period in controlled environment growth chambers. Percentages of plants which survived the test were determined and surviving bentgrass plants were returned to the plant breeder.

Methodology also was developed to conduct similar screening studies on vegetatively propagated bentgrasses. This research revealed the potential for further complexities to exist in the etiology of take-all patch of bentgrasses, which was thought at that time to be caused by *Gaeumannomyces graminis* var. *avenae*. In New York, it was demonstrated that *Phialophora graminicola* caused a hot weather form of take-all patch on bentgrasses, and this was confirmed during the development of disease screening methods for this project. These initial findings had relevance to the likelihood that certain disease management strategies would be ineffective during the summer months. Unfortunately, the project was terminated prematurely because the principal investigator moved to another university.

North Carolina State University - Dr. Leon T. Lucas

Spring Dead Spot Disease

The project on spring dead spot of bermudagrass was completed in the fall of 1987. Fungi were isolated from bermudagrass with spring dead spot symptoms throughout this study. Selected isolates of the fungi were used to inoculate bermudagrass in the greenhouse. The inoculated pots were exposed to outside winter conditions during January to May, and spring dead spot symptoms developed with two of the isolates used. The symptoms produced were typical of spring dead spot symptoms on golf course fairways. The fungus that caused the disease was identified as *Gaeumannomyces graminis*, and was the first report of this fungus being associated with spring dead spot of bermudagrass. The fungus was identified on the inoculated plants and from spring dead spot samples collected in May throughout North Carolina and Alabama.

Fungicides and fertilizer treatments were evaluated at four locations in the southeastern United States for the control of spring dead spot. Rubigan applied in September (1 oz. of product per 1000 square feet) and Tersan 1991 (8 oz. of product per 1000 square feet) applied in November were fungicides that gave the best control. Cold hardiness of bermudagrass following treatments with fungicides was evaluated in a study at Raleigh, North Carolina. Plugs of turf that were treated with Tersan 1991 in the fall survived cold temperatures better than other treatments.

Mississippi State University - Dr. J. V. Krans

Refinement of the Host-Pathogen Interaction System

The Host-Pathogen Interaction System (HPIS) is an *in vitro* cell selection system developed in conjunction with efforts to obtain creeping bentgrass with resistance to *Rhizoctonia solani*. The HPIS is a unique cell selection technique which permits the simultaneous transfer of various substances from a disease organism to a callus culture during concurrent growth, yet which avoids direct physical contact between the organisms. The assembly and application of HPIS evolved through a series of experiments dating back to 1988.

Isolates from the USGA culture collection of *Rhizoctonia* spp. (courtesy of Dr. Phil Colbaugh, Texas A&M University), were co-cultured (concurrently grown) with creeping bentgrass callus in the HPIS. The pathogenic isolates inhibited callus growth and development, whereas the non-pathogenic isolates had no effect on callus viability. Studies were conducted to determine effects of various tissue culture media on vigor and pathogenicity of *R. solani*, primarily hormones and energy source concentrations. Various HPIS cultural studies were conducted, focusing on the length of incubation, duration of concurrent growth-interactions, establishing cultural practices for calli following co-culturing in the HPIS, and examining the persistence of toxicity within the HPIS plates.

Some important questions pertaining to HPIS protocol were answered by these refinement studies: 1) pathogenicity at the whole plant level is similar to pathogenicity at the cellular level; 2) media components, especially growth hormones and energy sources, play an important role in the pathogenic expression of *R. solani* in the HPIS; and 3) the use of HPIS can be maximized with successive co-cultures.

Recent research efforts have focused on using HPIS to obtain creeping bentgrass germplasm with enhanced resistance to *Rhizoctonia solani*, as well as developing an *in vitro* screening technique to verify enhanced resistance at the plantlet level.

Two co-culture procedures, simultaneous and delayed, were evaluated for obtaining bentgrass callus with resistance to *R. solani*. The simultaneous co-culture procedure was designed to allow the callus a gradual exposure to the toxic substances of *R. solani* over a period of 10 days, whereas the delayed co-culture procedure exposed

the callus to various concentrations of the toxic substances for only 24 hours.

The results from both procedures indicate *R. solani* must be actively growing in the HPIS for at least 7 days before the level of toxic substances is such that only 25 percent of the viable callus population can be recovered. From that 25 percent viable callus population, an average of two plantlets are regenerated. Some of these plantlets display enhanced resistance to *R. solani.*

A special HPIS chamber was developed for screening the germplasm obtained from the HPIS refinement experiments. This system is similar to the HPIS in principle, but is adapted to allow unrestricted growth of the plantlets. The bottom compartment of the chamber consists of the actively growing *R. solani.* The top compartment has been modified by the addition of a 9.5 cm (high) by 9.0 cm (diameter) glass cylinder. This expended space in the upper compartment permits the use of additional growth medium required by larger plantlets, and provides adequate 'head space' which plantlets require for optimum development.

The plantlets were screened in the HPIS chamber for two weeks. Thirty-three percent of the plantlets exposed to *R. solani* died. The surviving plantlets were extremely stressed, displaying purple leaves and stunted growth. They were then transferred to tissue culture boxes where vigorous shoot and root development occurred. The plantlets subsequently have been transferred to soil and will be screened for resistance to *R. solani* at the whole plant level. This will provide critical and much needed evidence on the efficacy of the HPIS approach, as well as providing plants with enhanced resistance to *R. solani.*

Ohio State University - Dr. William W. Shane and Dr. Stephen T. Nameth

Monoclonal Antibodies for Rapid Diagnosis of Summer Patch and Necrotic Ring Spot Diseases of Turfgrasses

Slow-growing patch diseases are among the most difficult problems to diagnose on turfgrasses. This project focused on the development and use of immunological techniques for rapid diagnosis. A monoclonal antibody-producing clone, selective for necrotic ring spot (*Leptosphaeria korrae*), was produced. The antibody, a small protein that can bind to the fungus, can be grown in great quantity within a laboratory flask. The antibody was highly reactive against all fungal strains of *Leptosphaeria korrae* tested.

The usefulness of the antibody for *L. korrae* was tested thoroughly against diseased turfgrass samples collected thoughout the United States or submitted to the Ohio State University Plant and Pest Diagnostic Clinic. The *L. korrae* pathogen was successfully isolated from all Kentucky bluegrass samples exhibiting a significant reaction with the LK antibody. In addition, the LK antibody was successfully used to study the distribution of *L. korrae* in the various regions of "frog eye" patches, and on individual turfgrass plant parts to gain a better understanding of the life cycle of this disease. Through this research effort, sampling techniques for the detection of *L. korrae* with the LK antibody were optimized.

In addition, the LK antibody successfully detected *Leptosphaeria korrae* from certain bermudagrass sites with spring dead spot symptoms. Therefore, the antibody could be useful in determining the causal agent of spring dead spot. Currently, at least three fungi (*L. korrae*, *Ophiosphaerella herpotricha,* and *Gaeumannomyces graminis*) have been shown to be cause this disease. Despite the successes associated with the LK clone, no commercial company followed through with formal licensing of the technology.

Development of a monoclonal antibody for summer patch (*Magnaporthe poae*) was not completed. Difficulties occurred with the toxicity of the pathogen to immunized mice and rabbits. Reactivity of the mouse serum which was produced did not adequately select *M. poae* from diseased turfgrasses. Unfortunately, the project was terminated early when the principal investigator left Ohio State University for another position in industry.

Soil Compaction

Michigan State University - Dr. Paul E. Rieke

Hollow and Solid Tine Cultivation Effects on Soil Structure and Turfgrass Root Growth

Hollow and solid tine cultivation effects, as influenced by soil compaction and moisture content during cultivation, were evaluated on the basis of soil structural properties and root growth.

As expected, compaction resulted in pronounced detrimental effects on soil structure and root growth. Both cultivation methods resulted in positive and negative effects on soil structure. Cultivation increased the amount of large soil pores, with hollow tine coring being the most effective in producing this response. Regardless of