Letter to the Editor

# Epidemiological Mechanisms of Mycoherbicide Effectiveness

X. B. Yang and D. O. TeBeest

First author: Department of Plant Pathology, Iowa State University, Ames 50011; second author: Department of Plant Pathology, University of Arkansas, Fayetteville 72701.

We thank R. Charudattan for valuable comments.
Accepted for publication 9 July 1993.

The use of mycoherbicides is one of two major approaches for biological control of weeds; the other is the classical approach in which plant pathogens are released to control weeds through natural spread. Since the development of Collego (TUCO Div., Upjohn Co, Kalamazoo, MI), the commercial formulation of *Colletotrichum gloeosporioides* (Penz.) Sacc. f. sp. *aeschynomene* (CGA) used to control northern jointvetch (*Aeschynomene virginica* (L.) B.S.P.) in the southern United States, more than 160 fungal pathogens have been studied as potential mycoherbicides. However, many pathogens have not been successfully developed and used despite extensive research and development. In a recent review, Charudattan (4) found a high rate of failure in this area. This letter examines the question of weed-control efficacy based on epidemiological theory.

A mycoherbicide has been defined as a fungal pathogen that is applied to control weeds in the way chemical herbicides are applied (15). This definition was derived from the successful development of Collego. Control of northern jointvetch by Collego compared to chemical herbicide treatments showed Collego to be nearly as effective as chemical herbicides, based on stand counts or population densities of the weed. Research programs have been developed with the expectation that other pathogens would be as effective as Collego and chemical herbicides. Developed under such a definition, many mycoherbicide candidates have shown promise in the laboratory or greenhouse, but most have been ineffective in the field. Additionally, for some mycoherbicide candidates, control efficacy was not consistent from year to year or from field to field. These contradictions indicate a lack of understanding of one or more important ecological factors or mechanisms contributing to the suppression of weeds by plant pathogens in the field.

A mycoherbicide is a fungal pathogen that kills plants by causing disease at a lethal, or threshold, level. Epidemiologically, a disease reaches a threshold by two means: a high number of primary infections or a high rate of subsequent secondary infections during the growing season. Using this definition, current mycoherbicide research focuses on establishing a high number of primary infections; we may not have recognized the importance of secondary infection to control efficacy, which is measured by speed and completeness of control.

**Infection window.** The importance of environmental conditions to mycoherbicide-incited infections (primary infections) has been recognized since the early stages of mycoherbicide research (15,16). The effects of dew and temperature on the number of primary infections have been reported in many mycoherbicide field tests (7,10,11,15,18). For example, McRae and Auld (12) and Walker and Boyette (19) stated that mycoherbicide candidates should be applied when environmental conditions are favorable, such as after rain or before dew occurrence. There are only a limited number of days with optimum moisture and temperature conditions for infection, and the number of optimal days varies from field to field and from year to year. Differences in primary infections among years and locations for field tests were observed in experiments with *Colletotrichum gloeosporioides* (Penz) Sacc. f. sp. *malvae* (BioMal, Philom Bios, Saskatoon, Canada), a commercial mycoherbicide in Canada; 11) and *Alternaria alternata* (Fr.:Fr.) Keissl. (7), used against round leaf mallow (*Malva pusilla* Smith) and waterhyacinth (*Eichhornia crassipes* (Mart.) Solms.), respectively. Even for Collego, which is used in moisture-rich rice fields, low levels of initial infections have been recorded in some years. When the number of initial infections is low due to unfavorable environmental conditions, effectiveness or control must result from dispersal and secondary infections of these pathogens, which have a functional relationship with increase of the pathogen population.

**Dispersal, secondary infection, and control.** Numerous mycoherbicide studies indicate the importance of secondary infection and subsequent dispersal for effective control. Boyette et al (1) reported that anthracnose caused by *Colletotrichum gloeosporioides* (Penz) Sacc. f. sp. *jussiaeae*, which has an incubation period of 3–5 days, required 28 days to progress from 29% (primary infections) to 94% in winged waterprimrose (*Ludwigia decurrens* (Walter) DC.) in rice. Dispersal also was evident, because 25% of the plants in untreated plots 100 m away were infected (1). Recently, Hasan et al (8) reported that *Stagonospora* sp. required 3 wk after spraying to develop severe disease on *Calystegia sepium* (L.) R. Br. Elwakil et al (7) reported that *A. alternata*, a potential mycoherbicide for waterhyacinth in an aquatic environment, required 2 mo to achieve lethal levels, although the incubation period was only about 12 days. They also observed significant dissemination of inoculum to control plots. In Brazil, a *Helminthosporium* sp. required 36 days post-application to defoliate 73% of inoculated wild poinsettia (*Euphorbia heterophylla* L.; milkweed) plants in soybean fields. Spread of the fungus from inoculated plants to the entire field was evident because similar disease levels were reported in both control and treated plots (22). Field studies by Charudattan et al (5) and Morris (13) have clearly demonstrated that the diseases caused by fungal pathogens in their studies progressed from about 5% at application to 90% mortality 5 wk or 2 mo later, respectively. Morris (13) observed that rain-splashing contributed to the conidial dispersal for the secondary infection.

The importance of secondary infection on control efficacy also is evident for commercial mycoherbicides. Experiments with BioMal, showed that at high inoculum concentrations, the control increased from 30–50% at 22 days after application to about 90% at crop harvest. Dispersal of inoculum was evident from the severe disease levels in control plots (14). Collego required up to 5 wk to kill weeds (6). It is known that application of inoculum results in lesions within 3–5 days (6,16), and dispersal is evident in severe infection of plants in untreated plots (6). Our recent studies have demonstrated similar results (20,21). The fungus sporulates abundantly on diseased tissue and has several dispersal mechanisms (20,21).

Post-application disease development also is determined by the environment. It has been shown repeatedly that the rate of disease increase is a function of the environment. In the case of Collego, a flooded rice field provides consistently uniform, high-moisture conditions over wide areas (17), conditions that are nearly ideal for post-application disease development. In many pathosystems, however, the environmental conditions can vary significantly from year to year and from field to field. The influence of weather on secondary infections has been indicated by Yorinori and

© 1993 The American Phytopathological Society

TABLE 1. Summary of post-application development of mycoherbicides or mycoherbicide candidates

| Mycoherbicide or candidate | Weed | Days to control[a] | Latent period (days) | Maximum cycles[b] | Source |
|---|---|---|---|---|---|
| *Alternaria alternata* | Waterhyacinth | >60 | 12 | 4–5 | Elwakil et al (7) |
| BioMal[c] | Round leaf mallow | >50 | 14 | 4–5 | Makowski and Mortensen (11,14) |
| *Cerospora rodmanii* | Waterhyacinth | 30–35 | 7 | 4–5 | Charudattan et al (5) |
| Collego[c] | Northern jointvetch | 28–30 | 3–5 | 5–6 | TeBeest et al (15,16) |
| *Colletotrichum gloeosporioides* f. sp. *jussiaea* | Winged waterprimrose | 22–28 | 3–5 | 4–5 | Boyette et al (1) |
| *C. gloeosporioides* | St. Johnswort | 50 | 2–3 | 6–7 | Hildebrand and Jensen (9) |
| *C. gloeosporioides* | Hakea | 60 | ...[d] | ... | Morris (13) |
| *C. malvarum* | Prickly sida | 14–21 | ... | ... | Kirkpatrick et al (10) |
| *Helminthosporium* sp. | Wild poinsettia | 36–40 | <8 | 4–5 | Yorinori and Gazziero (22) |

[a] Number of days of disease development from application to lethal level.

[b] Presumptive maximum number of disease cycles post-application estimated by dividing days to control with latent periods.

[c] Registered mycoherbicide.

[d] Not available.

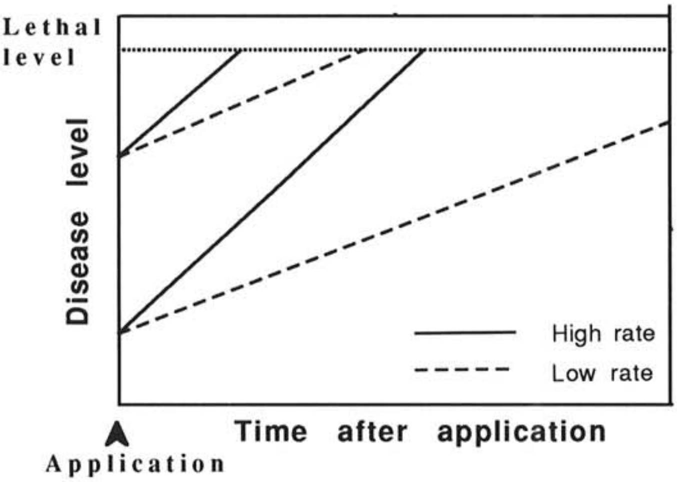

**Fig. 1.** Epidemiological mechanisms contributing to the effectiveness of mycoherbicides. Solid and broken lines represent different pathosystems. For pathogens with high rates of development, the disease reaches the lethal level from a low initial level. For pathogens with low epidemic rates, the disease fails to reach the lethal level from a low initial level. When initial infection level is high, disease with either a high (solid line) or low (broken line) rate of development can reach the lethal level.

Gazziero (22), Morris (13), and Mortensen and Makowski (14). Table 1 summarizes the studies in which epidemic progressions were recorded. A lag period exists between application and attainment of lethal levels in the epidemics incited by these pathogens. These pathogens have relatively short incubation periods, and several disease cycles can occur after the application.

**Control model.** Based on the above discussion, a model for control efficacy of mycoherbicides (Fig. 1) can be derived by modifying a scheme used by Zadoks and Schein (23). Control efficacy includes two components. The first is the primary infection established by the application of inoculum. The other is the secondary or post-application infection. Depending on weather conditions at application, the number of primary infections can vary from near-lethal levels to much lower levels. When the number of primary infections is high, the probability that the disease will develop to lethal levels in a given period also is high for a disease with either a high or a low rate of increase (Fig. 1). However, for cases in which the level of primary infection is low, only diseases with high rates of development may reach the lethal levels. Therefore, whenever primary infections are low, the rate of disease development will be critical for a disease to develop to lethal levels in a short period.

Current research does not directly address the importance of subsequent disease development because, conceptually, we are treating mycoherbicides as chemical herbicides. Figure 1 and available studies (8–14,19,22) suggest that control efficacy of mycoherbicide does not like that of chemical herbicide, which is "environmentally-independent." Charudattan (2) examined mycoherbicide control efficacy from the view of epidemiology and pointed out that control efficacy is affected by environment. He used the term "inundative control" for the mycoherbicide approach, with a definition including post-application disease development and environmental effects as components.

Available data on mycoherbicides show that we cannot rely only on the first component of control efficacy to provide consistently high levels of control because of narrow infection windows required for primary infection. We might be better served to select pathogens that also have a high potential for post-application development. This potential is a function of several factors: infection process, incubation period, sporulation, and dispersal. These factors also are affected by environment. When more information about these factors is available, better approaches toward reducing the environmental dependency of mycoherbicides may be developed.

**Common interests.** The lack of field effectiveness of potential agents has been a common problem in different areas of biological control over the past 20 yr. Biological control regulates pest populations by manipulating biocontrol agents, and control efficacy also depends on environment. To identify factors limiting control efficacy, a clear understanding of the ecological structure and the dynamics of plant pathosystems is needed. Studies that identify factors limiting the build up of biocontrol agents should share the same principles as those that determine the critical factors of epidemics for disease forecasting. In some ways, it is more difficult to enhance or manipulate the establishment of a fungal population than to control a pathogen population. Epidemiological studies on biocontrol agents could help us understand the mechanisms contributing to effectiveness (or ineffectiveness) of biocontrol. Epidemiological data can be used to devise systems to assess the potential effectiveness for the selection of these biocontrol agents, as has been done for mycoherbicides (3), and to determine when and where to apply biocontrol agents by forecasting the optimum infection days and assessing the potential regions suitable for a biocontrol agent.

In conclusion, two major epidemiological components contribute to control efficacy of mycoherbicides: 1) a window of temperature and moisture affecting the number of initial infections and 2) the subsequent dispersal and infection of the pathogen within the target weed population. Currently, most research with mycoherbicides does not address the importance of secondary infection. This may be because, conceptually, we treat mycoherbicides as chemical herbicides. The environmental dependency of biocontrol agents limits control efficacy in variable environments.

## LITERATURE CITED

1. Boyette, C. D., Templeton, G. E., and Smith, R. J. 1979. Control of winged waterprimrose (*Jussiaea decurrens*) and northern jointvetch

(*Aeschynomene virginica*) with fungal pathogens. Weed Sci. 27:479-501.

2. Charudattan, R. 1988. Inundative control of weeds with indigenous fungal pathogens. Pages 86-110 in: Fungi in Biological Control Systems. M. N. Burge, ed. Manchester University Press, Manchester, England.
3. Charudattan, R. 1990. Assessment of efficacy of mycoherbicide candidates. Pages 455-464 in: Proc. 7th Int. Symp. Bio. Control Weeds. E. S. Delfosse, ed. Rome, Italy.
4. Charudattan, R. 1991. The mycoherbicide approach with plant pathogens. Pages 24-57 in: Microbial Control of Weeds. D. O. TeBeest, ed. Chapman and Hall, New York. 283 pp.
5. Charudattan, R., Linda, S. B., Kluepfel, M., and Osman, Y. A. 1985. Biocontrol efficacy of *Cercospora rodmanii* on waterhyacinth. Phytopathology 75:1263-1269.
6. Daniel, J. T., Templeton, G. E., Smith, R. J., Jr., and Fox, W. T. 1973. Biological control of northern jointvetch in rice with an endemic fungal disease. Weed Sci. 21:303-307.
7. Elwakil, M. A., Sadik, E. A., Fayzalla, E. A., and Shabana, Y. M. 1990. Biological control of waterhyacinth with fungal plant pathogens in Egypt. Pages 483-498 in: Proc. 7th Int. Symp. Bio. Control Weeds. E. S. Delfosse, ed. Rome, Italy.
8. Hasan, S., Beglinger, C., Buehler, M., Daclinat, Sedlar, L., and Defago, G. 1992. Evaluation of *Stagonospora* sp. as a mycoherbicide for control of *Calystegia sepium*. Page 46 in: Proc. Int. Workshop Realizing Potential Bioherbicides. R. W. Medd, ed. Orange, NSW, Australia.
9. Hildebrand, P. D., and Jensen, K. I. N. 1991. Potential for the biological control of St. John's-wort (*Hypericum perforatum*) with an endemic strain of *Colletotrichum gloeosporioides*. Can. J. Plant Pathol. 13:60-70.
10. Kirkpatrick, T. L., Templeton, G. E., TeBeest, D. O., and Smith, R. J. Jr. 1982. Potential of *Colletotrichum malvarum* for biological control for prickly sida. Plant Dis. 66:323-325.
11. Makowski, R. M. D., and Mortensen, K. 1990. *Colletotrichum gloeosporioides* f. sp. *malvae* as a bioherbicide for round-leaved mallow (*Malva pusilla*): Conditions for successful control in the field. Page 513-522 in: Proc. 7th Int. Symp. Bio. Control Weeds. E. S. Delfosse, ed. Rome, Italy.
12. McRae, C. F., and Auld, B. A. 1988. The influence of environmental factors on anthracnose of *Xanthium spinosum*. Phytopathology 78:1182-1186.
13. Morris, M. J. 1989. A method for controlling *Hakea sericea* Shrad. seedlings using the fungus *Colletotrichum gloeosporioides* (Penz.) Sacc. Weed Res. 29:449-454.
14. Mortensen, K., and Makowski, R. M. D. 1990. Field efficacy at different concentrations of *Colletotrichum gloeosporioides* f. sp. *malvae* as a bioherbicide for round-leaved mallow (*Malva pusilla*). Pages 523-530 in: Proc. 7th Int. Symp. Bio. Control Weeds. E. S. Delfosse, ed. Rome, Italy.
15. TeBeest, D. O., and Templeton, G. E., 1985. Mycoherbicides: Progress in biological control of weeds. Plant Dis. 69:6-10.
16. TeBeest, D. O., Templeton, G. E., and Smith, R. J., Jr. 1978. Temperature and moisture requirements for development of anthracnose on northern jointvetch. Phytopathology 68:389-393.
17. Templeton, G. E., TeBeest, D. O., and Smith, R. J., Jr. 1979. Biological weed control with mycoherbicides. Annu. Rev. Phytopathol. 17:301-310.
18. Walker, H. L. 1981. Factors affecting biological control of spurred anoda (*Anoda cristata*) with *Alternaria macrospora*. Weed Sci. 29:505-507.
19. Walker, H. L., and Boyette, C. D. 1986. Influence of sequential dew periods on biocontrol of sicklepod (*Cassia obtusifolia*) by *Alternaria cassiae*. Plant Dis. 70:962-963.
20. Yang, X. B., and TeBeest, D. O. 1992. Analysis of dispersal mechanisms and spatial distribution of *Colletotrichum gloeosporioides* f. sp. *aeschynomene* in rice. (Abstr.) Phytopathology 82:1079.
21. Yang, X. B., and TeBeest, D. O. 1992. Rain dispersal of *Colletotrichum gloeosporioides* under simulated rice field conditions. Phytopathology 82:1219-1222.
22. Yorinori, J. T., and Gazziero, D. L. P. 1990. Control of milkweed (*Euphorbia heterophylla*) with *Helminthosporium* sp. Pages 571-578 in: Proc. 7th Int. Symp. Bio. Control Weeds. E. S. Delfosse, ed. Rome, Italy.
23. Zadoks, C. J., and Schein, R. D. 1979. Epidemiology and Plant Disease Management. Oxford University Press, New York. 427 pp.