New Challenges in Chestnut Research

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ABSTRACT.— Based on past and current chestnut research efforts an important challenge for future chestnut research is to maintain research programs that are realistic and properly designed so that the data can be analyzed. The tasks of reproducing and establishing new chestnut stands or altering natural hardwood stands to allow reintroduction of the chestnut species are monumental.

Almost three-quarters of a century has passed since chestnut blight was first discovered at the Bronx Zoological Park in New York City. The intervening years have seen almost total decimation of one of the most important hardwoods in the eastern deciduous forests. No other single tree species had captured the attention of the American public as did the chestnut during the first two to three decades of this century when the eastern forests were being ravaged by the blight. Historically, chestnut blight is unique in that it is the only known disease which has caused almost total destruction of a tree species. This situation occurred in only a relatively few years after the unfortunate introduction of a foreign plant pest into the country.

If the effects of chestnut blight can be described as unique, so can the efforts of scientists and administrators who were charged at the time with the responsibility of identifying and controlling the disease. Strongly controlled by emotions and prejudice, many early workers maintained a staunch "do or die" war until it became painfully obvious that the blight was uncontrollable. Hepting (1974), in his article on "Death of the American Chestnut," provides an excellent account of the attitudes that existed during the chestnut blight program and how the "surrender" finally came about.

Now, for the third or fourth time after chestnut blight was first reported, we appear to be embarking upon another major attack on this disease. Has the host, or the pest, or the opportunity for control changed sufficiently so that chances of success are great enough to justify this new thrust? The only rational answer to this question is that we simply do not know. Nevertheless, there have been a number of new developments in tree breeding and propagation methodology. Also, for the first time we are beginning to hear about a natural mechanism associated with hypovirulent strains of the fungus that may hold some promise for biological control. Our viewpoint concerning these new observations is still one of "cautious optimism" but the potential for application of the information is great enough to justify another serious look at the blight problem.

The Native Chestnut Population

To assess the probable success of new thrusts in chestnut research it may be enlightening to establish a few hypotheses about some of the changes that likely have occurred in both the host and the pathogen, and to take a look at the direction that new research seems to be heading (Table 1). Past research has usually been directed at either the host or the pathogen, so this provides a logical framework for reviewing some of these changes. A limited amount of work has been done on interactions between the host and pathogen, but, for the most part, it constitutes a relatively minor, but highly important, part of past programs.

Changes in the Host

Natural selection normally favors the more resistant individuals when pressure is exerted by a devastating agent such as a disease. Chestnut should be no exception, so theoretically the surviving population of chestnut should possess a higher level of resistance than the original stand. While we seldom stop to consider just how much resistance might be developing, the possibility of locating resistant clones has much appeal and has been the center of focus for much past research. Unfortunately, to date, no completely resistant clone of native American chestnut has ever been discovered.

The cards are stacked against developing increased genetic resistance to infection because of restricted natural reproduction. In the past, repeated attacks by the fungus have prevented most chestnut sprouts from attaining seed-bearing size. Chestnuts are self-sterile, so if pollen sources are not

Table 1
Probable Changes in Natural Populations of Host and Pathogen Since Outbreak of Chestnut Blight, and Focus of Current Research Programs.

	Castanea denata	*Endothia parasitica*
Selection Pressure	More Resistant	Unknown
Genetic Diversity in the		
Original Population	High	Low
Current Population	Low	High
Opportunity for Extending Genetic Diversity	Low	Moderate
Limiting Factors in Extending Genetic Diversity	Low Seed Production (Self Sterility) Sprouts maintain parental traits	Fewer host plants with high susceptibility
Probable current changes in natural diversity	**Unknown:** Increasing -More trees beginning to bear seed - More resistance developing Decreasing - More susceptible being killed off	**Unknown:** Increasing -Time favoring increased diversity Decreasing - Weaker strains dying off because of lack of host trees
Focus of Research Effort to Solve Blight Problem	**Selection and Propagation** - Vegetative propagation - Cell tissue culture - Tip meristem culture **Hybridization and Mutagenesis** - controlled pollination - irradiation	**Chemical Control** - Lignasan **Biological Control** - Hypovirulence
Probable Opportunity for Success	Low	Low-moderate

available within a few hundred feet, production of viable seeds even from large trees is quite limited. The chances that both parent trees possess exceptional disease resistance traits are almost impossible to calculate. Within the native chestnut population, the lack of resistance to the disease has been transmitted from generation to generation through the sprouts.

Changes in the Pathogen

Very little thought has been given to what might be happening to the original population of the fungus. Because inital infection was somewhat localized at first but spread rapidly from these general locations, it is fairly safe to assume that genetic diversity within the pathogen during the early stages of the blight was fairly narrow. Time has been on the side of the pathogen, and its reproductive processes have proceeded basically unchecked as it produces viable spores on live, as well as on dead woody material. Thus we might expect that the genetic diversity within the natural population of the pathogen would be greater today than when the fungus was introduced. However, it is impossible to estimate the net change that has taken place in the genetic diversity of the pathogen. The discovery of so-called "hypovirulent" strains may be evidence that new populations of the fungus are finally beginning to distinguish themselves. These new strains could hold some promise for use in control measures, but much research is needed to verify this hypothesis.

FOCUS OF CURRENT RESEARCH EFFORTS

Various approaches have been used in attempting to find a satisfactory solution to the chestnut blight problem—some have been with us since the early stages of the blight program, whereas others represent fairly new advances in biological research.

Selection and Propagation

The search for resistant chestnut trees from the native population has been unparalleled. Natural resistance became the focus of efforts very quickly after the outbreak of the blight when it became apparent that no direct control measures, including sanitation, were effective. Well-funded and organized at first, the selection program finally ended in publicized pleas to the public to report the presence

of apparently healthy trees that they found on their properties or during their travels. Thousands of responses were received and still continue today. While time has shown that some of these trees did seem to possess higher degrees of resistance than others, none were ever proven to be completely resistant, and most of the trees eventually succumbed to the disease. One fault of past selection research may be that we spent too much time looking for trees which were completely immune and ignored opportunities to work with semi-resistant clones.

But the question of resistance in the natural population is still not fully resolved. The fact that in both Europe and Asia, members of the chestnut family still coexist with the disease strongly suggests a genetic resistance system exists within the genus. As the selection pressure on the residual chestnut stand continues, the probability of finding a resistant clone also increases. Due to the self-sterility problem, it would not be realistic to count on seed production as a feasible means of reproducing a resistant clone if one could be found. The chances of finding a resistant flowering and a resistant pollen-producing tree, and then transferring this resistance to progeny are extremely limited. To reproduce a resistant clone we would have to depend on some form of asexual propagation. Unfortunately, most large seeded hardwoods have historically been difficult to propagate by vegetative means. Nevertheless, new developments in vegetative propagation of hardwoods have come about within the past decade. Several researchers (Shreve and Miles, 1972; Jaynes, 1974) have shown that by using cuttings of sprout origin collected at the proper time, treated with rooting hormones and grown under carefully controlled moisture-temperature regimes, a high degree of rooting success is possible.

Other forms of vegetative propagation under investigation include cell and tip meristem culture. The science for these techniques is relatively new. The methodology involves removal of small amounts of tissue from selected trees and then with the use of appropriate culture media and closely controlled growing conditions, producing plantlets which carry all of the genetic traits of the parent tree.

In terms of the blight problems, all of the asexual propagation methods are based on the assumption that we have a resistant clone worthy of propagation. As we know, this resistance has never been demonstrated among native American clones. Nevertheless, the techniques have much more far-reaching implications than immediate solution to the blight problem, and this alone justifies further research. If nothing more, then at least asexual propagation of selected hybrid clones can now be given more serious consideration.

Hybridization and Mutagenesis

In the hybridization approach to breeding, American chestnut is most commonly crossed with Chinese (*Castanets mollisima* B1.) or Japanese chestnut (*Castanea crenata* Sieb. & Zucc.) with the eventual goal of developing a forest tree with form and growth characteristics similar to the American chestnut plus the resistance to the blight found in the Asian chestnuts. In reality, hybridization is not difficult since there appears to be little evidence of incompatibility. A number of hybridization programs were initiated and some were quite successful in developing high nut-yielding varieties. Even before the disease was known, a number of crosses were made as early as 1890, and these were followed by a broader program in 1921 by Luther Burbank in California. Within the USDA, chestnut hybridization programs date back to 1894. At the height of the chestnut blight in 1925, Russ Clapper began an extensive chestnut breeding program in the course of which more than 10,000 hybrids were produced and field tested. A second breeding program was started by the Brooklyn Botanic Garden in 1929 and this has been continued to some degree by the Connecticut Agricultural Experiment Station to the present. USDA programs in hybridization of chestnut essentially ended in 1964 with the retirement of Jess Diller. The hybrids that were produced in these programs were not all that promising, and none possessed the vigor, form, and geographic adaptability which characterized the native American chestnut. Nevertheless, the research that accompanied these programs has provided evidence that resistance to the disease is polygenic and that there are certain inheritance linkages which complicate breeding programs.

Another approach to altering the genetic makeup of American chestnut in hopes of inducing disease resistance is irradiation. Ionizing radiation at high dosages has been found to create mutations in a number of horticultural and agronomic plants. Only a few mutations produced by irradiation, however, have ever been found to have desirable external characteristics. Nevertheless the procedure has appeal whenever the need exists to develop new varieties. Recently, Thor (1973) reported that although a number of odd-looking seedlings were developed by irradiation, very few of them survived in the nursery, and none have been discovered with any unusual resistance to chestnut blight.

Chemical Control

It would be almost impossible to list all of the chemicals that have been tested in search of a direct cure for chestnut blight. However, the results of all of these efforts are easy to summarize: no single chemical has ever been found that will completely control the disease. Nevertheless, within the past decade, research on Dutch elm disease has produced a number of chemical measures that appear to be partially effective with chestnut blight. One such chemical, Lignasan, has been found by Jaynes and Van Alfen (1974) to produce signigicant fungi static activity when injected into the bark and branches of chestnuts. Lignasan was most effective when it was

injected into uninfected trees. Partial healing of small cankers was also noted. Researchers at Virginia Polytechnic Institute and State University have also made tests with this chemical. A critical analysis of the potential value of Lignasan, however, would indicate that at this time it is not a practical control for wide-scale use because of need for repeated treatments and high cost. Furthermore, at effective concentrations the chemical tends to be phytotoxic.

Biological Control

The discovery of hypovirulent strains of the chestnut blight fungus in the 1950's and their isolation and identification in the 1960's is by far the most exciting event in chestnut blight research in recent years. It may be premature to consider use of hypovirulent strains as a biological control technique, but the potential appears to be great enough for initiating and funding new research programs. Much more work needs to be done with hypovirulent strains to learn more about the mode of transmission of the hypovirulent effect. Some of the problems already recognized deal with how to introduce hypovirulent strains into native stands of chestnut. Studies are needed on the effect of time of year of inoculations in various geographic locations on callus formation and dominance of the hypovirulent strains. As indicated earlier, time has been on the side of the fungus and its genetic diversity is probably much larger today than at the initial outbreak. In fact, experience in Connecticut has already shown that within a region, virulent cultures vary greatly in their compatibility with hypovirulent cultures. Practically nothing is known about how selection pressure over time in hypovirulent strains affects their compatibility with native virulent strains.

Perhaps one of the highest priority tasks in our new chestnut blight programs is an indepth survey of the old test plantings with native and hybrid selections. These plantings have not been visited for quite some time, and many unfortunately, have probably been destroyed. Nevertheless, they represent a wide range of genetic parentage and environmental growing conditions—an excellent situation for exploring genotype x environment interactions. Some of these trees may be beginning to show evidence of healing of cankers and could serve as a source of cultures for hypovirulent strains. Surviving trees could also be examined for evidence of partial resistance because, as stated earlier, we may have set resistance standards too high and overlooked opportunities for developing a semi-resistant clone.

Another related and highly important area of research that needs to be considered in the chestnut program is the development of appropriate silvicultural methods for establishing, tending, and managing plantations and natural stands of chestnut. Our first reaction may be that it is too early to think about these problems, but we could easily find ourselves with resistant or semi-resistant clones or an effective control method and totally inadequate knowledge of how to grow the trees. Planting of hardwoods has historically been extremely difficult and it has only been in recent years that the necessary requirements for site selection, site preparation, spacing, and competition control have been worked out for even a few selected hardwoods. How much of this knowledge is directly translatable to chestnut culture is not known, but the problem should be ever present in our discussions. Most of the native chestnut has been relegated to an understory position. If hypovirulent strains are effective in controlling the disease, what cultural prescriptions are needed to get the sprouts into the overstory?

CONCLUSIONS

It thus appears that one of the most important challenges which we must face in chestnut research is to maintain research programs with realistic goals. Furthermore, these programs should be conducted in a truly scientific manner and followed by unbiased, objective analysis of the results. The frustrations and mistakes of the past are well documented and it is inexcusable to repeat them in new programs. There is reason for optimism but it must be kept in proper perspective to the extent of the problem ahead. There sometimes exists the attitude that discovery of a resistant chestnut would immediately bring the chestnut back to its place of prominence in the eastern hardwood forests. The truth is, even if we had a resistant clone tomorrow, it would be decades, or even centuries, before we could restore it to even a fraction of its original importance. The tasks of reproducing and establishing new chestnut stands or altering the ecology of natural hardwoods stands to allow reintroduction of a former species are monumental.

LITERATURE CITED

Hepting, George H.
1974. DEATH OF THE AMERICAN CHESTNUT. J. For. Hist. 18(3):61-67.

Jaynes, R. A.
1974. PROPAGATION OF CHESTNUT AND OTHER DIFFICULT-TO-ROOT PLANTS FROM SOFTWOOD CUTTINGS. Ann. Rep. North Nut Growers Assoc. 65:128-131.

Jaynes, R. A. and N. K. Van Alfen.
1974. CONTROL OF AMERICAN CHESTNUT BLIGHT BY TRUNK INJECTION WITH METHYL-2-BENZIMIDAZOLE CARBAMATE(MBC). Phytopathology 64:1479-1480.

Shreve, L. W. and N. W. Miles.
1972. PROPAGATING BLACK WALNUT CLONES FROM ROOTED CUTTINGS. Ann. Rep. North Nut Growers Assoc. 63: 45-49.

Thor, Eyvind.
1973. THE AMERICAN CHESTNUT: 1973. National Parks and Conservation Magazine 47(9): 9-11.