Inhibition was estimated by microscopic examination of the number of aggregates formed in the agglutinin and sugar solution (100 or 20 mM) compared to agglutinin plus PBS. All experiments were repeated at least three times.

suspended in PBS for 24 hr were agglutinated by *S. rolfsii* CA, whereas very slight or no agglutination was observed with washed conidia of *T. harzianum* isolate 110 and *T. harzianum* isolate 233, respectively (Fig. 2). Moreover, suspended conidia of *T. hamatum* isolate 244, poured onto the pathogen's colonies, became attached to the hyphae immediately after application, while those of *T. harzianum* isolates 110 and 233 did not attach to the hyphae. Preincubating CA with D-glucose or D-mannose (20 mM) for 30 min completely inhibited the agglutination of conidia of *T. hamatum* isolate 244, as could be observed with light microscopy.

Agglutinin purification. Hyphae of *S. rolfsii* produce polysaccharides (Fig. 3), which are excreted to the growth medium (16). The agglutinin was also excreted to the growth medium and seemed to be firmly associated with the extracellular polysaccharide, as their separation was not achieved by certain methods as follows: Precipitation of the fungal polysaccharide with 60% ethanol followed by dialysis of the supernatant solution did not afford any soluble agglutinin activity. All the agglutinating activity was recovered in the sedimented polysaccharide fraction. Incubation of the crude agglutinin with either 10% glucose, 5% NaCl, 10 mM Na_2-EDTA or with a β-1,3-glucanase preparation obtained from *Trichoderma* (12) did not release the agglutinin. Moreover, affinity chromatography on Sephadex G-25 was not effective in separation of the agglutinin from the polysaccharide. Separation of agglutinin from the polysaccharide was, however, achieved by precipitation with ammonium sulfate followed by gel filtration on a Sepharose 6B column. Both the polysaccharide and the agglutinin precipitated in the ammonium sulfate (60%). In the gel filtration, however, over 90% of the polysaccharide (containing part of the agglutinin) was eluted immediately after the void volume (Fig. 4, peak I), whereas the main agglutinin peak emerged later and contained only traces of polysaccharide (Table 3). Absorbance at 280 μm of the effluent was almost zero. The specific activity of the agglutinin increased from 4,600 in the CA to 25,600 in the purified agglutinin (Table 3).

Gel electrophoresis. SDS-PAGE of the Sepharose-purified agglutinin (peak II) and the polysaccharide-containing agglutinin (peak I) showed the same protein profile. Two discrete bands of molecular weights 60 and 55 kdaltons were observed after the gel was stained and scanned (Fig. 5). Both peak I and II agglutinins showed sugar specificity identical to that of the CA.

DISCUSSION

The presence of agglutinin in extracts and culture filtrates of *S. rolfsii* was demonstrated with cells of *E. coli*. Mycelial extracts showed high titer in comparison to the culture filtrate. However, culture filtrate was the preferred agglutinin source throughout this study because it has comparatively low protein content and low viscosity. These properties facilitated the agglutination tests and the purification.

Agglutinin production was found to depend on the age of the culture; its maximal rate was at 5 days of incubation. The agglutinin was specifically inhibited by D-glucose and D-mannose. The cations Mn^{++} and Ca^{++} were essential for agglutination because they reversed the inhibitory effect of the chelating agent Na_2-EDTA. The prevention of agglutination by incubating CA with trypsin, a proteolytic enzyme, suggests the presence of an essential protein moiety in the agglutinin. The agglutinin of *S. rolfsii* is strongly bound to the extracellular polysaccharide in the culture filtrate. The association of proteins with the polysaccharide can be deduced from earlier findings of Kritzman et al (20), who showed the presence of amino acids in hydrolyzate of the extracellular polysaccharide of *S. rolfsii*. The accumulation of an extracellular polysaccharide in submerged culture of *S. rolfsii*, shown by Hadar et al (16), can be correlated with the pattern of agglutinin production demonstrated in this study.

Binding of proteins to fungal glucans was studied by Dickerson and Baker (8), who mentioned the great stability of fungal glucan-associated enzymes and the comparatively rigorous methods required for separation of the carbohydrate and the protein fractions. Indeed, in our work, part of the agglutinin was not separated from the polysaccharide (peak I). The two peaks of agglutination activity were apparently the same agglutinin; their SDS/PAGE pattern and sugar specificities were identical. The two bands obtained after gel electrophoresis may be subunits of the lectin.

TABLE 3. Purification of agglutinin from *Sclerotium rolfsii*

Extraction step	Volume (ml)	Protein (mg/ml)	Total polysaccharide dry weight (mg)	Titer[a]	Specific[b] activity
Crude agglutinin	10	0.007	60	32	4,600
Ammonium sulfate precipitate	1	0.042	52	256	8,000
Gel filtration:					
Peak I	20	0.006	47	128	21,300
Peak II	20	0.010	traces	256	25,600

[a] Reciprocal of the highest agglutinin dilution that agglutinated cells of *Escherichia coli*.
[b] Titer · mg^{-1} protein · ml.

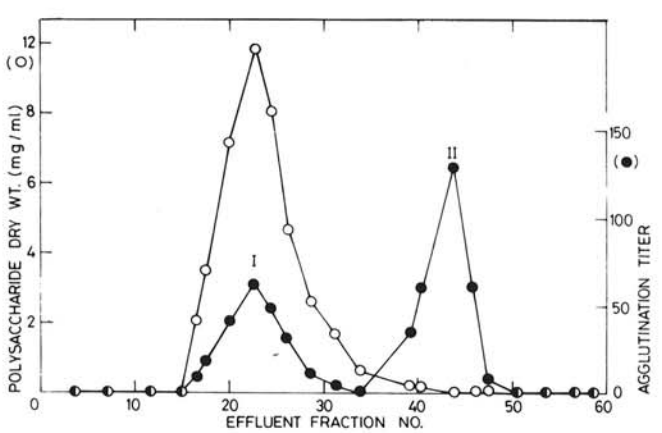


Fig. 4. Elution pattern of crude agglutinin of *Sclerotium rolfsii* on a column of Sepharose 6B. One milliliter of ammonium sulfate precipitate, dissolved in PBS, was applied to the column and eluted with PBS. Agglutination activity was eluted with (peak I) or without (peak II) the polysaccharide. o——o polysaccharide dry weight and •—• agglutination titer.

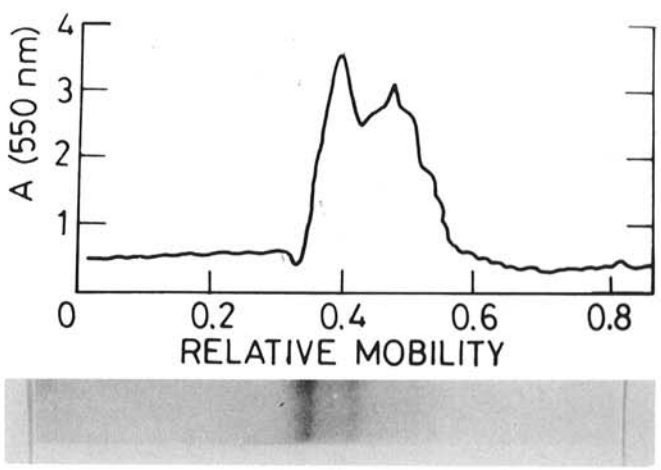


Fig. 5. SDS-PAGE profile of *S. rolfsii* agglutinin purified on a column of Sepharose 6B. The electrophoresis was performed on 10% polyacrylamide gels at pH 8.8. Sample contained 30 μg of protein. The gel was stained with Coomassie blue and scanned with a densitometer at 550 nm.

We previously reported the presence of agglutinin in extracts of *R. solani* (9). Both *S. rolfsii* and *R. solani* are soilborne plant pathogenic fungi belonging to the basidiomycetes, yet the sugar specificity, hemagglutination activity, and stability of their agglutinins are quite different.

The agglutinin of *S. rolfsii* seems to belong to a class of soluble lectins defined recently by Barondes (4). This group includes the lectins of vertebrates and slime molds and some fungal agglutinins like those of *Conidiobolus lamprauges* (18) and the yeast *Hansenula wingei* (17). Barondes (4) suggested that the common function of soluble lectins is to bind to the glyco-conjugates on and around the cells that release them, so that an extracellular environment may be formed.

This may explain the firm binding of the agglutinin of *S. rolfsii* to its extracellular polysaccharide and its role in nature. This study suggests that the agglutinin of *S. rolfsii* may also be involved in the recognition of *S. rolfsii* by the *Trichoderma* spp. Even though all of them excreted lytic enzymes (12), isolates of this antagonistic fungus that were tested in this study differed in ability to attack *S. rolfsii*. We have, therefore, tested the possibility that it is the recognition, between the host and the *Trichoderma* isolate, that is responsible for this phenomenon. Indeed, the agglutinin of *S. rolfsii* was found capable of agglutinating conidia of only *T. hamatum* isolate 244, which was shown to attack this fungal host. It appears, therefore, that agglutinin-carbohydrate binding may play a role in mycoparasitism.

LITERATURE CITED

1. Banerjee, P. C., Ghosh, A. K., and Sengupta, S. 1982. Hemagglutinating activity in extracts of mycelia from submerged mushroom cultures. Appl. Environ. Microbiol. 44:1009-1011.
2. Barkai-Golan, R., Mirelman, D., and Sharon, N. 1978. Studies on growth inhibition by lectins of penicillia and aspergilli. Arch. Microbiol. 116:119-124.
3. Barondes, S. H. 1981. Lectins: Their multiple endogenous cellular functions. Annu. Rev. Biochem. 50:207-231.
4. Barondes, S. H. 1984. Soluble lectins—A new class of extracellular proteins. Science 223:1259-1264.
5. Cassone, A., Mattia, E., and Boldring, L. 1978. Agglutination of blastospores of *Candida albicans* by concanavalin A and its relationship with distribution of mannan polymers and ultrastructure of cell wall. J. Gen. Microbiol. 105:263-277.
6. Chet, I., Harman, G. E., and Baker, R. 1981. *Trichoderma hamatum*: Its hyphal interactions with *Rhizoctonia solani* and *Pythium* spp. Microb. Ecol. 7:29-38.
7. Chet, I., Henis, Y., and Mitchell, R. 1967. Chemical composition of hyphal and sclerotial walls of *Sclerotium rolfsii* Sacc. Can. J. Microbiol. 13:137-141.
8. Dickerson, A. G., and Baker, C. F. 1979. The binding of enzymes to fungal β-glucans. J. Gen. Microbiol. 12:67-75.
9. Elad, Y., Barak, R., and Chet, I. 1983. The possible role of lectins in mycoparasitism. J. Bacteriol. 154:1431-1435.
10. Elad, Y., Barak, R., Chet, I., and Henis, Y. 1982. Ultrastructural studies of interaction between *Trichoderma* spp. and plant pathogenic fungi. Phytopathol. Z. 107:168-175.
11. Elad, Y., Chet, I., Boyle, P., and Henis, Y. 1983. The parasitism of *Trichoderma* spp. on plant pathogens—Ultrastructural studies and detection by FITC lectins. Phytopathology 73:85-88.
12. Elad, Y., Chet, I., and Henis, Y. 1982. Degradation of plant pathogenic fungi by *Trichoderma harzianum*. Can. J. Microbiol. 28:719-725.
13. Elad, Y., Hadar, Y., Hadar, E., Chet, I., and Henis, Y. 1981. Biological control of *Rhizoctonia solani* by *Trichoderma harzianum* in carnation. Plant Dis. 65:675-677.
14. Gold, E. R., and Balding, P. 1975. *Thallabiota* (Protista): Algae, lichens and fungi. Pages 117-150 in: Receptor-Specific Proteins: Plant and Animal Lectins. Excerpta Medica, Amsterdam, Netherlands.
15. Goldstein, I. J., Hughes, R. C., Monsigny, M., Osawa, T., and Sharon, N. 1980. What should be called a lectin? Nature (Lond.) 285:66.
16. Hadar, Y., Henis, Y., and Chet, I. 1981. The potential for the formation of sclerotia in submerged mycelium of *Sclerotium rolfsii*. J. Gen. Microbiol. 122:137-141.
17. Harrison, F. L., and Chesterton, J. 1980. Factors mediating cell-cell recognition and adhesion. FEBS (Fed. Eur. Biochem. Soc.) Letters 122:157-165.
18. Ishikawa, F., Oishi, K., and Aida, K. 1983. Chitin-binding hemagglutinin associated with cell wall of *Conidiobolus lamprauges*. Agric. Biol. Chem. 47:587-592.
19. Kochibe, N., and Furukawa, K. 1980. Purification and properties of a novel fucose-specific hemagglutinin of *Aleuria aurantia*. Biochemistry 19:2841-2846.
20. Kritzman, G., Chet, I., and Henis, Y. 1979. Isolation of extracellular polysaccharides from *Sclerotium rolfsii*. Can. J. Bot. 57:855-859.
21. Laemmli, V. K., and Favre, M. 1973. Maturation of the head of bacteriophage T_4. J. Mol. Biol. 80:575-599.
22. Lis, H., and Sharon, N. 1981. Lectins in higher plants. Pages 371-447 in: The Biochemistry of Plants. Vol. 6. A. Marcus, ed. Academic Press, New York.
23. Lockhart, C. M., Rowell, P., and Stewart, W. D. P. 1978. Phytohemagglutinin from TM nitrogen-fixing lichens *Peltigera canina* and *P. polydactyla*. FEMS (Fed. Eur. Microbiol. Soc.) Microbiol. Lett. 3:127-130.
24. Lotan, R., Sharon, N., and Mirelman, D. 1975. Interaction of wheat-germ agglutinin with bacterial cells and cell-wall polymers. Eur. J. Biochem. 55:257-262.
25. Lowry, O. H., Rosebrough, N. J., Farr, L. A., and Randall, R. J. 1951. Protein measurement with the Folin phenol reagent. J. Biol. Chem. 193:265-275.
26. Mirelman, D., Galun, E., Sharon, N., and Lotan, M. 1975. Inhibition of fungal growth by wheat-germ agglutinin. Nature 256:414-416.
27. Nordbring-Hertz, B., and Mattiasson, B. 1979. Action of nematode-trapping fungus shows lectin-mediated host-microorganism interaction. Nature 281:477-479.
28. Ofek, I., Mirelman, D., and Sharon, N. 1977. Adherence of *Escherichia coli* to human mucosal cells mediated by mannose receptors. Nature 265:623-625.
29. Okon, Y., Chet, I., and Henis, Y. 1973. Effect of lactose, ethanol and cycloheximide on the translation pattern of radioactive compounds and on sclerotium formation in *Sclerotium rolfsii*. J. Gen. Microbiol. 74:251-258.
30. Pistole, G. T. 1981. Interaction of bacteria and fungi with lectins and lectin-like substances. Annu. Rev. Microbiol. 35:85-112.
31. Presant, C. A., and Kornfeld, S. 1972. Characterization of the cell surface receptor for the *Agaricus bisporus* hemagglutinin. J. Biol. Chem. 247:6937-6945.
32. Sage, H. J., and Connett, S. L. 1969. Studies on a hemagglutinin from the meadow mushroom. J. Biol. Chem. 244:4713-4719.
33. Springer, W. R., Haywood, P. L., and Barondes, S. H. 1980. Endogenous cell surface lectin in *Dictyostelium*: Quantitation, elution by sugar, and elicitation by divalent immunoglobulin. J. Cell Biol. 87:682-690.
34. Summer, J. B., and Howell, S. F. 1936. The identification of the hemagglutinin of the jack bean with concanavalin A. J. Bacteriol. 32:227-237.
35. Taylor, I. E. P., and Cameron, D. S. 1973. Preparation and quantitative analysis of fungal cell walls: Strategy and tactics. Annu. Rev. Microbiol. 27:243-259.
36. Tsuda, M. 1979. Purification and characterization of a lectin from the mushroom *Flammulina velutipes*. J. Biochem. (Tokyo) 86:1463-1468.