Evaluation of Organisms Antagonistic to the Sclerotioid Organs of *Drechslera teres*, the Causal Agent of Barley Net Blotch

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ABSTRACT

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Twenty-seven microorganisms were evaluated for their ability to reduce sclerotioid organ formation and myceliogenesis in barley straw when applied prior to and following colonization by *Drechslera teres*. Ten of them were able to partly or totally inhibit the formation of sclerotioid organs on barley straw when applied as a precolonization treatment. All antagonists inhibited sclerotioid organ morphogenesis, whether through direct application or via the crude culture filtrates. Form *maculata* was more sensitive than form *teres* to culture filtrates of the antagonists. Treated barley straw did not display new sclerotioid organs in the postcolonization treatment, irrespective of the pathogen form. The postcolonization treatment of straw with certain antagonists also had a strong inhibitory effect on myceliogenesis of *D. teres* sclerotioid organs. The mycelium application also appeared to more effectively inhibit myceliogenesis than did crude culture filtrates of the antagonists. All antagonists employed reduced the aggressiveness of the mycelium from germinating sclerotioid organs previously treated with antagonists in the *maculata* form.

Net blotch, caused by *Drechslera teres* (Sacc.) Shoemaker, teleomorphic stage: *Pyrenophora teres* Drechs., is a major foliar fungal disease of barley (*Hordeum vulgare* L.). The two known forms of this pathogen, *D. teres* f. *teres* Smedeg. and *D. teres* f. *maculata* Smedeg., are present in France. The disease, which was previously latent, suddenly became epidemic about 15 yr ago (6). Studies initiated for the biological control of *D. teres* demonstrated that there was efficient antagonistic action of some microorganisms against *D. teres* in vitro and in vivo (1,5,21).

An important feature of the biology of *D. teres* is the diversity and longevity of its resting forms on straw. In this regard, crop residues can play a decisive role in the epidemiology of the disease (6,17). The sclerotioid organs of *D. teres* are subspherical fructifications which can be converted either into perithecia if fertilized by spermatia, or into sclerotia if fertilization is unsuccessful (9). Sclerotioid organs can remain viable for 2 yr in the soil (6). The inhibition of morphogenesis of sclerotioid organs by antagonists could serve as an important strategy in reducing primary inoculum levels. This report describes the effectiveness of 10 fungal organisms in reducing the formation of sclerotioid organs and myceliogenesis in *D. teres*.

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MATERIALS AND METHODS

Pathogen isolation and storage. Barley leaves were harvested from plots on the Agricultural Experimental Station of Monlon near Toulouse, France. Two polyconidial strains of *D. teres* were isolated from surface-sterilized foliar tissues exhibiting typical symptoms and transferred onto 5% V8 medium (86% tomato, 6.4% carrot, 6.6% other vegetables, and 1% salt) containing 2% agar, until a pure culture was obtained. One strain belonged to the *maculata* or spot form of *D. teres*, and the other one to the *teres* or reticulate form. The pathogen was stored as sclerotia in the dark at 10 C using the method described by Barrault (6).

Production of sclerotioid organs. Strains of the *maculata* and the *teres* forms of *D. teres* were transferred onto a Czapek agar medium low in carbohydrates (1 g of K_2HPO_4, 0.5 g of $MgSO_4$, 0.5 g of KCl, 0.01 g of $FeSO_4$,

$7H_2O$, 3 g of $NaNO_3$, and 1 g of sucrose per liter; pH 5.5), which favors formation of sclerotioid organs (30). Following incubation for 10 days in the dark at 23 C, barley straw fragments, previously autoclaved at 120 C for 20 min, were applied onto the surface of the cultures. Fructifications appeared after about 2 wk of incubation at 20 C in the dark.

Isolation of antagonists. Putatively antagonistic fungi and actinomycetes were isolated from barley straw sampled from heavily infected field plots of barley, as described above, and stored on agar slants of V8 juice medium. Putative fungal antagonists were transferred to a static liquid medium containing 10% V8 juice (pH 5.5) and incubated in the dark for 8 days at 23 C, and putative actinomycetous antagonists were transferred to a peptone yeast extract medium (5 g of peptone and 1 g of Difco yeast extract per liter; pH 5) for 13 days at 29 C. Each culture was then cool filtered on a 0.2-μm Gelman filter to separate mycelium from the secondary metabolites contained in the culture medium (crude filtrate). The mycelium was washed several times in sterile distilled water and ground with an Ultra-Turrax grinder.

Assessment of the effect of antagonists. *1. Sclerotioid organ morphogenesis.* Both mycelial suspensions or crude culture filtrates of the putative antagonists were evaluated for their antagonistic effects on sclerotioid organ morphogenesis by *D. teres.* In the precolonization treatment, autoclaved barley straw fragments were immersed into a 20 ml solution of either the culture filtrate or the mycelial suspension of the various antagonistic fungal strains for 4 hr, then applied onto the *D. teres* cultures and incubated at 23 C in the dark. In the postcolonization treatment (i.e., after the appearance and counting of *D. teres* fructifications on straw), the straw fragments were dipped into the various solutions (mycelium or filtrates), then reapplied to the surface of the cultures and reincubated. With each treatment, the controls consisted of sterile straw fragments dipped into sterile distilled water (mycelium control) or into sterile culture medium (filtrate control). Each trial included four straw fragments (3 cm × 0.5 cm) with three replications in a randomized-block design. Experimental units were examined at 2-day intervals, and the rate of existing sclerotioid organs or the occurrence of new sclerotioid organs was recorded. After incubation for 15 days, the visible (approximately 0.5 mm) sclerotioid organs forming on straw were counted using a dissecting microscope (magnification: 10 × 10) and expressed as numbers per unit surface area.

2. Sclerotioid organ myceliogenesis. After counting, sclerotioid organs were excised from the straw pieces under sterile conditions and transferred individually onto 5% V8 medium (2% agar). After incubation for 15 days at 23 C in the dark, the average diameter of each colony was measured.

3. Pathogen aggressiveness. Seeds of the susceptible barley cultivar Thibaut (4) were planted 1 cm deep in 60 × 40 × 8 cm tanks filled with vermiculite, with two seeds per furrow and six furrows per tank. Vermiculite was watered with Knop's nutrient solution every week (30). The experiments were carried out in growth chambers with 12 hr of irradiance at 90 $\mu mol \cdot m^{-2} \cdot s^{-1}$ at 23 C, and 12 hr of darkness at 18 C. The pure culture mycelium produced from treated sclerotioid organs was transferred onto a static liquid medium (10% V8) and incubated at 23 C in the dark. After 8 days, cultures were cool filtered (Durieux n°111 ash-free filter), and the mycelium (10 g/100 ml) were ground in sterile distilled water containing 0.25% gelatin and 0.01% polyoxyethylene ether (Triton CS 7, Rohm and Haas, France). Since inoculation with mycelium under controlled conditions had been shown to give the same infection types and levels on barley as inoculation with conidia (3), barley seedlings were inoculated at the two-leaf stage (27) by spraying a mycelium suspension (volume of 25 ml) over each tank. Six days after inoculation, disease intensity (measured as percentage of the foliar surface attacked) was estimated visually using the method described by Arabi (3). Infected leaves were surface sterilized, and the pathogen was isolated from necrotic foliar tissue and used for inoculation of new barley seedlings as described above. The percentage of the foliar surface attacked, obtained from the average of 10 observations per replication with three replications per treatment and per experiment, were then transformed into angular coordinates (back transformed means presented). Data were analyzed by ANOVA, and the means were separated by Newman and Keuls test (homogeneous groups) at the 0.05 level.

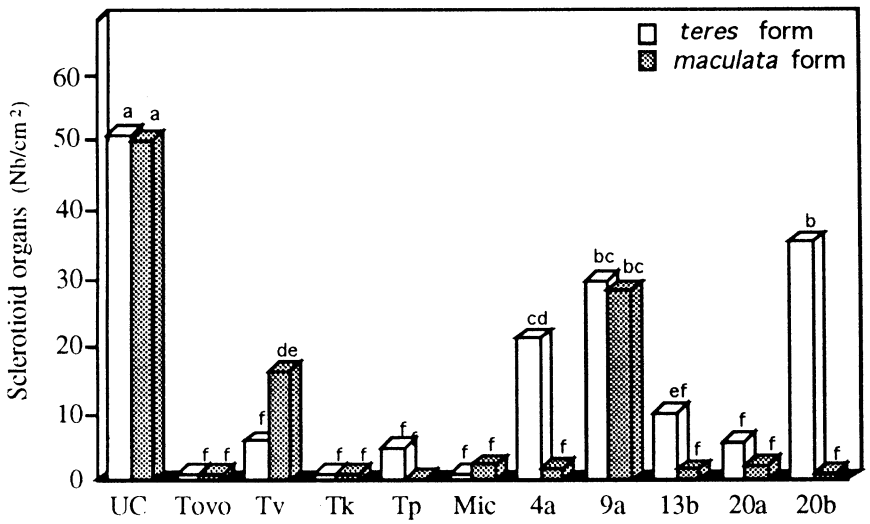

Fig. 1. Effect of a pretreatment of barley straw with antagonists on the morphogenesis of the sclerotioid organs of the *teres* and *maculata* forms of *Drechslera teres.* UC = untreated control. See Table 1 for code identifications. Bars with the same letter are not significantly different according to Newman-Keuls test ($P = 0.05$).

RESULTS

1. Morphogenesis of sclerotioid organs. Of the 27 microorganisms initially screened for antagonism to *D. teres,* 10 were able to partly or totally inhibit the formation of sclerotioid organs on barley straw when applied as a precolonization treatment (Fig. 1). These microorganisms included *Trichoderma viride* Pers.:Fr. (Tv); *T. koningii* Oudem. (Tk); *T. pseudokoningii* Rifai (Tp); one actinomycete (*Micromonospora* sp. α6 (Mic)); and five other unidentified fungi designated as 4a, 9a, 13b, 20a, and 20b. The most efficient antagonists of sclerotioid organ formation in order of decreasing efficiency were *T. koningii; T. viride* strain Tovo; the actinomycete *Micromonospora* sp. α6; *T. viride* strain Tv; *T. pseudokoningii*; and the unidentified fungi 4a, 13b, 20a, 20b, and 9a. Antagonists 4a and 20b were less effective against form *teres* than against form *maculata,* where sclerotioid organ morphogenesis was almost totally inhibited. Conversely, *T. viride* strain Tv was less effective against the *maculata* form than against the *teres* form (Fig. 1). All antagonists inhibited sclerotioid organ morphogenesis, whether through direct application or via the crude culture filtrates. The effects on morphogenesis of the sclerotioid organs varied with the antagonist. The antagonists also induced abnormalities in the morphology of sclerotioid organs. The majority of fructifications formed were less developed, displayed fewer setae, and were unable to develop myceliogenically. All mycelial treatments applied directly, inhibited completely the morphogenesis of sclerotioid organs of both *D. teres* forms;

whereas the inhibition was only partial with the filtrate of strain 4a, mostly with form *teres,* and ineffective with filtrate 9a. In contrast, the filtrate of strain 20b significantly stimulated sclerotioid ontogeny in form *teres* (Fig. 2). Form *maculata* was more sensitive than form *teres* to culture filtrates of the antagonists, with the exception of the *T. viride* filtrate, which only induced a slight inhibition of sclerotioid organ formation.

Treated barley straw did not display new sclerotioid organs in the postcolonization treatment, irrespective of the pathogen form, in contrast to nontreated straw, where a significant number of new sclerotioid organs appeared (Table 1).

2. Myceliogenesis of sclerotioid organs. Precolonization of barley straw with all the antagonists inhibited totally germination of the sclerotioid organs and therefore myceliogenesis (*data not shown*). The postcolonization treatment of straw with certain antagonists also had a strong inhibitory effect on myceliogenesis of *D. teres* sclerotioid organs (Fig. 3). Antagonists *T. viride* strains Tv and Tovo, and *T. pseudokoningii* completely inhibited germination of the sclerotioid organs. The other antagonists reduced myceliogenesis of these organs to a lesser extent. Myceliogenesis by form *maculata* was more sensitive than form *teres* to *T. koningii* and antagonists 4a and 20b, and to others to a lesser extent (Fig. 3).

The mycelium application also appeared to inhibit myceliogenesis more effectively than did crude culture filtrates of the antagonists. The inhibition of sclerotioid organ myceliogenesis was greater when the mycelium of antagonists 4a, 9a, 13b, 20a, and 20b was used against form *teres* (Fig. 3). *Micromonospora* sp. α6 was less effective in reducing myceliogenesis in both *D. teres* strains.

3. Aggressiveness of *D. teres*. Because the treated sclerotioid organs were unable to germinate, the effect of the precolonization treatment on *D. teres* aggressiveness could not be investigated. A high degree of variability occurred in the aggressiveness of both *D. teres* types of mycelium that developed from postcolonization-treated sclerotioid organs that had retained the ability to germinate on an agar medium (Fig. 4). All antagonists employed reduced the aggressiveness of the *maculata* form. In the case of the *teres* form and depending on the antagonist considered, an increase (*Micromonospora* sp. α6) or decrease (4a, 9a, 13b, 20a, and 20b) in level of aggressiveness by the pathogen was observed. Thus, only *Micromonospora* sp. α6 displayed a double effect: aggressiveness of the *teres* form was enhanced, whereas that of the *maculata* form was impaired. These effects were still reproduced after isolating the pathogen from surface-sterilized diseased tissue and inoculating new barley seedlings.

DISCUSSION

All antagonists selected for this study inhibited formation of sclerotioid organs and myceliogenesis in *D. teres,* particularly the microorganisms belonging to the genus *Trichoderma,* which were inhibitory when applied both as mycelium and as crude culture filtrates. This phenomenon was previously reported for *D. teres* in vitro (22) and was widely re-

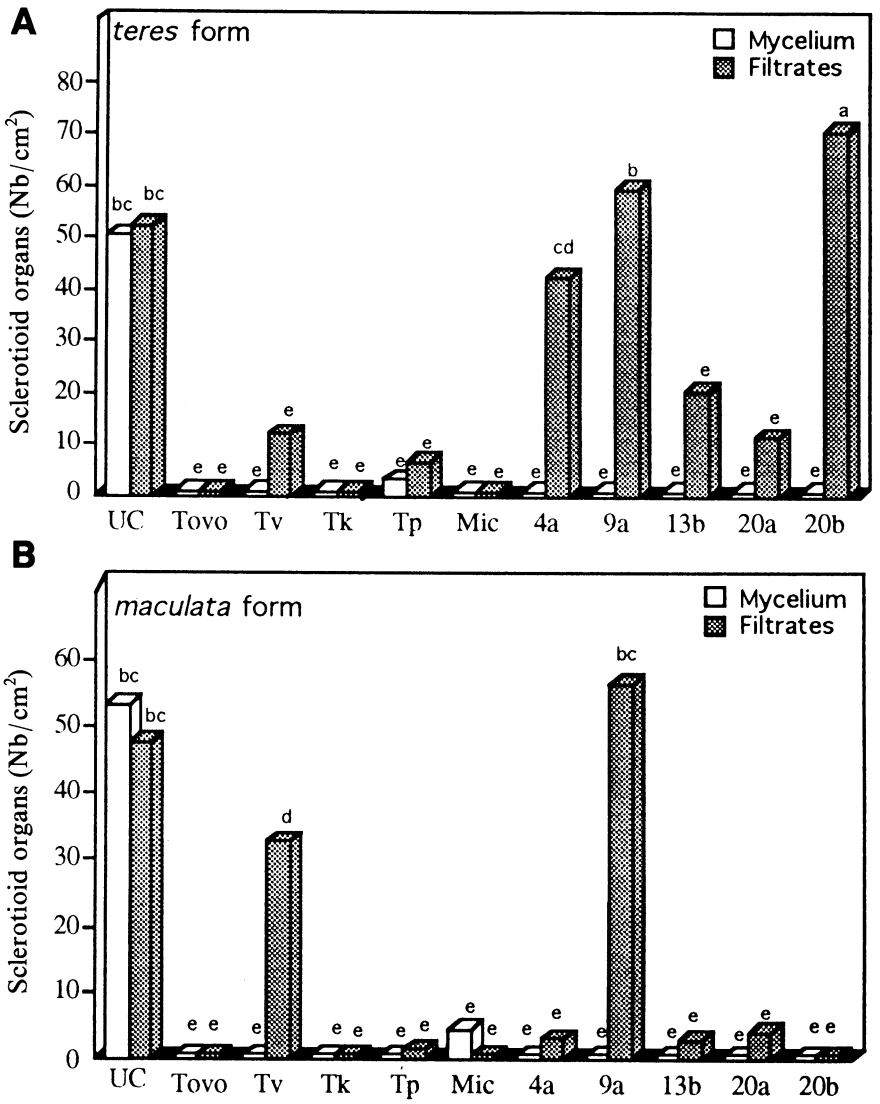

Fig. 2. Effect of a pretreatment of barley straw with mycelium or culture filtrates of antagonists on the morphogenesis of the sclerotioid organs of the **(A)** *teres* and **(B)** *maculata* forms of *Drechslera teres* on straw. UC = untreated control. See Table 1 for code identifications. Bars with the same letter are not significantly different according to Newman-Keuls test ($P = 0.05$).

Table 1. Effect of different antagonist strains on morphogenesis of the sclerotioid organs of *Drechslera teres* f. *teres* and *D. teres* f. *maculata* on barley straw previously inoculated with either form of *D. teres*

	Number of sclerotioid organs			
	Before treatment		After treatment	
Treatments	*teres* form	*maculata* form	*teres* form	*maculata* form
Untreated control	53.7 a[z]	49.3 a	84.0 b	78.0 b
Trichoderma viride (Tovo)	55.1 a	50.8 a	56.0 a	51.0 a
T. viride (Tv)	52.9 a	48.8 a	54.4 a	49.7 a
T. koningii (Tk)	54.8 a	48.9 a	55.0 a	50.3 a
T. pseudokoningii (Tp)	55.1 a	50.0 a	56.0 a	52.0 a
Micromonospora α6 (Mic)	45.6 a	49.8 a	47.0 a	51.0 a
4a	51.3 a	49.1 a	53.0 a	50.8 a
9a	47.8 a	51.2 a	49.8 a	52.0 a
13b	53.9 a	53.1 a	56.0 a	54.0 a
20a	51.3 a	49.3 a	52.0 a	51.0 a
20b	47.8 a	45.2 a	48.0 a	47.0 a

[z] Means within columns followed by the same letter are not significantly different according to Newman-Keuls test ($P = 0.05$).

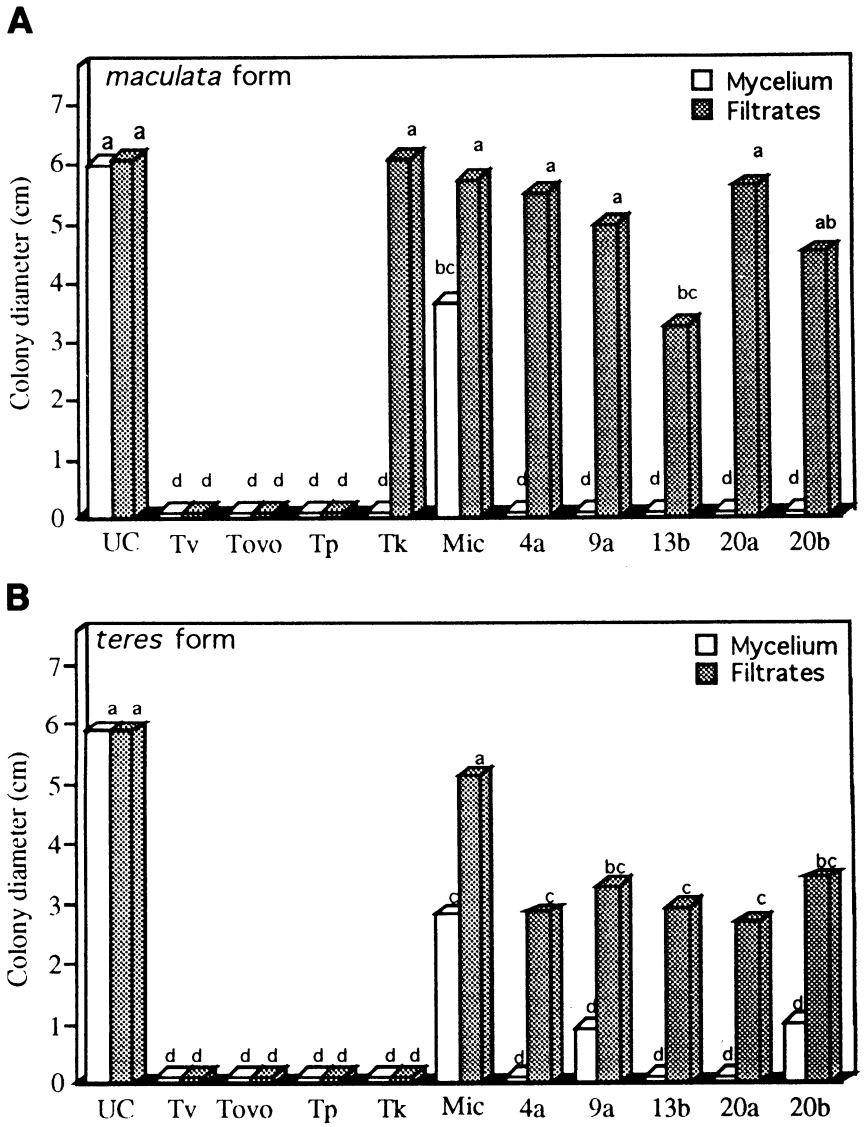

Fig. 3. Effect of a postcolonization treatment of the sclerotioid organs with mycelium or culture filtrates of antagonists on myceliogenesis of the **(A)** *maculata* and **(B)** *teres* forms of *Drechslera teres*. UC = untreated control. See Table 1 for code identifications. Bars with the same letter are not significantly different according to Newman-Keuls test ($P = 0.05$).

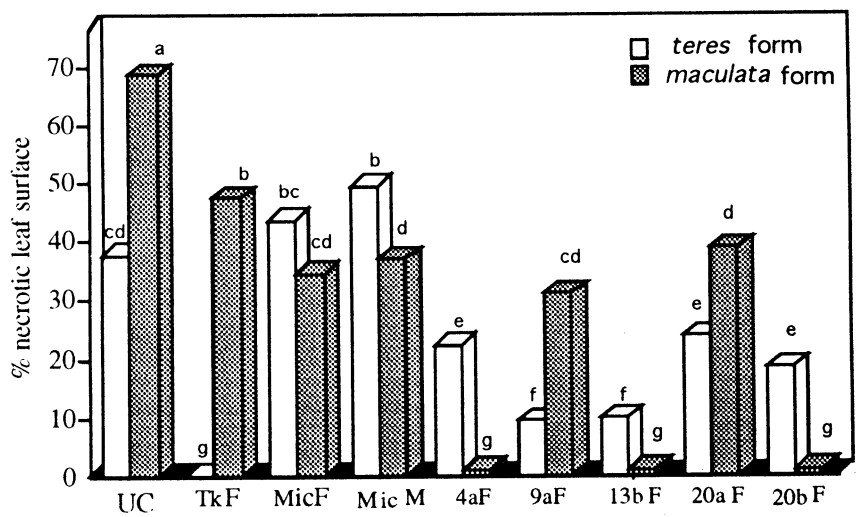

Fig. 4. Effect of a postcolonization treatment of the sclerotioid organs with mycelium (M) or culture filtrates (F) of antagonists on the aggressiveness of the *teres* and *maculata* forms of *Drechslera teres* on barley. UC = untreated control. See Table 1 for code identifications. Bars with the same letter are not significantly different according to Newman-Keuls test ($P = 0.05$).

ported for other microorganisms in vitro and in vivo (11,19,20–23,26,32). These results indicate that potential antagonists of *D. teres* can be selected from the fungal populations naturally occurring on barley straw. The antagonists proved to be efficient inhibitors of *D. teres* development when applied prior or subsequent to *D. teres* colonization of the barley straw. Pfender (24) reported the inhibition of morphogenesis of *Pyrenophora tritici-repentis* (Died.) Drechs. sclerotia by a basidiomycete isolated from crop residues. Inhibition of perithecial morphogenesis and/or of in vivo sclerotia germination in *Rhizoctonia solani* Kühn by *Gliocladium virens* J.H. Miller, J.E. Giddens, & A.A. Foster (16), or in *Sclerotium rolfsii* Sacc. by actinomycetes (26), mycorrhizae (18), or various *Trichoderma* sp. (2,7,12,15,22) has been reported. These studies were carried out in vitro or in vivo under controlled conditions, and verification of the activity of antagonists under field conditions is necessary. Further studies are necessary to understand the mechanisms of sclerotia morphogenesis inhibition under natural conditions and the interaction between antagonists and *D. teres,* as well as among antagonists. It is possible that the suppression of sclerotia morphogenesis might be the result of the application of massive amounts of antagonist inocula, which induces an imbalance in the microbial population that is favorable to secondary colonizers of crop residues under natural conditions.

The antagonistic action of microorganisms investigated may proceed through mycoparasitism (contact, enzyme, or exocellular component production) via toxins or through a "toxin-mycoparasitism" synergistic effect (31–33), which could account for the striking differences observed between the effects of mycelia of the various antagonists and those of crude filtrates, particularly for *Trichoderma,* where the release of antibiotics has been demonstrated (7,22,32). A cytobiochemical investigation of the action of antagonists on *D. teres* mycelium that developed from treated sclerotioid organs should be considered, to provide information that would increase the efficiency of antagonists and to assess the possible ecological consequences of field applications. Decreases in disease severity through the application of various antagonists have often been reported (11–14,19,26,28–30). However, there are few published reports which have recorded inhibition or stimulation of the aggressiveness of *D. teres* or of any other pathogenic mycelium that developed from the germination of resting organs treated with an antagonist. Additional studies are required, involving more strains of the two pathogen forms, to account for the large spatiotemporal variability of *D. teres* aggressiveness (3,4,6). The forms *teres* and *maculata* reacted

differently to the antagonists; this is not surprising since both pathogen forms were already known to behave differently in intensive barley cropping systems (8,30). These results would suggest that two different subspecies are actually involved, as already proposed by some authors (10,25). The use of techniques in biochemistry and molecular biology (restriction fragment length polymorphism and RAPD) might contribute to advances in chemical and/or biological control through the development of markers for early detection of the infected seeds.

It would be interesting to evaluate the antagonists identified in this study on the other resting forms of *D. teres* such as chlamydospores, resting mycelium, or conidia, and on epidemiological parameters such as disease intensity and duration of lag phase and sporulation. An efficient biological control strategy will most likely be the result of a combination of different antagonists that will vary with the target species, strains, and even the nature of the substrate.

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