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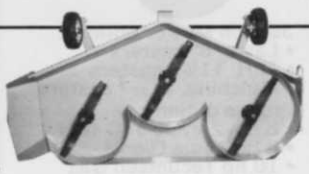

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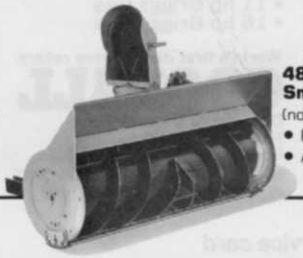

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use. Each had its own rate of nitrogen release, and each had its own set of requirements for release.

New types of turf maintenance equipment began to make their way into field use. Each made its own contribution to the enhancement of grass growth and each placed its own forms of stress on the plants.

Each of these innovations added to the latitude of selectivity within the field of turfgrass culture for producing particular types of turf. However, they also presented to the turfgrass pathologist extremely complex patterns of mobility where the expression of disease was concerned.

The basis for the pathologist's capacity to respond to the newly developing pattern of turfgrass culture also came into being during this decade. The English translation of Principles of Plant Infection by Ernst Gaumann was printed in 1950. This action gave a much broader exposure within the scientific community to what was unquestionably the most significant publication in plant pathology in the first half of the twentieth century.

The principles put forth by Gaumann in his book established the mentality by which the dynamics of disease development could be viewed with equal clarity at both the reductionist and constructionist levels. They permitted the development of a basic concept of disease that was truly functional in all circumstances. It was a concept that transcended such previously limiting factors as the nature of the incitant and/or the magnitude of the plant's response. The groundwork laid by the nineteenth century concepts, and built upon by the emphasis placed in the research of the first half of this century on determining the nature of the response of the suscept, now found full expression in the concept of disease proneness.

Disease proneness views each plant as being genetically endued with its own innate capacity to become diseased. Expression of the various facets of this proneness is made manifest when the appropriate combinations of the physical environments are brought into being. Through this concept, disease is seen in its absolute reductionist sense as simply the moment of the initiation of aberrent metabolism, and in its absolute constructionist sense it is seen as the moment of expression of the most salient features of its clinical symptoms.

Both of these moments of disease, and the acceptance of the legitimacy of their being, establishes the means by which all of the factors relating to the ultimate outcome of the pathogen-suscept interactions can by given proper perspective. Disease proneness, then, becomes the route to a truly holistic view of disease. Within the concept of disease proneness, for example, the causality of disease is seen as a matrix of events rather than a single episode. This means that the determination of the etiology of a disease is more than a search for a single entity. Rather, the objective of research on disease etiology is to determine the order of occurence of these events and how they interrelate in both the initiation of the process of disease and in the fostering of the development of its clinical phase.

The opportunity to apply these concepts to research on a turfgrass disease of unknown etiology came during the first years of the following decade. In 1959, a previously undescribed disease of Kentucky bluegrass characterized in

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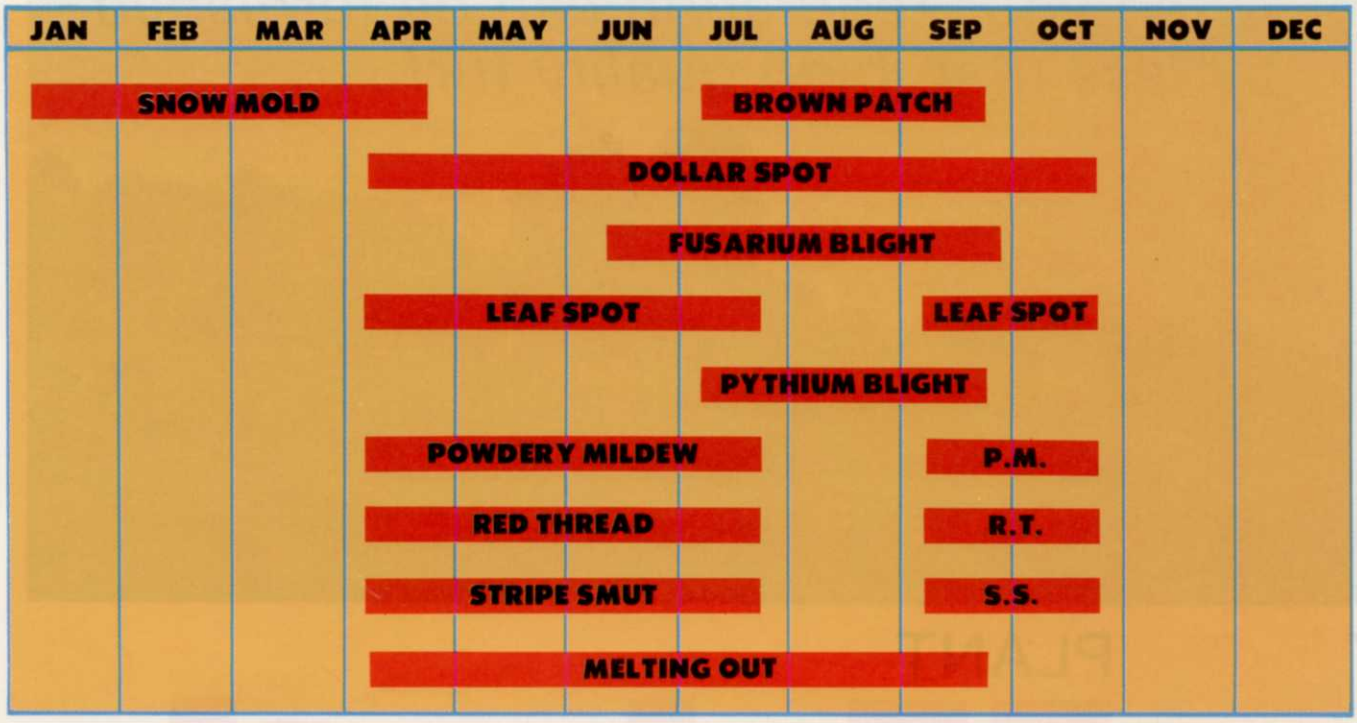
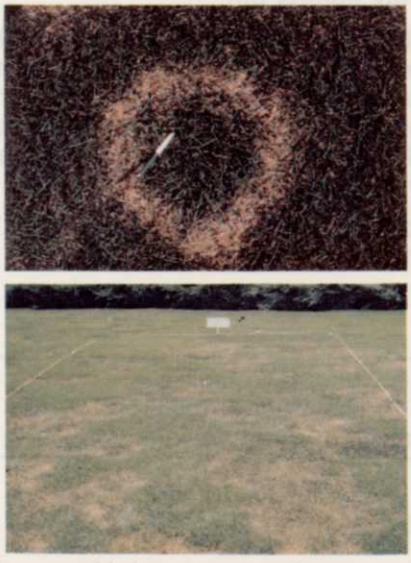


its final stages of development by more or less circular patches of blighted grass 0.6-1 meter in diameter was observed in southeastern Pennsylvania. During 1960, 1961 and 1963, the disease became epiphytotic in stands of Kentucky bluegrass and creeping bentgrass in the south central and eastern parts of the state. Also, during this time, the malady was observed on a wide range of cultivars of bluegrass, as well as creeping bentgrass and creeping red fescue in Ohio, New York, New Jersey, Delaware, Maryland and the District of Columbia.

The experiments for the purpose of determining the etiology of the disease were designed to take the candidates through series of multiple factoral tests for levels of pathogenicity. The factors in the respective experiments included variations in (i) air temperatures, (ii) nutritional levels of the test plants, (iii) test plant geneotype, (iv) isolates of the same species of pathogen candidate, and (v) levels of propagule density of the same isolate of pathogen candidate. Isolates taken over a 5 year period were subjected to these series of tests. It was found that the biotic components of the etiology of this disease were *Fusarium roseum* f. sp. *cerealis* 'Culmorum' and *Fusarium tricinctum* f. sp. *poae*, and that these entities were able to function in a primary capacity — both in infection and in colonization of the suscept tissue. In addition, it was found that the degree of resistance to colonization is influenced by the nutrition of the suscept. Also, the level of resistance within *Poa pratensis* was found to be determined by an interaction of suscept genotype, isolate in question of the pathogen species, and air temperature. Thus, the complexity of this particular disease syndrome, as established by the variables of culture to which the various species of grass were being subjected, was accommodated in the search for the causality of the disease in question by utilization of this newly broadened concept of etiology.

It was during the early part of the decade of the 1950's that a full appreciation of the potential of parasitic nematodes as turfgrass pathogens was developed. Tests for the purpose of determination of population levels of ec-



Fusarium blight *is most often characterized by a circular area of reddish-brown grass with green grass in the center.*

toparasitic forms in the root zones of turfgrasses soon became commonplace. Midway through this decade, post planting nematicides were being included as regular entries in the lists of plant protectants used in many turf management programs.

The close of the 1950's and the beginning of the 1960's was also the time period in which the first books on the nature and control of turfgrass diseases were published. In 1959, the British Sports Turf Research Institute issued FUNGAL DISEASES OF TURFGRASSES by J. D. Smith. Three years later, DISEASES OF TURFGRASSES by H. B. Couch was printed. Smith's book covered the more important diseases of turfgrasses in Great Britain. It went into its second edition in 1965. The book by Couch was a treatment of all known turfgrass diseases, and its second edition was released in 1973.

Throughout the 1950's, and on into the following decade, there was a sharp increase in the frequency of introduction of new fungicides for use in the field control of turfgrass diseases. By 1964, this rapid influx of new pesticides had slowed considerably.

It was in 1963 that the existence of resistance to anilizine by *Sclerotinia homoeocarpa* was reported. This was the first verified instance on the part of a turfgrass pathogen of the development of resistance in the field to a pesticide. Within a few years, episodes of both anilizine resistant and cadmium resistant *Sclerotinia homoeocarpa* had been reported from several locations in central and northeastern

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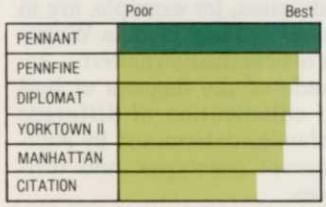
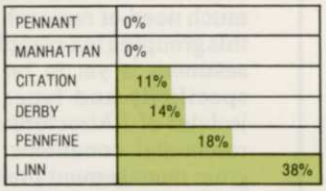
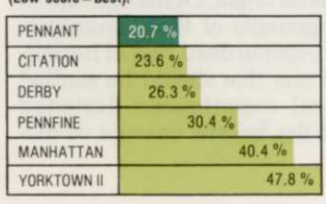
Warm and cool season average turf score, Southern California first-year tests, 1979.



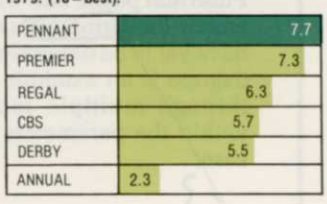
Percent winter injury. Adelphia, N.J. March 1978. (10 of 26 varieties tested showed no significant injury.)



Pennant was best among 12 varieties tested for red thread disease (Corticium fuciforme) in 1-year average, 1979-80. Western WA. (Low score = Best).

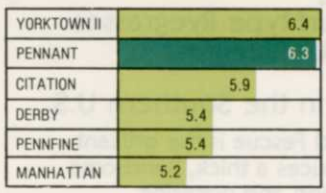


Average Turf Performance scores, February 1980. Southern Arizona turf overseeding on Tifgreen Bermuda. Sixteen entries seeded October 1979. (10 = Best).

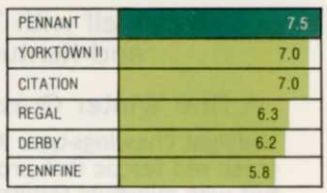


*Plant variety protection pending and anticipated

Turf performance scores. North Brunswick, N.J. 1975-78. (9 = Best).



Reaction to brown patch disease (Rhizoctonia), Adelphia, N.J., 1978. (9 = least damage)



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United States and southeastern Canada.

By the end of this decade, the expressions of public concern over the possible harmful side effects of pesticides on the quality of the environment began to be felt in turfgrass disease control programs. In 1970, the manufacturer of Semesan voluntarily removed the product from the market. The use life of this material as a mainstay in turfgrass disease control programs had spanned almost five decades. It had served well in the control of a broad spectrum of important diseases, and its departure was lamented by many.

The first commercially available systemic fungicides for use on turfgrass were marketed in 1970- 71. These were benomyl and thiabendazole. Later investigation of the interactions of various aspects of the physical environment and certain practices in turfgrass culture on the effects of these materials on both the incidence and severity of target and non-target diseases, as well as the growth patterns of the suscepts, pointed to the need for the establishment of more precisely defined parameters for the field testing of systemic fungicides. Also, within a few years, instances were being reported of resistance in the field of *Erysiphe graminis* and *Sclerotinia homoeocarpa* to members of this benzimidazole grouping. These observations served as an additional impetus to the development of specific guidelines for field use of systemic fungicides in turfgrass culture.

The Future

In the future, the design of major research efforts in turfgrass pathology will become more closely oriented with the concepts of holopathology. Turfgrass culture is unusually well suited for the development of research models based on the holistic view of disease. The wide range of suscept genotypes that have been developed within the various turfgrass species provides a broad array of potential responses to various environmental stresses. The equally wide range of growing conditions to which the plants are systematically subjected establishes the vehicle through which these innate abilities can be brought into full expression. Holopathology is the vehicle by which this matrix of events can be described, and their relative degrees of interdependence and individual roles in the initiation and fostering of the disease process can be defined.

In addition to continued work with Fusarium blight, there are several other known turfgrass diseases that need to be subjected immediately to research that has been designed within the concept of holopathology. The Rhizoctonia-incited diseases, for example, are in much need of research based on these models. Within this grouping is a complex of colonization patterns. Assessment is yet to be made of the degrees of host specificity and types of colonization of different isolates of *Rhizoctonia solani* as determined by environmental conditions, suscept genotypes, and turfgrass management practices.

This same research approach needs to be applied to the Pythium-incited diseases, Sclerotinia dollar spot, Fusarium patch, and Typhula blight. Of the diseases in which the principle components of their causalities have yet to be determined, spring dead spot of Bermudagrass is an example of one that should be studied through multiple factoral experiments designed within the parameters of the holistic concept of disease.

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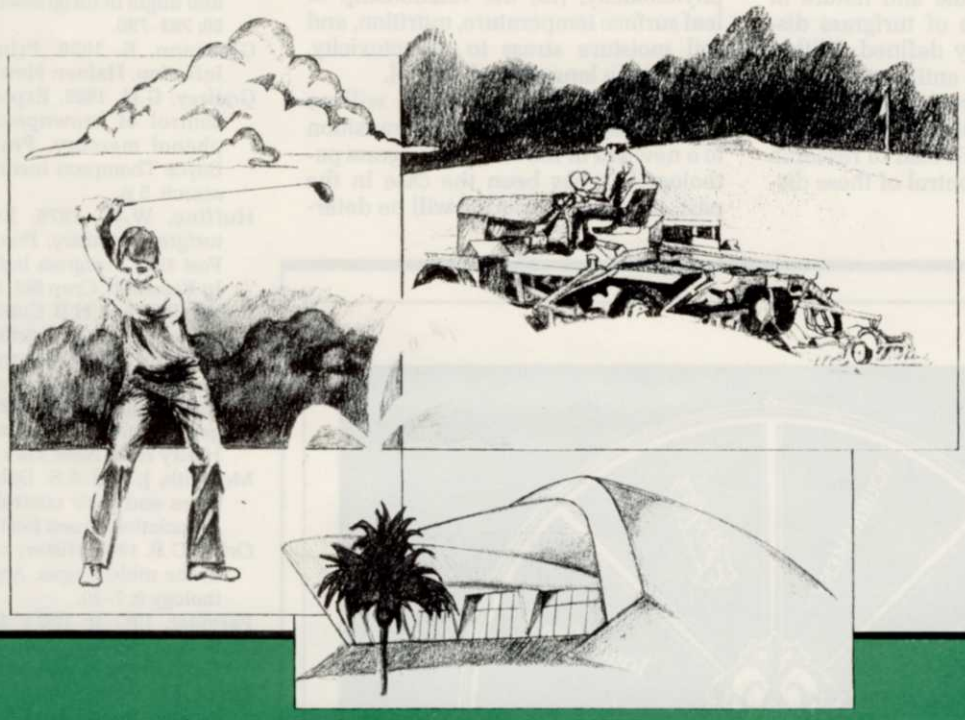

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In the near future, the basic principles that have been established in the field of epidemiology will begin to be utilized more widely in research on forecasting the outbreaks of turfgrass diseases. The evident benefits of being able to time applications of fungicides on a disease preventive schedule that has been determined by a system of objective analyses of the physical and biological environments will foster increased research in this area.

The view of the spectrum of entities with pathogenic potential to turfgrasses will continue to expand in the years ahead. Additional viruses and mycoplasm-like organisms that are pathogenic to turfgrass will be included in this list. Also, the role and nature of bacteria as incitants of turfgrass diseases will be clearly defined. Within the realm of abiotic entities, the presently increasing appreciation of the importance of air pollutants as incitants of turfgrass diseases will lead to research on the nature and control of these disorders.

In the area of turfgrass disease control, the development of cultivars will include screening techniques that are based on the presently increasing knowledge of the need to identify the degree of stability of the suscept genotype to nutrition-induced changes in disease susceptibility. Research on the chemical control of turfgrass diseases will become more sophisticated. Techniques of pesticide application will be receiving more attention than has been given to this area in the past. Also tests for the field screening of systemic fungicides and nematicides will include such parameters as (i) possible increase in incidence of non target diseases, (ii) the possibility of latent phytotoxicity, (iii) the relationship of leaf surface temperature, nutrition, and soil moisture stress to phototoxicity, and (iv) the longevity of control.

In the distant future, there will no doubt come another time of transition to a new era in the field of turfgrass pathology. As has been the case in the past, however, its timing will be determined by the nature of the changes in the techniques and procedures of turfgrass culture and the development of principles and concepts in the science of plant pathology that are applicable to the solution of the new expressions of disease that they will have fostered.

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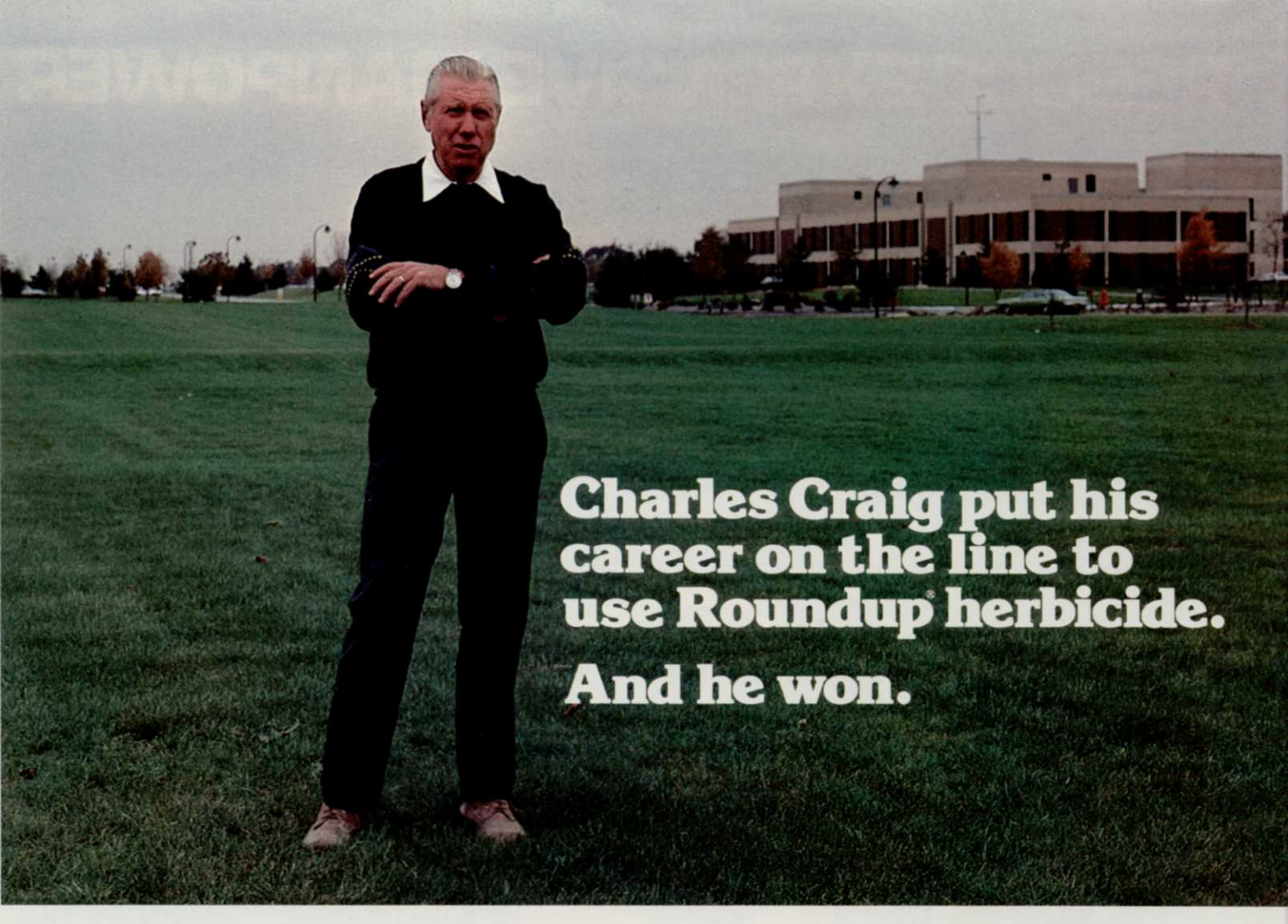

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