Plant Pathology Bulletin 7:155-166, 1998

General mechanisms of action of microbial biocontrol agents

Chaur-Tsuen Lo[1,2]

1. Taiwan Agricultural Research Institute, 189 Chung-cheng Rd. Wufeng, Taichung, Taiwan.
2. Corresponding author: E-mail:ctlo@wufeng.tari.gov.tw ; Fax:886-4-3338162.
Accepted for publication: November 1, 1998

ABSTRACT

Lo, C.-T. 1998. General mechanisms of action of microbial biocontrol agents. Plant Pathol. Bull. 7:155-166.

Biological disease control is an attractive alternative strategy for the control of plant diseases. Meanwhile, it also provides practices compatible with the goal of a sustainable agricultural system. Understanding the mechanisms of biological control of plant diseases through the interactions between antagonists and pathogens may allow us to select and construct the more effective biocontrol agents and to manipulate the soil environment to create a conducive condition for successful biocontrol. In the past decade, the innovative applications of molecular techniques have broadened our insight into the basis of biological control of plant diseases. Particularly, molecular approaches are useful in determining the relative contributions of different genetic traits in complicated processes. Insofar as we know, the mechanisms of biocontrol may involve and be divided into (i) antibiosis, (ii) competition, (iii) mycoparasitism, (iv) cell wall degrading enzymes, and (v) induced resistance. However, these mechanisms of biological control are probably never mutually exclusive. They may include one and more processes.

Key words: biocontrol, antibiosis, competition, mycoparasitism, cell wall degrading enzymes, induced resistance

INTRODUCTION

Biocontrol of plant diseases involves the use of an organism or organisms to inhibit the pathogen and reduce disease (11). There are many definitions for biological control, however, the basic idea involves a strategy for reducing disease incidence or severity by direct or indirect manipulation of microorganisms (5, 62). Consequently, understanding the mechanisms of biological control of plant diseases through the interactions between biocontrol agent and pathogen may allow us to manipulate the soil environment to create conditions conducive for successful biocontrol or to improve biocontrol strategies (18, 26). Recently several methodologies for genetic analysis, such as the approach of mutant analysis, have provided promise for the study of mechanisms of biocontrol agents and their targets (96). Handelsman and Parke (1989) have suggested the application of Koch's postulates to demonstrate a cause-effect relationship in the involvement of a particular mechanism in biocontrol because it may not be adequate to demonstrate that a mechanism exists in vitro (26). The following steps suggested by Handelsman and Parke should be demonstrated in either biocontrol agents or their targets to ascertain the role of a particular mechanism. These steps are as follows: (i) the activity must be associated with a strain that is effective as a bioprotectant, or a metabolite must be identified in situ, such as in the disease situation, (ii) the gene(s) coding for the particular product or process must be cloned, (iii) the activity of the mutant should be less effective than the wild-type parent if the particular gene(s) is deleted, (iv) replacing the gene(s) encoding for the activity should restore the biocontrol activity, (v) mutants of the pathogen that are not affected by the activity of the metabolite or process should be able to incite disease in the presence of the biocontrol agent, and (vi) restoring sensitivity of the pathogen to the activity should reduce its ability to cause disease (26). In addition, other steps such as transformation of the gene and expression in heterologous organisms or induced over-expression in the same bioprotectant also may be adequate to demonstrate the particular mechanism.

Possible inhibition-processes of biocontrol agents to plant pathogens

The mechanisms of biocontrol have been discussed and reviewed in several papers and books (1, 11, 18, 19, 26, 41, 44) and mainly include antibiosis, competition, mycoparasitism, cell wall degrading enzymes, and induced resistance (52, 55). These mechanisms are probably never mutually exclusive; these terms are meant to organize the examples into general groups to facilitate comparisons. This article is not an attempt to cover all the work on biocontrol agents as I will cite only a few major studies that have apparent evidence in biocontrol. The following paragraphs are separately to describe the mechanisms of biological control of plant diseases. The data are summarized in Table 1.

Antibiosis:

Antibiosis may be involved and play an important role in plant disease suppression by certain bacteria and fungi. The process has been defined as the interactions that involve a low-molecular weight compound or an antibiotic produced by a microorganism that has a direct effect on another microorganism (26, 93, 94). The role of antibiotics in biocontrol has been studied by genetic analysis, e.g., mutants that do not produce antibiotics to demonstrate a correlation between antibiotic productivity and biocontrol activity. A few systems have been throughly examined. For example, a phenazine antibiotic (Phz) produced by *Pseudomonas fluorescens* strain 2-79 has been implicated in control of take-all of wheat caused by *Gaeumannomyces graminis* var. *tritici* (26, 85, 93, 94). Phenazine could be isolated from roots of wheat by strain 2-79 inoculation; results indicated that roots with the antibiotic were healthier than roots without the antibiotic (44, 94). Single-site Tn5 insertions were used to make mutants defective in the production of the phenazine antibiotic. All of these mutants were noninhibitory to *G. graminis* var. *tritici* in vitro and were less suppressive to take-all in greenhouse tests. Biocontrol ability was restored in the mutant strains when antibiotic production was restored by wild-type DNA introduced on a cosmid (85). Although antibiotic production plays major role in suppression of the take-all pathogen, it is not the only factor; some suppression of the pathogen is retained by the nonproducing mutants (67). Strain 2-79 also produces anthranilic acid, a substance that may play a minor role in biocontrol (72). Similar results were obtained with the *P. fluorescens* strain I11 (77). However, the mutants that demonstrated increased antibiosis (overexpression) were equal to the wild-type strain in disease suppression (77).

P. fluorescens strain CHA0 produces a number of antibiotic substances including 2,4-diacetylphloroglucinol

Table 1. Mechanisms of specific biocontrol agents for controlling plant pathogens [1]

Biocontrol agents	Strain	Possible Process/metabolite	Target pathogen	Crop	Evidence for involvement
		Antibiosis			
Agrobacterium radiobacter	K84	Agrocin 84	*Agrobacterium tumefaciens*	Stone fruits and roses	Genetic analysis in vivo
Bacillus subtilus		Iturin group	Most fungi	Various crops	Genetic analysis in vivo and vitro
Erwinia herbicola	Eh1087	β -lactam	*Erwinia amylovora*	Rosaceous plants	Genetic analysis In vitro
Pseudomonas fluorescens	CHA0	2,4-diacetyl-phloroglucinol (Phl)	*Thielaviopsis basicola,* others	Wheat tobacco	Genetic analysis in vivo
Pseudomonas fluorescens	CHA0	Pyoluteoin And Phl	*Fusarium oxysporum f* sp. *cucumerinum, Phomopsis sclertioides*	Cucumber	Genetic analysis in vivo
Pseudomonas fluorescens	CHA0	HCN	*Thielaviopsis basicola, others*	Wheat tobacco	Genetic analysis in vivo
Pseudomonas fluorescens	Hv37aR2	Oomycin A	*Pythium ultimum*	Cotton	Genetic analysis in vivo

1. Modified primarily from a report of Harman and Hayes to the Office of Technology Assessment of the US Congress 1994, and up-dated by C-T., Lo

Table 1. (Continued)

Biocontrol agents	Strain	Possible Process/metabolite	Target pathogen	Crop	Evidence for involvement
Pseudomonas fluorescens	2-79	Phenazine-1-carboxylic acid and Phl	*Gaeumannomyces graminis var. tritici*	Wheat	Genetic analysis in vivo
Pseudomonas fluorescens 119	2-79	Anthranilic acid	*G. graminis var. tritici*	Wheat	Genetic analysis in vivo
Pseudomonas fluorescens	Pf-5	Pyoluteorin and Phl	*P. ultimum; R. solani, Erwinia carotovora subsp. atroceptica*	Cotton cucumber	Genetic analysis in vivo
Streptomyce hygroscopicus var. geldanus	3602	Geldanamycin	*R. solani*	Pea	Demonstrated in field
Chaetomium globosum	Cg-13	Chaetomin	*P. ultimum*	Sugarbeet	In vitro demonstration
Trichoderma (*Gliocladium*) *virens*		Gliovirin	*P. ultimum*	Cotton	Genetic analysis in vivo
Trichoderma virens	G-20	Gliotoxin	*P. ultimum*	Cotton	Genetic analysis in vivo
Trichoderma harzianum, T. koningii		Akyl pyrones	Various fungi	Various crops	Antibiotics isolated in vitro
Trichoderma harzianum	ATCC-36042	Peptaibol antibiotics	*Botrytis cinerea*, other fungi	Grapvine	Antibiotics isolated
		Competition			
Pseudomonas fluorescens	3551	Siderophore	*P. ultimum*	Potato	Genetic analysis in vivo
Pseudomonas putida	WCS 358	Siderophore	*Fusarium oxysporum* f. sp. *raphani*	Radish	Genetic analysis in vivo
Pseudomonas putida	N1R	Volatile substances	*P. ultimum*	Pea, soybean	In vitro demonstration
Enterobacter cloacae		Inactivation of stimulants of pathogen germination	*P. ultimum*	Cotton cucumber	Genetic analysis in vivo
Trichoderm harzianum		Nutrients and space	Various fungi	Grapevine	Inferred from in vivo activity
Phlebia (*Peniophora*) *gigantea*		Infection sites	*Heterobasidion* (*Fomes*) *annosum*	Pine, conifers	In field demonstration

(Phl), hydrogen cyanide, and pyloluteorin (38, 45, 65, 66) that are involved in suppression of take-all disease of wheat caused by *G. graminis* var. *tritici*, black rot of tobacco caused by *Thielaviopsis basicola*, and other root diseases (38, 44). In addition to mutation analysis as described above, the gene(s) were also transferred into *P. fluorescens* strain 2-29 that was not normally a Phl producer. The resulting Phl-producing transconjugants were shown to have more broad antibacterial and antifungal activity than the parental strain (38).

Among other bacteria, antibiotic agrocin 84 produced by *Agrobacterium radiobacter* strain K84 is one of best described examples of biocontrol to control crown gall caused by virulent *A. tumefaciens* strains (40). Several studies have implicated agrocin K84 in the disease control process (26,

Table 1. (Continued)

Biocontrol agents	Strain	Possible Process/metabolite	Target pathogen	Crop	Evidence for involvement
		Cell-wall degrading Enzymes			
Serratia marcescens		Chitinolytic enzyme	Various fungi	Soybean	Genetic analysis heteroexpression
Trichoderma harzianum	ATCC-36042	Chitinolytic enzymes, Glucanases	Various fungi	Pea, soybean	In vitro demonstration
		Mycoparasitism			
Coniothyrium minitans		Mycoparasitism	*Sclerotinia* spp.	Sunflower	In field demonstration
Pythium nunn		Mycoparasitism	*Pythium* spp.	Various crops	In vitro demonstration
Sporidesmium sclerotivorum		Mycoparasitism	Sclerotinia minor	Lettuce	In field demonstration
Trichoderma various spp.		Mycoparasitism	Various and numerous fungi	Various crops	In vitro demonstration
		Induced resistance			
Pseudomonas fluorescens	WCS 374	Induced resistance	Various pathogens	Radish	Genetic analysis
Pseudomonas fluorescens	CHA0	Induced resistance	Tobacco necrosis virus	Tobacco	In vitro demonstration
Pseudomonas fluorescens	S97	Induced resistance	P. syringae pv. phaseolicola	Bean	In vitro demonstration
Pseudomonas putida		Induced resistance	P. syringae pv. lachrymans	Cucumber	In field demonstration
Binucleate *Rhizoctonia*	BNR-AG-K	Induced resistance	Rhizoctonia solani AG-4	Soybean	In vitro Demonstration

44). *Bacillus subtilis* strains show broad suppression of various plant pathogens by producing an antibiotic iturin in vitro and in vivo (22, 63). Recently, the gene(s) encoding the iturin has been cloned and identified by Matsuno et al. (63). Similarly, studies performed with nonproducing antibiotic strains of *Erwinia herbicola* and with antibiotic-resistant mutants of *E. amylovora* have also indicated that β-lactam antibiotic are important in the inhibition of *E. amylovora by E. herbicola* (37). *Streptomyces hygroscopicus* var. *geldanus* produces the antibiotic geldanamycin, which has been directly purified from soil and demonstrated to effectively suppress root rot of pea caused by *Rhizoctonia solani* in the field (79). Numerous strains of *Enterobacter cloacae* have also been shown to be effective biocontrol agents in suppressing *Pythium* damping off of cucumber and cotton. One study has also indicated that ammonia produced by the bacterium may be involved in disease suppression in vitro (44).

Apparently, antibiotic production is not specific for certain species.Different species may produce the same antibiotics or secondary metabolites, while different strains of the same species may possess quite different antibiotics or toxic substances (see Table 1). For example, *P. aureofaciens* strain 30-84 produces Phz while *P. aureofaciens* strain Q2-87 produces Phl (44). *Pseudomonas fluorescens* strain CHA0 produces Phl, hydrogen cyanide, and pyoluteorin (38, 45, 65, 66), while other strains of the same bacterial species may produce oomycin A, phenazine-1-carboxylic acid, anthranilic acid, and pyoluteorin (33, 42, 72).

The role of antibiotic production by antagonistic fungi has been less studied than with bacteria. One reason may be that these substances have merely been identified, and the role has not yet been elucidated clearly by molecular approach. The role of antibiosis in biocontrol fungi has been considered, however. *Gliocladium* (now *Trichoderma*) *virens*, which controls damping off of cotton caused by *P. ultimum*, produces gliovirin. Mutant analysis has been used to demonstrate that the antibiotic gliovirin plays a role in biocontrol (18, 32). In addition, the importance of gliotoxin

produced by *Trichoderma virens* in the suppression of Pythium damping-off of cotton seedlings has also been confirmed recently by mutational analysis (16,96). Other antibiotics from fungi are listed in Table 1. For example, chaetomin is produced by *Chaetomium globosum*, peptaibols are produced by *Trichoderma harzianum*, and pyrones are produced by *Trichoderma* spp. (15,19,21,80). However, the roles of these antibiotics have not yet been demonstrated in vivo by genetic analysis.

Competition:

This process is considered to be an indirect interaction whereby pathogens are excluded by depletion of a food base or by physical occupation of site (61).Generally, nutrient competition has been believed to have an important role in disease suppression, although it is extremely difficult to obtain conclusive evidence. Biocontrol by nutrient competition can occur when the biocontrol agent decreases the availability of a particular substance thereby limiting the growth of the pathogen. Particularly, the biocontrol agents have a more efficient uptake or utilizing system for the substance than do the pathogens (26, 30, 69). For example, iron competition in alkaline soils may be a limiting factor for microbial growth in such soils (47). Some bacteria, especially fluorescent pseudomonads produce siderophores that have very high affinities for iron and can sequester this limited resource from other microflora thereby preventing their growth (57). A few studies have demonstrated that siderophore biosynthesis in *P. fluorescens* plays a role in pathogen suppression (12, 47, 56, 57). For example, Schippers and coworkers have demonstrated that siderophores are involved in the biocontrol of minor pathogens of potato when they use a single Tn5 insertion to inactivate both the pyoverdine production (Pvd-) and plant growth promotion phenotypes of *P. putida* strain WC358 (6). Similarly, *P. fluorescens* 3551 controls Pythium damping-off of cotton, while Pvd- derivatives do not control this disease (56). However, some studies have also found siderophores to play little or no role in disease control, particularly with *Pythium* species (23,39,76). For example, Paulitz and Loper obtained Tn5 mutants of *P. putida* strain N1R, which is deficient in pyoverdine production, and showed no reduction in ability to protect cucumber from Pythium damping off (70). Lam and Gaffney have thus suggested that the role of siderophores in biocontrol should be viewed with caution because some strains may synthesize a variety of antifungal compounds under iron-limited conditions (44). More recently, Leeman et al. have reported that iron-chelating salicylic acid produced by selected *P. fluorescens* strains at low iron availability may be involved in the induction of systemic resistance to Fusarium wilt of radish. They thus suggested that the role of siderophore-mediated competition for iron in the suppression of disease by fluorescent *Pseudomonas spp*. might need reevaluation (46).

Competition for specific substances or stimulants for germination of microorganisms may also occur in soil since most resting structures of microbes cannot germinate without specific stimulants due to soil fungistasis (43). Infection of plants by pathogens occurs only after dormancy is broken in the presence of stimulants from plant hosts. Consequently, microbes including pathogens may compete for specific stimulants of germination that may come from germinating seeds or growing roots. These factors may include fatty acids, or their peroxidation products (30), or volatile components such as ethanol and acetaldehyde (20,48,75). Generally, seeds are more vulnerable to infection of *Pythium* spp. during the first 6-12 hr of seed germination. After that, however, developing seedlings become less susceptible to *Pythium* infection (30). Thus, the volatile molecules produced by seeds and/or roots may be particularly important in stimulating "rapid response" pathogens such as *Pythium* spp. due to extension-speed of the substances. Nelson (68) has reported that germination of sporangia of *Pythium* spp. is evident within 2 hr after exposure to volatiles from germinating cotton and other plant seeds. Maximum germination occurs following 8 hr of exposure to volatiles. Norton and Harman also observed approximately 60-fold increases in populations of *P. ultimum* after exposure to volatiles from aged pea seeds in soil (71). In addition, C_{16}-C_{18} fatty acids also stimulated the germination of endoconidia and chlamydospores of *Thielaviopsis basicola* in vitro (73). Harman et al. have also proposed that the volatile peroxidation products of unsaturated fatty acids may be the active stimulants for fungal germination, since as little as 200 mg/L of 2,4- hexadienal in aerial solution stimulated germination of *Alternaria alternata* conidia (28).

Consequently, a biocontrol agent can provide plant protection by efficient interception of these stimulating factors before pathogens can use them (17, 69, 75).Molecular evidence shows that strain E6 of *Enterobacter cloacae* inactivates the fatty acid that stimulates *Pythium* spp. germination, thereby protecting seeds (88). Elad and Chet have also proposed that some bacterial strains may catabolize exudate components that are responsible for stimulating

oospore germination of *Pythium* spp.; these effective strains do not produce inhibitory metabolites and do not directly interact with oospores of *Pythium* spp. (17). In other studies, stimulant inactivation may also play a role for other biocontrol agents such as *Enterobacter cloacae*, *Trichoderma harzianum*, or *Pseudomonas putida*; pea, cotton, and soybean seeds developed significantly lower levels of ethanol and acetaldehyde during germination when treated with these bioprotectants as compared with untreated seeds (20, 69, 74). Thus, with the identification of specific pathogen stimulants, it should be possible to determine the role of microbial metabolism of these stimulants in biocontrol by examining mutants that can no longer metabolize them.

Microbes may also compete for physical space (site) and nutrients for growth.For example, spraying pine sumps with spore suspensions of *Peniophora* (now *Phlebia*) *gigantea* prevents infection by *Heterobasidion* (*Fomes*) *annosum*. Because the pathogen cannot gain a foothold for establishment on the host, the biocontrol agent can thus reduce the disease severity of root rot of pine (11, 62). Although there may be some antagonism (e.g., antibiosis) between the two fungi, the primary mechanism may be simply competition for the physical occupation of specific infection sites (62).Thus, the competition for infection site also plays a role in biological control of plant diseases.

Parasitism:

This process involves the direct utilization of one organism as food by another (26, 62). Fungi that are parasitic on other fungi are usually referred to as mycoparasites (5). Many mycoparasites occur on a wide range of fungi and some of them have been proposed to play an important role in disease control (1, 52, 62). For example, *Darluca filum* (now *Sphaerellopsis filum*) was described by Saccardo as a parasite of some rust fungi, especially *Puccinia* and *Uromyces* (84). Weindling in 1932 observed *Trichoderma lignorum* (*T. viride*) parasitizing hyphae of *Rhizoctonia solani* and suggested inoculating soil with *Trichoderma* spores to control damping off of citrus seedling (92). This and other *Trichoderma* species were observed to parasitize *Rhizoctonia bataticola* and *Armillaria mellea* (5,55). More recently *T. harzianum* and *T. hamatum* have been marketed as wound dressings for ornamental and forest trees and decay inhibitors for utility poles (1,62). Other mycoparasites include *Pythium oligandrum* and *Pythium nunn* for parasitizing various *Pythium* spp.; *Coniothyrium minitans* and *Sporidesmium sclerotivorum* that parasitize various sclerotia forming fungi also have been used in disease control (1, 84).

Mycoparasitism is a process by which biocontrol fungi may attack pathogenic fungi (61). Generally, mycoparasitism can be described as a four-step process (8, 26, 30, 44, 52, 86): The first stage is chemotropic growth. The biocontrol fungi grow tropistically toward the target fungi that produce chemical stimuli. For example, a volatile or water-soluble substance produced by the host fungus serves as a chemoattractant for parasites. However, the lack of available data for statistical comparison of different conditions or host-parasite combinations is a limitation to understanding the phenomenon (13). The next step is recognition. Lectins of hosts (pathogens) and carbohydrate receptors on the surface of the biocontrol fungus may be involved in this specific interaction (35, 36). The third step is attachment and cell wall degradation. Mycoparasites can usually either coil around host hyphae or grow alongside it and produce cell wall degrading enzymes to attack the target fungus (8, 30). These enzymes such as chitinases and b-1,3-glucanase may be involved in degradation of host cell walls and may be components of complex mixtures of synergistic proteins that act together against pathogenic fungi (15, 29, 51, 52, 58, 59, 61). The final step is penetration. The biocontrol agent produces appressoria-like structures to penetrate the target fungus cell wall (8,13).

Evidence for these processes in *Trichoderma* spp. and other fungi has been presented. Most of these events have been described from in vitro studies even though mycoparasitic structures have been observed in situ on seeds (34, 55). Recently, a lectin from *Sclerotium rolfsii* has been isolated and proven to play an important role in recognition (34). However, the biochemical basis for this phenomenon is not understood. Similarly, cell- wall-degrading enzymes have also been shown to be involved in the inhibition of pathogenic fungi. The gene(s) for some of those enzymes has (have) been isolated (29, 31, 59, 61).

Another mycoparasitic fungus, *Sporidesmium sclerotivorum*, is a biotrophic parasite and is often found only on sclerotia of plant pathogenic fungi such as *Sclerotinia minor* and *Sclerotium cepivorum* (the causal agents of lettuce drop) (3, 26). In field experiments, the evidence has shown that mycoparasitic activity is consistently correlated with a reduction in pathogen inoculum density and a reduction in disease incidence (1, 2).

Cell-wall degrading enzymes:

Extracellular hydrolytic enzymes produced by microbes

may also play a role in suppression of plant pathogenic fungi. Chitin and b-1,3-glucans are major constituents of many fungal cell walls (44). Several studies have demonstrated in vitro lysis of fungal cell walls either by chitinase or b-1,3-glucanase alone or in combination (29, 44, 58, 59, 61). Recently, genetic evidence for the role of these enzymes in biocontrol has been obtained. A chitinase (ChiA) deficient mutant of *Serratia marcescens* was shown to have reduced inhibition of fungal germ tube elongation and reduced biocontrol of Fusarium wilt of pea seedling in a greenhouse assay (44). Furthermore, when ChiA from *S. marcescens* was inserted into the nonbiocontrol agent *Escherichia coli*, the transgenic bacterium reduced disease incidence of Southern blight of bean caused by *Sclerotium rolfsii* (82). Similarly, *Trichoderma harzianum* was transformed with ChiA from *S. marcescens* (27). The transformed strains were more capable of overgrowing *Sclerotium rolfsii* in vitro than the original strain from which it was derived. More recently, several species of transgenic plants containing the gene for endochitinase from *T. harzianum* have been produced by Harman and coworkers (28). These transgenic plants als, have increased resistance against plant pathogenic fungi (60). These results indicate that these enzymes play a role in biocontrol and the biocontrol ability of some microbes may be improved by transformation with chitinolytic enzymes.

Induction of systemic resistance:

The inducible resistance in plants to a variety of pathogens is known as systemic acquired resistance (SAR). SAR may be induced by inoculating plants either with a necrogenic pathogen or nonpathogen or with certain natural or synthetic chemical compounds (41, 44, 81, 83). These defense responses may include the physical thickening of cell walls by lignification, deposition of callose, accumulation of antimicrobial low-molecular-weight substances (e.g., phytoalexins), and synthesis of various proteins (e.g., chitinases, glucanases, peroxidases, and other pathogenesis-related (PR) proteins) (24, 25, 41, 81, 83).

This defense system is also triggered when plants are colonized by plant growth- promoting rhizobacteria (PGPR) (83) and a few binucelate *Rhizoctonia* (BNR) AG-K (78). Recently, many strains of PGPR have been shown to be effective in controlling plant diseases by inducing plant systemic resistance (4, 9, 49, 50). Similarly, Paromarto et al., also implied that induced resistant is the mechanism of biocontrol of *Rhizoctonia solani* on soybean by BNR (78). Plants colonized by these strains are more resistant to foliar diseases, even though the PGPR is present only on roots (90).Strain CHA96 is a mutant of strain CHA0 that is defective in the production of antibiotics and the suppression of black root rot of tobacco. Maurhofer et al. (1994) reported that strain CHA96 could induce PR-proteins (e.g., endochitinases and b- 1,3-glucanases) in the intercellular fluid of leaves and thus could increase resistance to TNV as did the wild-type strain (64). van Peer et al. also found increased amounts of phytoalexins in *P. fluorescens* strain WCS417r-inoculated plants when compared to nonbacterized plants after challenge inoculation (89). Although induced systemic disease resistance has been studied mainly in laboratories and greenhouses, some recent reports have indicated that microbial-induced SAR can protect crops from pathogen infection under field conditions by treatment of these beneficial microorganisms (14, 87, 90).

The chemical compounds that induce resistance of plants to pathogens may include polyacrylic acid, ethylene, salicylic acid and acetyl salicylic acid, various amino acid derivatives, the herbicide phosphinotricin, and harpin produced by *Erwinia amylovora* (7, 41, 81, 91). Recently, Leeman et al. have also found that the lipopolysaccharide with the O-antigenic side chain produced by strain WCS374 of *P. fluorescens* is involved in induction of systemic resistance in radish to Fusarium wilt (46). *P. fluorescens* strain CHA0 effectively controlled take-all of wheat caused by *G. graminis* var. *tritici* and has been found in the root cortex. This strain can produce metabolites that may result in enhanced stress for the plant when the metabolites are delivered into the plant cells (64, 66). It is known that stress can induce defense mechanisms against pathogens (64). However, the hypothesis should be proved by genetic analysis such as heterologous expression, which shows that inducing ability may be transferred to other potent strains as an additional complementary mode of action, and gene mutation, which knocks out the ability and leads to less disease control.

Conclusion and perspectives

A successful biocontrol requires considerable understanding of cropping system; disease epidemiology; the biology, ecology, and population dynamics of biocontrol organisms; and the interactions among these variables (52, 55). Understanding the mechanisms or activities for antagonist-pathogen interactions will be one of important steps because it may provide a reasonable basis for selection and construction of more effective biocontrol agents (26, 53, 54). Over the past few years, the novel applications of

molecular techniques have broadened our insight into the basis of biological control of plant diseases. New molecular approaches have been available for assessment of interaction between the antagonist and pathogen, ecological traits of antagonists in rhizosphere and improving the efficacy of bacterial, fungal and viral biocontrol agent (55). Consequently, there has been a significant increase in the number of biological disease control agents registered or on the market worldwide in the last few years (51, 54, 95). For example, there currently are approximately 30 bacterial and fungal products for control of foliar, soil-borne and post-harvest diseases (10, 95).

Currently, the Department of Agriculture in the USA has posted four agriculturally motivated movements, which can use biocontrol for solving disease problems. Particularly, the mandate to convert a large percentage (75 %) of US agriculture to integrated pest management (IPM) requires biocontrol technologies to reduce chemical pesticide usage. Biological control will thus be an alternative strategy for the control of plant diseases given the history of fungicides in the near future. However, other methods in IPM for crop disease control are still necessary in various environmental conditions, because an agroecosystem is a variable and functioning system that includes several factors that influence disease and crop development (62). Consequently, for economic threshold, other control strategies of IPM besides biological control should be also considered and applied to effectively reduce the disease development and the yield loss of crops in the different crop systems.

LITERATURE CITED

1. Adams, P. B. 1990. The potential of mycoparasites for biological control of plant diseases. Annu. Rev. Phytopathol. 28:59-72.
2. Adams, P. B., and Ayers, W. A. 1982. Biological control of Sclerotinia lettuce drop in the field by *Sporidesmium sclerotivorum*. Phytopathology 72:485-488.
3. Adams, P. B., and Ayers, W. A. 1983. Histological and physiological aspects of infection of sclerotia of *Sclerotinia* species by two mycoparasites.Phytopathology 73:1072.
4. Alstrom, S. 1995. Evidence of disease resistance induced by rhizosphere pseudomonad against *Pseudomonas syringae pv. phaseolicola*. J. Gen. Appl. Microbiol. 41:315-325.
5. Baker, K. F., and Cook, R. J. 1974. Biological Control of Plant Pathogens.Am. Phytopathol. Soc., St. Paul. MN. 433 pp.
6. Bakker, P. A., Lamers, H. M., Bakker, A. W., Marrugo, J. D., Weisbeek, P. J., and Schippers, B. 1986. The role of siderophores in potato tuber yield increase by *Pseudomonas putida* in a short rotation of potato. Neth. J. Pl. Path. 92:249-256.
7. Bol, J. F., Linthorst, H. J. M., and Cornelissen, B. J. C. 1990. Plant pathogenesis-related proteins induced by virus infection. Annu. Rev. Phytopathol. 28:113-138.
8. Chet, I. 1987. *Trichoderma* application, mode of action, and potential as biocontrol agent of soil-borne pathogenic fungi. Pages 137-160. in: Innovative Approaches to Plant Disease Control. I. Chet, ed., John Wiley, New York.
9. Chet, I., and Baker, R. 1980. Induction of suppressiveness to *Rhizoctonia solani* in soil. Phytopathology 70:994-998.
10. Cook, R. J. 1998. Biological control in the 21th century: meeting the challenges and expectations. 7th international Congress of Plant Pathology, invited papers abstract Vol. 1: 5.2.10S. BSPP., Edinburgh, Scotland.
11. Cook, R. J., and Baker, K. F. 1983. The Nature and Practice of Biological Control of Plant Pathogens. American Phytopathological Society, St. Paul, MN.
12. Costa, J. M., and Loper, J. E. 1994. Characterization of siderophore production by the biological control agent *Enterobacter cloacae*. MPMI 7:440-448.
13. Deacon, J. W., and Berry, L. A. 1992. Models of action of mycoparasites in relation to biocontrol of soilborne plant pathogens. Pages 157-167. in: Biological Control of Plant Diseases: Progress and Challenges for The Future. E. C. Tjamos, G. C. Papavizas, and R. J. Cook, eds., Plenum Press, New York.
14. Dehne, H. W., Stenzel, K., and Schonbeck, F. 1984. The efficiency of induced resistance under practical culture conditions. III. Reproduction of powdery mildew on induced resistant plants. J. Plant Dis. Prot. 91:258-265.
15. Di Pietro, A., Gut-Rella, M., Pachlatko, J. P., and Schwinn, F. J. 1992. Role of antibiotics produced by *Chaetomium globosum* in biocontrol of *Pythium ultimum*, a causal agent of damping-off. Phytopathology 82:131-135.
16. Di Pietro, A., Lorito, M., Hayes, C. K., Broadway, R. M., and Harman, G. E.1993. Endochitinase from *Gliocladium virens*: isolation, characterization and synergistic antifungal activity in combination with gliotoxin. Phytopathology 83:308-313.
17. Elad, Y., and Chet, I. 1987. Possible role of competition

for nutrients in biocontrol of Pythium damping-off by bacteria. Phytopathology 77:190-195.

18. Fravel, D. R. 1988. Role of antibiosis in the biocontrol of plant diseases.Annu. Rev. Phytopathol. 26:75-91.
19. Ghisalberti, E. L., and Sivasithamparam, K. 1991. Antifungal antibiotics produced by *Trichoderma* spp. Soil Biol. Biochem. 23:1011-1020.
20. Gorecki, R. J., Harman, G. E., and Mattick, L. R. 1985. The volatile exudates from germinating pea seeds of different viability and vigor. Can. J. Botany 63:1035-1039.
21. Graeme-Cook, K. A., and Faull, J. L. 1991. Effect of ultraviolet-induced mutants of *Trichoderma harzianum* with altered antibiotic production on selected pathogens in vitro. Can. J. Microbiol. 37:659-664.
22. Gueldner, R. C., Reilly, C. C., Pusey, P. L., Costello, C. E., Cox, R. H., Crumley, F. G., and Cutler, H. G. 1988. Isolation and identification of iturins as antifungal peptides in biological control of peach brown rot with *Bacillus subtilis*. J. Agric. Food Chem. 36:366-370
23. Hamdan, H., Weller, D. M., Thomashow, L. S. 1991. Relative importance of fluorescent siderophores and other factors in biological control of *Gaeumannomyces graminis var. tritici* by *Pseudomonas fluorescens* 2-79 and M4- 80R. Appl. Environ. Microbiol. 57:3270-3277.
24. Hammerschmidt, R., and Kuc, J. 1982. Lignification as a mechanism for induced systemic resistance in cucumber. Physiol. Plant Pathol. 20:61-71.
25. Hammerschmidt, R., Lamport, D. T. A., and Muldoon, E. P. 1984. Cell wall hydroxyproline enhancement and lignin deposition as an early event in the resistance of cucumber to *Cladosporium cucumerinum*. Physiol. Plant Pathol. 24:43-47.
26. Handelsman Jo., and Parke, J. L. 1989. Mechanisms in biocontrol of soilborne plant pathogens. Pages 27-61. in: Plant-Microbe Interactions, Molecular and Genetic Perspectives, Vol. 3. T. Kosuge, and E. W. Nester, eds., McGraw-Hill, New York.
27. Haran, S., Schickler, H., Peer, S., Logeman, S., Oppenheim, A., and Chet, I.1993. Increased constitutive chitinase activity in transformed *Trichoderma harzianum*. Biol. Control 3:101-108.
28. Harman, G. E., and Hayes, C. K. 1994. Biologically-based Technologies for Pest Control: Pathogens that are Pests of Agriculture. A Report to the Office of Technology Assessment, US Congress. 75 pp.
29. Harman, G. E., Hayes, C. K., Lorito, M., Broadway, R. M., Di Pietro, A., Peterbauer, C., and Tronsmo, A. 1993. Chitinolytic enzymes of *Trichoderma harzianum* purification of chitobiosidase and endochitinase. Phytopathology 83:313-318.
30. Harman, G. E., and Nelson, E. B. 1994. Mechanisms of protection of seed and seedlings by biological control treatments: Implications for practical disease control. Pages 283-292. in: Seed Treatment: Progress and Prospects. T. Martin, ed., BCPC, Farnham, UK.
31. Hayes, C. K., Lorito, M., Di Pietro, A., Harman, G. E., and Tronsmo, A. 1994. Isolation and sequence of an endochitinase gene from *Trichoderma harzianum*. Gene 135:143-148.
32. Howell, C. R., and Stipanovic, R. D. 1983. Gliovirin, a new antibiotic from *Gliocladium virens*, and its role in the biological control of *Pythium ultimum*.Can. J. Microbiol. 29:321-324.
33. Howie, C. R., and Suslow, T. V. 1991. Role of antibiotic biosynthesis in the inhibition of *Pythium ultimum* in the cotton spermosphere and rhizosphere by *Pseudomonas fluorescens*. Mol. Plant Microbe Interact. 4:393-399.
34. Hubbard, J. B., Harman, G. E., and Eckenrode, C. J. 1982. Interaction of a biological control agent, *Chaetomium globosum*, with seed coat microflora. Can. J. Microbiol. 28:431-437.
35. Inbar, J., and Chet, I. 1992. Biomimics of fungal cell-cell recognition by use of lectin-coated nylon fibers. J. Bacteriol. 174:1055-1059.
36. Inbar, J., and Chet, I. 1994. A newly isolated lectin from the plant pathogenic fungus *Sclerotium rolfsii*: purification, characterization and role in mycoparasitism. J. Microbiol. 140:651-657.
37. Kearns, L. P., and Mahanty, H. K. 1998. Antibiotic Production by *Erwinia herbicola* Eh1087: Its role in inhibition of *Erwinia amylovora* and partial characterization of antibiotic biosynthesis genes. Appl. Environ. Microbiol. 64:1837-1844.
38. Keel, C., Schnider, U., Maurhofer, M., Voisard, C., Laville, J., Burger, U., Wirthner, P., Haas, D., and Defago, G. 1992. Suppression of root diseases by *Pseudomonas fluorescens* CHA0 importance of the bacterial secondary metabolite 2,4-diacetylphloroglucinol. Mol. Plant Microbe Interact. 5:4-13.
39. Keel, C., Wirthner, P., Berling, C. H., Kahr, G., and Defago, G. 1989. Iron sufficiency, a pre-requisite for the suppression of tobacco black root rot by *Pseudomonas fluorescens* strain CHA0 under gnotobiotic condition.

Phytopathology 79:584-589.

40. Kerr, A. 1989. Commercial release of a genetically-engineered bacterium for the control of crown gall. Agric. Sci. 2:41-48.
41. Kessmann, H., Staub, T., Hofmann, C., Maetzke, T., Herzog, J., Ward, E., Uknes, S., and Ryal, J. 1994. Induction of systemic acquired disease resistance in plants by chemicals. Annu. Rev. Phytopathol. 32:439-459.
42. Kraus, J., and Loper, J. 1995. Characterization of a genomic region required for production of the antibiotic pyoluteorin by biological control agent *Pseudomonas fluorescens* Pf5. Appl. Environ. Microbiol. 61:849-854.
43. Ko, W. H., and Lockwood, J. L. 1970. Mechanism of lysis of fungal mycelia in soil. Phytopathology 60:148-154.
44. Lam, S. T., and Gaffney, T. D. 1993. Biological activities of bacteria used in plant pathogen control. Pages 291-320. in: Biotechnology in Plant Disease Control.I. Chet, ed., John Wiley, New York.
45. Laville, J., Voisard, C., Keel, C., Maurhofer, M., Defago, G., and Haas, D. 1992. *Pseudomonas fluorescens* mediating antibiotic synthesis and suppression of black root rot of tobacco. Proc. Natl. Acad. Sci. (USA) 89:1562-1566.
46. Leeman, M., Den Ouden, F. M., Van Pelt, J. A., Dirkx, F. P. M., Steijl, H., Bakker, P. A. H. M., and Schippers, B. 1996. Iron availability affects induction of systemic resistance to Fusarium wilt of radish by *Pseudomonas fluorescens*. Phytopathology 86:149-155.
47. Leong, S. A., and Expert, D. 1989. Siderophores in plant-pathogen interactions. Pages 62-83. in: Plant-Microbe Interactions, Molecular and Genetic Perspectives, Vol. 3. T. Kosuge, and E. W. Nester, eds., McGraw-Hill, New York.
48. Linderman, R. G., and Gilbert, R. G. 1969. Stimulation of *Sclerotium rolfsii* in soil by volatile components of alfalfa hay. Phytopathology 59:1366-1372.
49. Liu, L., Kloepper, J. W., and Tuzun, S. 1995a. Induction of systemic resistance in cucumber against Fusarium wilt by plant growth-promoting rhizobacteria.Phytopathology 85:695-698.
50. Liu, L., Kloepper, J. W., and Tuzun, S. 1995b. Induction of systemic resistance in cucumber against bacterial angular leaf spot by plant growth-promoting rhizobacteria. Phytopathology 85:843-847.
51. Lo, C-T. 1997. Development and application of biological control on crop disease management. Pages 141-150, in : Proceeding of New Techniques of Plant Protection. C-T., Lo and L-Y., Chou, eds., TARI., Taichung, Taiwan. (In Chinese with English Abstract)
52. Lo, C-T. 1997. Biological control of turfgrass diseases using *Trichoderma harzianum*. Plant Prot. Bull. 39: 207-225.
53. Lo, C. T., Nelson, E. B., and Harman, G. E. 1996. Biological control of turfgrass diseases with a rhizosphere competent strain of *Trichoderma harzianum*. Plant Dis. 80:736-741.
54. Lo, C. T., Nelson, E. B., and Harman, G. E. 1997. Improved biocontrol efficacy of *Trichoderma harzianum* 1295-22 for foliar phases of turf diseases by use of spray application. Plant Dis. 81:1132-1138.
55. Lo, C. T., Nelson, E. B., Hayes, C. K.,and Harman, G. E. 1998. Ecological studies of transformed *Trichoderma harzianum* strain 1295-22 in the rhizosphere and on the phylloplane of creeping bentgrass. Phytopathology 88: 129-136.
56. Loper, J. E. 1988. Role of fluorescent siderophore production in biological control of *Pythium ultimum* by *Pseudomonas fluorescens* strain. Phytopathology 78:166-172.
57. Loper, J. E., and Buyer, J. S. 1991. Siderophores in microbial interactions on plant surfaces. Molec. Plant Microbe Interact. 4:5-13.
58. Lorito, M., Harman, G. E., Hayes, C. K., Broadway, R. M., Tronsmo, A., Woo, S. L., and Di Pietro, A. 1993. Chitinolytic enzymes produced by *Trichoderma harzianum* : antifungal activity of purified endochitinase and chitobiosidase.Phytopathology 83:302-307.
59. Lorito, M., Hayes, C. K., Di Pietro, A., Woo, S. L., and Harman, G. E. 1994.Purification, characterization, and synergistic activity of a glucan 1,3-glucosidase and an N-acetyl-beta-glucosaminidase from *Trichoderma harzianum*. Phytopathology 84:398-405.
60. Lorito, M., Hayes, C. K., Peterbauer, C., Tronsmo, A., Klemsdal, S., and Harman, G. E. 1993. Antifungal chitinolytic enzymes from *Trichoderma harzianum* and *Gliocladium virens*: purification, characterization, biological activity and molecular cloning. Pages 383-392. in: Chitin Enzymology. R A. Muzzarelli, ed., Eur. Chitin Soc., Ancona, Italy.
61. Lorito, M., Hayes, C. K., Zoina, A., Scala, F., Del-Sorbo, G., Woo, S. L., and Harman, G. E. 1994. Potential of genes and gene products from *Trichoderma* spp. and *Gliocladium* spp. for the development of biological pesticides.Molecular Biotechnology 2:209-217.

62. Maloy, O. C. 1993. Plant Disease Control: Principles and Practice. John Wiley & Sons, Inc., New York. 346 pp.
63. Matsuno, Y., Hitomi, T., Ano, T., and Shoda, M. 1992. Transformation of *Bacillus subtilis* antifungal antibiotic iturin producers with isolated antibiotic resistance plasmids. J. Gen. Appl. Microbiol. 38:13-21.
64. Maurhofer, M., Hase, C., Meuwly, P., Metraux, J. P., and Defago, G. 1994.Induction of systemic resistance to tobacco necrosis virus. Phytopathology 84:139-146.
65. Maurhofer, M., Keel, C., Haas, D., and Defago, G. 1995. Influence of plant species on disease suppression by *Pseudomonas fluorescens* strain CHA0 with enhanced antibiotic production. Plant Pathology 44:40-50.
66. Maurhofer, M., Keel, C., Schnider, U., Voisard, C., Haas, D., and Defago, G.1992. Influence of enhanced antibiotic production in *Pseudomonas fluorescens* CHA0 on its disease suppressive capacity. Phytopathology 82:190-195.
67. Mazzola, M., Fujimoto, D. K., Thomashow, L. S., and Cook, R. J. 1995.Variation in sensitivity of *Gaeumannomyces graminis* to antibiotics produced by fluorescent *Psudomonas* spp. and effect on biological control of take-all of wheat. Appl. Environ. Microbiol. 61:2554-2559.
68. Nelson, E. B. 1987. Rapid germination of sporangia of *Pythium* species in response to volatiles from germinating seeds. Phytopathology 77:1108-1112.
69. Nelson, E. B. 1990. Exudate molecules initiating fungal responses to seeds and roots. Plant Soil 129:61-73.
70. Nelson, E. B. 1995. Nontarget effects of fungicide applications. Turfgrass Trends 4:1-9.
71. Norton, J. M., and Harman, G. E. 1985. Responses of soil microorganisms to volatile exudates from germinating pea seeds. Can J. Bot. 63:1040-1045.
72. Ownely, B. H., Weller, D. M., and Thomashow, L. S. 1992. Influence of in situ and in vitro pH on suppression of *Gaeumannomyces graminis* var. *tritici* by *Pseudomonas fluorescens.* Phytopathology 82:178-184.
73. Papavizas, G. C., and Kovacs, M. F., Jr. 1972. Stimulation of spore germination of *Thielaviopsis basicola* by fatty acids from rhizosphere soil. Phytopathology 62:688-694.
74. Paulitz, T. C. 1990. The stimulation of *Pythium ultimum* by seed volatiles and the interaction of *Pseudomonas putida*. Phytopathology 80:994-995.
75. Paulitz, T. C. 1991. Effect of *Pseudomonas putida* on the stimulation of *Pythium ultimum* by seed volatiles of pea and soybean. Phytopathology 81:1282- 1287.
76. Paulitz, T. C., and Loper, J. E. 1991. Lack of a role for fluorescent siderophore production in the biological control of Pythium damping-off of cucumber by a strain of *Pseudomonas putida*. Phytopathology 81:930-935.
77. Poplawsky, A. R., Peng, Y. F., and Ellingboe, A. H. 1988. Bacterial Tn5 mutants affected in antibiosis to *Gaeumannomyces graminis var. tritici*. Phytopathology 78:426-432.
78. Poromarto, S. H., Nelson, B. D., and Freeman, T. P. 1988. Association of binucleate *Rhizoctonia* with soybean and mechanism of biocontrol of *Rhizoctonia solani*. Phytopathology 88:1056-1067.
79. Rothrock, C. S., and Gottlieb, D. 1984. Role of antibiosis in antagonism of *Streptomyces hygroscopicus var. geldanus* to *Rhizoctonia solani* in soil. Can. J. Microbiol. 30:1440-1447.
80. Schirmbock, M., Lorito, M., Wang, Y-L., Hayes, C. K., Arisan-Atac, I., Scala, F., Harman, G. E., and Kubicek, C. 1994. Parallel formation and synergism of hydrolytic enzymes and peptaibol antibiotics, molecular mechanisms involved in the antagonistic action of *Trichoderma harzianum* aganist phytopathogenic fungi. Appl. Environ. Microbiol. 60:4364-4370.
81. Sequeira, L. 1983. Mechanisms of induced resistance in plants. Ann. Rev. Microbiol. 37:51-79.
82. Shapira, R., Ordentlich, A., Chet, I., and Oppenheim, A. B. 1989. Control of plant diseases by chitinase expressed from cloned DNA in *Escherichia coli*. Phytopathology 79:1246-1249.
83. Sticher, L., Mauch-Mani, and Metraux, J. P. 1997. Systemic Acquired resistance. Annu. Rev. phytopathol. 35:235-270.
84. Sundheim, L., and Tronsmo, A. 1988. Hyperparasites in biological control. Pages 53-69. in: Biocontrol of Plant Diseases. K. G. Mukerji, and K. L. Garg, eds., CRC Press, Boca Raton, FL.
85. Thomashow, L. S., and Weller, D. M. 1988. Role of phenazine antibiotic from *Pseudomonas fluorescens* in biological control of *Gaeumannomyces graminis var. tritici*. J. Bacteriol. 170:3499-3508.
86. Tunlid, A., Johansson, T., and Nordbring-Hertz, B. 1992. Fungal attachment to nematodes. Mycol. Res. 96:401-412.
87. Tuzun, S., Nesmith, W., Ferriss, R. S., and Kuc, J. 1986. Effects of stem injections with *Peronospora tabacina* on growth of Tobacco and protection against blue mold in the field. Phytopathology 76:938-941.
88. van Dijk, K., and Nelson, E. B. 1997. Inactivation of seed

exudate stimulants of *Pythium ultimum* sporangium germination by strains of *Enterobacter cloacae* and other seed-associated bacteria. Appl. Environ. Microbiol. 63:331-335.

89. van Peer, R. G., Niemann, G. J., and Schippers, B. 1991. Induced resistance and phytoalexin accumulation in biological control of Fusarium wilt of carnation by Pseudomonas sp. strain WCS417r. *Phytopathology* 81:728-734.
90. Wei, G., Kloepper, J. W., and Tuzum, S. 1996. Induced systemic resistance to cucumber diseases and increased plant growth by plant growth-promoting rhizobacteria under field conditions. Phytopathology 86:221-224.
91. Wei, Z-M., Laby, R. J., Zumoff, C. H., Bauer, D. W., He, S. Y., Collmer, A., and Beer, S. V. 1994. Harpin, elicitor of the hypersensitive response produced by the plant pathogen *Erwinia amylovora*. Science 257:85-88.
92. Weindling, R., and Fawcett, H. S. 1936. Experiments in the control of Rhizoctonia damping off of citrus seedling. Hilgardia 10:1-16.
93. Weller, D. M. 1988. Biological control of soilborne plant pathogens in the rhizosphere with bacteria. Annu. Rev. Phytopathol. 26:379-407.
94. Weller, D. M., and Thomashow, L. S. 1993. Microbial metabolites with biological activity against plant pathogens. Pages 173-180. in: Pest Management: Biologically Based Technologies. R. D. Lumsden, and J. L. Vaughn, eds., American Chemical Society, Washington, D. C.
95. Whipps, J. M. 1998. Microbial interactions as basis for biological control of fungi diseases. 7th international Congress of Plant Pathology, invited papers abstract Vol. 1:2.10.1S. BSPP., Edinburgh, Scotland.
96. Wilhite, S. E., Lumsden, R. D., and Straney, D. C. 1994. Mutational analysis of gliotoxin production by the biocontrol fungus *Gliocladium virens* in relation to suppression of Pythium damping-off. Phytopathology 84:816-821.

摘　要

羅朝村. 1998. 微生物防治作物病害之一般作用機制 . 植病會刊 7:155-166. ([1.] 台中縣、農業試驗所植物病理系. [2.] 連絡作者：電子郵件 ctlo@wufeng.tasi.gov.tw；傳真 04-3338162)。

生物防治是目前一極具吸引力的作物病害防治替代策略；亦是執行永續農業體系目標發展的重要步驟之一。由於作物病害生物防治主要是在利用一或多種有益微生物來減少病害，因此瞭解病害生物防治的機制，特別是拮抗微生物與病原菌間的相互作用，將有助於吾人去管理或創造一個適合環境，以利生物防治的成功或改進生物防治策略。近年來，由於生物技術被引入病害生物防治機制的探討，使得人們對於拮抗微生物如何抑制病原菌的複雜過程；特別是不同的遺傳基因特性等，有了更深一層的瞭解。根據目前研究所知，病害生物防治的主要機制過程，大略可被區分為 (i) 抗生作用 (ii) 競生作用 (iii) 微寄生作用 (iv) 分解酵素作用 (v) 誘引作物產生抗性等。至於每一種拮抗微生物對病原菌的有效作用，則可能含著上述一種以上的作用過程。

關鍵詞：生物防治、抗生作用、競生作用、微寄生作用、分解酵素作用、誘引作物產生抗性