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Lignasan is labelled for two rates of application: preventative and therapeutic, the latter at twice the preventative dosage. Research indicates that the higher therapeutic dosage gives better distribution and a higher level of disease protection; increases beyond that do not appear to give added protection. Although some studies indicate that the therapeutic dosage will protect the tree for two years or more, the current recommendation is to inject annually.

Negative aspects of the above process include the need for repeated applications, which not only damages the pocketbook but also may lead to expanding injury to the tree; the somewhat complicated and time-consuming procedure involved, particularly when treating a large number of trees; the need to inject during a rather short time in the spring, which is next to impossible in a large population; the necessity, rarely realized, to achieve complete distribution in the tree; and the fact that some strains of DED appear to be resistant to Lignasan.

More research is needed on techniques for achieving optimal distribution of the fungicide in the tree, on alternative methods which will reduce the severity of tree wounding, and on the chemicals themselves, to make them sufficiently toxic to the specific fungus yet safe to the tree and other organisms, and to provide a period of protection longer than one year.

Lignasan (or similar products variously marketed as Elmosan, Ulmasan, Noculate, Arboreal Fungicide, Correx, Elmpro, and Arbotect 20S) certainly carries no guarantee for DED prevention, much less cure. Research and use in the field indicate it can, if properly applied, lower the statistical incidence of DED infection. In some cases, particularly in smaller trees, it can stop the progress of DED when infection is minimal (five percent or less of the crown) and when injection is followed in ten days by sanitary pruning. When infection has spread beyond a small isolated branch, or when the disease is contracted through root graft, or when the tree is large, a cure is highly unlikely.

Certainly these fungicides should be considered in a strong control program—but effectiveness and cost are considerations too. Perhaps its best use is as a substitute for broadcast spraying in individuals or small populations of highest priority. Lignasan can be a preventative, sometimes a cure, but unfortunately it is no miracle. And it is most emphatically not a substitute for good sanitation.

Other Treatments. Like cancer and its many miracle cures, the DED epidemic has spawned many strange recommendations, from painting the elm trunk with used motor oil to pounding galvanized nails at intervals around the circumference, and from vascular injections of antibiotics to vascular injections of turpentine. A large compost pit dug at the base of the elm and filled with worms is perhaps the most earthy. None of these has been shown to be effective.

Fertilizer application has been recommended occasionally in an effort to strengthen the tree, but recent studies indicate excessive fertilization may actually aid the disease by enlarging the vessel size, thereby making the tree more susceptible rather than more resistant to DED.

Breeding. The search for an American elm resistant to DED, and attempts to create a hybrid elm possessing

both resistance and the many excellent growth qualities of the American elm, have been going on for forty years. These efforts have just begun producing elms for the market.

A small number of American elms which show above-average DED resistance have been located or selected. Attempts to breed or clone them give hope of preserving the pure species. One notable example among several—and indicative of the time needed and problems encountered—is the research at Cornell University. Since the early 1930's they have been running trials on 21,000 American elm seedlings, periodically infecting each individual with an inoculum of the fungus, and culling those which fail the test. After approximately forty years of testing, sixteen trees remained in 1974. Each has been inoculated with a variety of fungal strains at least seven times in five separate years, including three successive years. In each case inoculation initially led to DED symptoms, but this was followed by remission of symptoms. Quite simply, the elms resist the disease. The mechanism of resistance was attributed to compartmentalization of the infection and to subnormal dimensions of the wood vessels. The resistant trees typically had slower growth rates than normal.

These sixteen elms have been closed, bred with each other, and bred with resistant elms from studies elsewhere. Unfortunately—and oddly—neither progeny nor clones appear to have the resistance of the parents. It is hypothesized that longer studies of the second generation are needed, coupled with less severe early culling—that resistance may increase over time in the seedlings.

Given the remarkably high susceptibility of the American elm, the large variation in the fungus, and the decades needed for selection in large woody plants, these less-than-ideal results are not surprising. Efforts continue, and the hope remains that the selection program will, quite literally, bear fruit.

Development of a resistant hybrid (American elm crossed with a more resistant elm species) is more promising at this time. Many crosses have been and are being made, with particular emphasis on hybridizing the American and Siberian elms (*U. americana* X *U. pumila*). None has been released, but researchers are guardedly optimistic.

The most widely publicized hybrid has been the 'Urban Elm,' developed over the last 25 years and made available to the public in the late 1970's. Actually 'Urban' has no American elm sap in it, being rather a cross between the Siberian elm and a Netherlands hybrid. *U. pumils* X (*U. hollandia* var. *vegetata* X *U. carpinifolia*). Reports indicate it will be a good city tree—fast-growing, hardy, resistant to pollution and DED, and adaptable to many soils and climates. It is not, of course, *Ulmus american*; in comparison to the American elm, 'Urban' is shorter and has upright branches, lacking the classic highly prized umbrella shape.

New elms for landscaping and parks are being developed, but it is definitely a long-term project, taking decades. Thus far, the ideal combination of DED resistance and American elm growth and habit has not been found. And in a real sense it is a last-ditch effort, predicated on the continued decimation of the current elm population. Indeed, it is painful to replace a massive, century-old elm with a one-inch sapling.

Nonetheless, this approach may prove over the long term to be the most valuable, given the efficacy of DED and the unreliability of all current treatments.

Current Recommendations

In 1936 *The Garden Dictionary*, a massive compendium of information on cultivated plants, stated in reference to elms and DED: "Prompt eradication and destruction of affected trees is the only known method of control." Today, more than forty years later, thorough sanitation remains the most safe and effective preventative yet developed. To achieve additional protection for selected trees, rigorous sanitation can be used in combination with other of the treatments noted above—most preferably, systemic fungicides.

The soundest and safest DED prevention program at this time would include the following practices:

1. Periodic *pruning* of natural dieback, and removal of unhealthy, injured, or weak elms, in order to destroy preferred beetle breeding sites. This should be done every four to six years.

2. Frequent (preferably weekly) *inspections* of area elms for symptoms of DED during the growing season.

3. Annual spring root or root-flare *injection* of Lignasan (or similar product) at therapeutic dosage, per label instructions.

4. Immediate *sampling* for presence of *Ceratocystis* fungus upon noting symptoms of DED. Many communities have their own testing centers; if not, state universities or county extension facilities are available. You can perform the tests yourself at little outlay of money and with little training.

5. Upon confirmation of DED, immediate *injection* of Lignasan at therapeutic dosage, per label. However, if symptoms are noted in more than five percent of the tree crown, or if the entry of DED is via root graft, immediate isolation, removal, and destruction of the tree is indicated.

6. Immediate *isolation* of the diseased tree by cutting all possible root grafts between the infected elm and neighboring elms. The recommended method for severing such grafts is trenching to a depth of two feet in the area of root overlap. Careful and judicious use of a chemical soil fumigant is an alternative to trenching.

7. Ten days following therapeutic injection and root isolation, *pruning* of diseased branches. Studies indicate this should be severe, ten to fifteen feet beyond the signs of infection (wilting and yellowing of leaves, dark staining of wood.) All tools used in pruning or removal should be cleaned in alcohol after use.

8. Immediate *removal* and destruction of the elm if, despite the above efforts, DED persists and progresses through the tree. If symptoms are present in more than five percent of the crown, or if symptoms indicate root-graft infection, remove. Again, tools should be thoroughly cleaned after use. If the tree cannot be quickly removed, chemical debarking by cacodylic acid is indicated.

9. In the midst of treatment and removal, consider anticipatory *replacement*. Designing a new landscape plan and beginning to implement it, assuming an elm disaster, will ensure a smooth transition to established, growing trees rather than the possible sudden shock of a vast wasteland. Needless to say, use a variety of species, with none comprising more than ten percent of the total.

10. Along with all the above, and of equal importance, *educating* your neighbors and the public. Local and state governments must have strict regulations for removal and destruction of diseased elms, and the regulations must be enforced. The most preferable method of enforcement is knowledge: people must understand the personal and community value of sanitation and the high cost of failure to practice it. Information on DED and on regulations must be distributed and re-distributed. Any number of community projects will further this effort (e.g., incentive programs, clean-up days, free detection labs).

This complete program—excepting Steps 3 and 5 (Lignasan)—should be adhered to for each elm under your jurisdiction. Lignasan injection, if that is determined to be an option, should be reserved for selected individual trees: the elms must be ranked in order of importance and value (aesthetic, historical, age, health, location, etc.), and then grouped into treatment classes—highly valued elms receiving preventative and/or therapeutic injections, and elms of lower value receiving weekly inspection and, upon infection, prompt pruning, isolation and removal. Accurate and detailed records should be kept on all elms in the grounds manager's domain: a number or code for each tree; data on location, size, inspections, prunings, injections, and other treatments; dates of DED detection, trenching or Vapam use, tree and stump removal, site repair, etc.; and costs of all the above. This is not overly time-consuming and is a necessary part of the battle.

And the battle does go on. It has been estimated that by 1930, 77 million American elms had been planted in urban areas; of these, approximately 30 million remain today. On the negative side, after almost fifty years of research, there is no absolutely reliable preventative or cure or satisfactory replacement. However, room for optimism remains. The total US elm population may approach one billion. New treatments are being tested continually, from systemic fungicides to more natural biological controls. Plant breeders strive for a statuesque but disease-resistant American elm variety or hybrid. Meanwhile, the urban areas which have practiced rigorous sanitation have shown remarkably low mortality—low enough to suggest many more decades beneath the shade of the mighty American elm. Even in New England, where the disease has been present for fifty years, many elms survive: as the elm population is reduced, the vector population is reduced and the rate of new infections decreases. Extinction is definitely not in the immediate future of *Ulmus Americana*.

To return to an equally famous "extinction" case cited earlier, the American chestnut may stage a comeback in the near future. Long considered exterminated as a species following importation of the chestnut blight, recent field surveys have located one hundred mature (flowering) individuals in scattered portions in New York alone. In addition, a new strain of fungus has developed, not only less virulent but capable of neutralizing the virulence of the original strain. Not only will a chestnut tree survive and grow when infected with the new strain, but the new strain actually replaces the virulent strain in a previously infected tree. Thus, the outlook for the return of the American chestnut is encouraging.

Let's not give up on the American elm just yet.

WTT

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TURF MANAGEMENT SERIES
PART 5

A black and white photograph showing three individuals, likely maintenance workers, standing on a golf course. They are wearing light-colored shirts and dark overalls. Each person is operating a large, walk-behind turf-cutting machine, which is designed to lift and cut sods of turf from the ground. The machines have long handles and large, circular cutting heads. The background shows a grassy field with some trees in the distance. The overall scene is a typical view of turf cutting on a golf course.

**Turfgrass
Pathology**

Turfgrass Pathology

Internationally recognized turfgrass pathologist Houston B. Couch of Virginia Polytechnic Institute, Blacksburg, takes charge of the pen for this part of the Turf Management Series. Dr. Couch wrote the following history for the Turfgrass Disease Symposium held in Columbus, Ohio, in 1979. The proceedings of the seminar will soon be available in book form from Harcourt Brace Jovanovich, Inc., Book Department, One East First Street, Duluth, Minnesota 55802.

Disease on fine turf has been a major problem since the late 19th Century. Piper and Oakley broadly termed most symptoms as "Brown Patch." Monteith took this information and refined it further. Today, the identity of many diseases is still less than exact. There are arguments over terminology and nomenclature. But much more is known and a great portion of damage by turf disease has been prevented through resistant turfgrass cultivars and maintenance practices.

Knowledge of turfgrass disease will play a vital role in integrated pest management in the future. Relationships between disease and maintenance practices will be clarified. Effects of herbicides, aerification, soil pH, insects, and traffic on turfgrass will be better understood.

Certainly, a basic level of information on turfgrass pathology is vital for the manager of any fine turf area.



Houston B. Couch

Bruce F. Shank

Bruce F. Shank, Editor

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PAST, PRESENT & FUTURE

In its fullest sense, turfgrass pathology is an integration of the concepts and principles of the science of plant pathology with those of the practice of turfgrass culture. Consequently, the level of understanding of the nature and control of turfgrass diseases at any point in time is a direct reflection of the extent of knowledge in these two areas of activity, and the degree of skill that has been employed in bringing this information together.

The Past

In Europe, lawns of pure stands of grass were first purposely established in the thirteenth century. It was during this time that the game of "bowls" became popular. The original bowling green was the forerunner of the modern golf course green. Near the close of this century, "club ball", an early form of crickets, came into being.

By the sixteenth and seventeenth centuries, gardens had become more elaborate. Also, more care was being taken in the establishment of bowling greens. In Northern Europe, lawns had become fairly common features of home grounds and village squares. Most towns had a turfed "common" or "green". A form of soccer was being played on these public greens. The height of the grass in these areas was maintained at a low level by the grazing of sheep and goats.

During this same time period, the concepts and practices related to the development and control of diseases of plants were also in a primitive state. The autogenic concept of disease was the order of the day. This was an outgrowth of the theory of spontaneous generation. Its view of disease causality held that maladies of plants were due to internal disturbances, and that the fungi found in association with these disorders were the product, not the cause, of the diseases in question.

Certain inventions and discoveries of this period contributed materially to efforts directed toward mounting a



Dusting a putting green in 1922 with Bordeaux mixture for disease control. This was the first fungicide in general use for controlling turfgrass diseases on golf courses.

successful challenge to the autogenic concept of disease causality. Among these was the development of the compound microscope in 1590. Improvements were made in the microscope in 1665, and there followed within the next 25 years a series of studies that laid the foundations needed for the progressive and systematic study of plant anatomy and the establishment of the science of microbiology.

With the advent of the eighteenth century, specific biological evidence for the disproof of autogenesis began to accumulate at a more rapid pace. In 1705, the view was expressed that fungi reproduced by developing spores. As the century progressed, the concept that fungi are autonomous organisms was reinforced by a succession of studies and observations. By 1785, the evidence that they were indeed distinct biotic entities in their own right had been well established.

The information that had been gained in the eighteenth century relative to the nature of fungi found direct application in the development of an expanded concept of disease in the nineteenth century. In 1807, the first report giving clear evidence that fungus spores could germinate and infect a plant was published. With this, the allogenic view of disease causality was given a firm, scientific base. Allogenesis perceives disease as being engen-

dered by forces from without the plant, rather than from within. Through its applications, research on the nature and control of plant disease was placed on the proper course. While it would still be some 50 years before the total weight of evidence in support of this concept would finally reduce the voices of the advocates of autogenesis to a faint whisper in the scientific community, the stage had now been set for the development of the science of plant pathology.

During the eighteenth century, turf maintenance became more sophisticated. Instructions for the proper care of grass walks and bowling greens called for them to be rolled and mowed every 15 days. Many of the gardening books of this period contained instructions on the mowing, rolling, edging and weeding of lawns.

A single event of this century that had a significant effect on the promotion of the development of the art of turfgrass culture was the establishment of the Royal and Ancient Golf Club of St. Andrews in Scotland in 1754. With this, the game that was to become universally known as "golf" received recognition as an established, on-going sport. The evolution of golf through the years, and the various requirements it has placed on turf for play, has served as a major impetus for the development of the framework of the basic concepts now used in various aspects of turfgrass culture.

The equipment used in turfgrass culture during this time was borrowed from the farm. Cutting of the grass, for example, was accomplished with hand scythes and cradles. The early part of the nineteenth century brought the invention of the first mowing machine for turf. The device was patented in 1830, and its manufacture began two years later.

The impact this machine had on the development of turfgrass culture as a systematic endeavor in which the

various practices are centered on basic principles was equivalent to that of the establishment of the concept of allo-genesis on the science of plant pathology. The capacity to maintain both specified and uniform heights of cut continuously with rather low investments in labor was the innovation needed in order for the unique features of the turfgrass plant to be fully utilized in a wide range of landscape and utilitarian situations. The motivation to exploit these now-recognized potentials led to the systematic programs of research and testing that have in turn established the various concepts and principles that comprise the art of turfgrass management.

As the nineteenth century progressed, the science of plant pathology developed both form and substance. A continuing series of discoveries firmly reinforced the allogenic concept of disease causality. In 1858, the first book based entirely on this concept was published.

Through the course of the century, the fungal incitants of several of the more important diseases of plants were identified. In addition to fungi, certain species of bacteria came to be recognized as being pathogenic to plants. At the close of the century, research was begun on determining the nature of what was being referred to as a "contagious living fluid". The pathogenic principle of this fluid would later become known as "virus", a previously unknown biotic entity.

It was during the final quarter of the nineteenth century that a major breakthrough in the area of chemical control of plant diseases was made. In 1882, Bordeaux mixture was discovered. With the advent of this very effective, low cost fungicide, the era of systematic research for the purpose of developing programs of plant disease control through the use of pesticides was ushered in.

While these various events were making their contributions to the nurturing of plant pathology into a mature science that would be fully capable of addressing itself to the task of determining the nature and control of disease, turfgrass culture was also becoming more clearly defined—both in the expectations from its efforts and its capacity to respond to these requirements. By the latter part of the nineteenth century, golf had become a very popular sport throughout the British Isles.

The year 1885 stands as a hallmark in the United States for both turfgrass culture and plant pathology. The first

official golf club in the country was established in Yonkers, New York in 1885. This was also the year that turf research started in the United States. The location of this work was the Olcott turf gardens in Connecticut. It was also in 1885 that the United States Department of Agriculture's Division of Botany was established. This unit was to serve as the first administrative base for plant disease research in this country.

By the close of the nineteenth century, there were over 80 golf courses in the United States, and the first games of two other turf-dependent sports, football and baseball, had been played. The United States Golf Association had been formed. Research on turf management was being conducted on a much broader scale, and the nature and control of plant disease was being investigated at many of the state agricultural experiment stations.

As the twentieth century began to unfold, then all of the components needed for the establishment of the field of turfgrass pathology were in place. Many of the basic methods and techniques of turfgrass culture had been defined, and the science of plant pathology had matured to the extent that it could address itself constructively to identifying the causes of specific diseases and developing programs for their control. All that was needed to bring the parts together was a clear and present need. Ideally, this would be a disease capable of combining high incidence with high severity within a short span of time. While we now know of several diseases of turfgrasses that could have functioned well in this capacity, the lot fell to *Rhizoctonia brown patch*.

In 1914, a disease was observed to be causing extensive damage to a turf garden in Philadelphia, Pennsylvania. The owner of the garden, F. W. Taylor, was keenly interested in turfgrass culture and was active in both the development of management techniques and in the search for superior strains of grass. He was particularly interested in bentgrass culture, and his garden in Philadelphia contained several selections he had obtained from the Olcott turf gardens in Connecticut.

In his efforts to determine the cause of the disease at hand, Mr. Taylor secured the assistance of C.D. Piper, a member of the administrative staff of the United States Department of Agriculture and Director of the United States Golf Association Green Section. Isolations from the diseased plants yielded the fungus *Rhizoctonia solani* and it was determined that this organ-

ism was the incitant. Based on its characteristic clinical symptom pattern of foliage blighting and death of plants in irregular patches measuring up to 1 meter in diameter, Taylor assigned the disease the name "brown patch". The climatic conditions in 1915 were again particularly conducive to the development of the disease, and with the experience in diagnosis gained from the previous year, it was determined that brown patch was capable of causing severe damage to bentgrass putting greens.

With the pathogen identified, the symptoms known, and the scope of the disease defined, the next step was to search for a control. In 1917, field tests were begun by the United States Golf Association to determine the feasibility of using Bordeaux mixture for brown patch control. Although it was found that the material had certain limitations due to its toxicity to bentgrass after repeated applications, it was effective in controlling the disease, and there were no alternatives. By 1919, Bordeaux mixture was in general use on golf courses for control of brown patch.

Through this 5 year period, then, the "rest disease" had appeared. The extent of its occurrence had been established, its incitant was identified, and control measures had been worked out. The components had been brought together and they had matched. The practice of turfgrass pathology had begun.

By the end of this decade, another turfgrass disease and its causal agent had been identified. This malady was first recognized on putting greens. Its symptom pattern was somewhat similar to *Rhizoctonia brown patch*, and it occurred at about the same time in the growing season. However, the individual blighted areas of turf were usually lighter in color and smaller in diameter. The two were distinguished from each other in name, then, by referring to the former malady as "large brown patch" and the latter disease as "small brown patch". Small brown patch (or "small patch") eventually became known as "dollar spot", and the pathogen was finally given the name *Sclerotinia homoeocarpa*.

During the 1920's, the clinical symptoms were described, the incitants identified, and the epidemiological patterns worked out for several newly recognized turfgrass diseases. In 1920, mercuric chloride was used successfully in the Chicago, Illinois area for control of *Rhizoctonia brown patch* on bentgrass putting greens. An organic mercury, Semesan (chlorophenol mer-

cury) was tested in 1924 on the putting greens of a golf course near Yonkers, New York, and found to be very effective in the control of *Sclerotinia* dollar spot. By the end of the decade, the inorganic mercury chlorides and Semesan had become the primary fungicides used in the field control of turfgrass diseases.

In 1929, a turfgrass research and advisory service was established in Great Britain. The work was conducted under the auspices of the Board of Greenkeeping Research. The name of the organization was later changed to the British Sports Turf Research Institute. From the outset, the staff addressed itself to the solution of a broad range of problems in turfgrass culture, including determining the nature and control of certain diseases. The papers that have been published on the subject of turfgrass pathology in its journal are a valuable addition to the body of knowledge in this field.

The First Publication

The first comprehensive publication on the nature and control to turfgrass diseases was published in 1932. It was

issued as an entry in the Bulletin of the United States Golf Association under the title **TURF DISEASES AND THEIR CONTROL**. The authors, John Monteith and Arnold S. Dahl, were two of the primary researchers in the field of turfgrass pathology in the late 1920's and early 1930's.

This publication stands as a classic, both for the thorough manner in which it integrates the principles and concepts of plant pathology with those of the practice of turfgrass culture, and the completeness of detail in its descriptions of the nature of many of the more important diseases of turfgrasses. Consideration was given to diseases incited by both biotic and abiotic entities. Control was approached from the standpoint of the use of resistant varieties and cultural methods, as well as through the use of fungicides.

The contribution of **TURF DISEASES AND THEIR CONTROL** to the development of the field of turfgrass pathology was far more reaching than bringing together in one volume a compilation of disease symptoms and control procedures. During this time, in plant pathology teaching and research the strongly pathogen-oriented school

of thought of the nineteenth century was giving way to plant disease concepts centered more directly on the nature of the response of the susceptible. The thinking of this more contemporary view of disease was very skillfully employed in the development of this publication. As the result, in addition to serving as a model for the design of the turfgrass disease research of its time, it also effectively set the stage for moving these investigations toward the holistically-oriented studies of the future.

As the 1930's began, turfgrass disease control programs were almost entirely dependent on either Semesan or the inorganic mercury chlorides. In 1931, however, it was discovered that thiram, an organic compound that had been developed as an accelerator in the manufacture of rubber, had fungicidal properties. Field tests showed that this material was effective in controlling several of the more important diseases of turfgrasses. Within a few years, thiram was in general use in turfgrass disease control programs.

The impact of thiram on turfgrass disease control programming was an interesting one in that it provided a basis for expanding rather than replacing the use of the organic and inorganic mercuries. It was found that when this compound was used as a tank mix with either mercuric chloride or Semesan, in addition to providing its own spectrum of fungicidal activity, it reduced to some extent the phytotoxic potential of the mercuries. As the result, the introduction of thiram established a new dimension in disease control — greater efficiency with less possibility of injury to the grass.

The transition to the present era in turfgrass pathology occurred during the 1950's and early 1960's. This was a time of major and highly innovative developments in both the field of turfgrass culture and the science of plant pathology. The Kentucky bluegrass cultivar 'Merion' was released in 1952. This was the first of what was to be a continuing series of releases of new genotypes of *Poa pratensis*. Within the following two decades, it would be joined with similar series of releases of bentgrasses, fine bladed perennial ryegrasses, tall fescues, Bermuda-grasses, and zoysia. Each cultivar brought with it certain peculiarities of management requirements, and each had its own pattern of response to the various pathogenic entities.

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