THE SEARCH FOR DISEASE-RESISTANT TREES

by Malcolm C. Shurtleff

Infectious disease is the result of the interaction of a pathogen, a host and the environment. All conditions must be just right for infection and disease development to occur (1, 34, 49, 52). For example, the temperature, humidity and moisture conditions must be within certain specific ranges. Some means is also necessary to transmit the pathogen from where it has reproduced and/or survived to the susceptible area of the host where infection can occur.

Development of plant disease can be prevented or at least controlled by four basic principles: Exclusion, Eradication, Protection, and Immunization. Exclusion generally involves legislative measures — imposing of quarantines or embargoes against the movement of plants from one state or country to another. Eradication usually means destruction of diseased individuals or some of their parts to protect the remainder of the population. Eradication of citrus canker from the United States is an example of protecting an entire industry against a major disease. We tried eradication with chestnut blight, white pine blister rust, and Dutch elm disease but our efforts were too little and too late (1, 5). Protection, the third method of control, generally means preventing the disease-causing agent or pathogen from entering plants and producing disease — mostly by chemical sprays (1, 22, 34, 49).

The principles of Exclusion, Eradication and Protection are functional by excluding or inhibiting the pathogen *prior* to its coming into intimate contact with the host. The principle of Immunization allows intimate contact between the pathogen and host. Penetration of the host occurs but subsequent disease development is checked; the host is resistant or immune (1, 34, 49).

Immunization can be achieved through the use of resistant varieties or chemotherapy (1, 22, 34). We are just entering the age of chemotherapy with trees using systemic fungicides or chemotherapeutants applied to the soil, buttress roots, trunk, or foliage.

There are four sources of genes for resistance: 1) adapted species, varieties, and cultivars presently in commercial use; 2) source populations from which adapted varieties and cultivars can be derived; 3) exotic sources maintained away from the center of origin of the host; and 4) wild (or near) relatives of the host from the center of origin. This material can be screened for the presence of resistance against the pathogen or pathogens. Hopefully, resistance can be found in populations closer genetically to adapted varieties and cultivars than from populations of near relatives. This facilitates transfer of resistance genes into a commercially acceptable cultivar or clone whose only principal fault is that it is susceptible to a particular pathogen (2, 8, 16, 20, 30, 31, 33, 42).

If resistance is found, the genetics of inheritance should be determined to choose the proper breeding procedure and incorporate this resistance into a commercially acceptable variety. Three main types of inheritance of disease resistance exist: 1) Mendelian inheritance, 2) polygenic inheritance, and 3) extrachromosomal or cytoplasmic inheritance (2, 16, 20, 21, 30, 31, 33, 42).

Resistance due to Mendelian inheritance is usually conditioned by only one or two major genes, or a single gene pair. This is often called specific or vertical resistance. Mendelian inheritance is identified by discrete ratios of resistant and susceptible plants in evaluation of test cross progenies to the pathogen. History has shown that mutations of the pathogen commonly break down this type of major gene resistance, often in a period of a few months or years (5, 30, 31, 35, 48).

Resistance due to polygenic inheritance is controlled by several to many minor genes or gene pairs. This type of inheritance is identified by a continuous distribution of resistant to susceptible plants in evaluation of test cross progenies to the pathogen. This type of resistance is sometimes referred to as general or horizontal resistance. The pathogen must undergo many mutations to

competely break down this type of resistance. Polygenic inheritance gives "permanent protection." In most cases, a combination of major and minor genes for resistance are used against a pathogen (5, 30, 31, 36, 48).

Whenever satisfactory resistant species, varieties or cultivars are available, they are preferred over other means of control because they add little to the cost of production and maintenance. Disease resistance is "built into" the plant, whereas other means of control can be affected by weather, mechanical or other failure, and improper planning resulting in a delay of applying control measures as is common with protective chemical sprays. The basic principles underlying plant breeding programs are the same regardless of species, be it wheat, corn, elms, or maples (2, 5, 31, 42, 49).

Considerations in a Breeding Program

In initiating a program for selecting and breeding for disease resistance, detailed knowledge of the genetics of the host and pathogen and their resulting interactions are required for intelligent planning (5, 42).

With the rediscovery of Mendel's laws in 1900, it became evident that phenotypic characters in plants are heritable. In 1905, resistance to yellow or stripe rust of wheat was shown to be governed by a single recessive gene (3). This work started the science of breeding for disease resistance which quickly became a whole new scientific discipline. With the knowledge that resistance can be inherited, procedures to incorporate resistance into adapted varieties or cultivars can be explored (2, 5, 8, 31).

The great bulk of research dollars, time and manpower has been placed on annual food and fiber crops, especially small grains (which includes rice), beans, corn, potatoes, tomatoes, and cotton (7, 16, 30, 33). With these crops you can achieve one to several generations per year by using greenhouses or field plots in a southern state, Puerto Rico or a country in the southern hemisphere. Administrators in both the public and private sectors have chosen to put the bulk of their dollars, people and facilities into food and fiber crops with the improvement of shade and ornamental trees relatively low on their scale of priorities. Is this good or bad realizing that the world's population is growing at about 70 million new people each year — a total equal to the population of France (1, 32)?

In breeding for disease resistance, a gene or genes *must* be present and the nature of inheritance should be understood.

Choosing a proper breeding procedure to incorporate disease resistance into acceptable varieties depends primarily on the type of variety or cultivar desired. One of two types is usually desired: 1) the susceptible variety is wanted to be made resistant, or 2) an entirely different resistant variety is desired to replace the susceptible one (5, 30, 31).

In replacing a susceptible variety with a very similar but resistant variety, a backcrossing system of incorporating disease resistance into the susceptible variety is commonly used. This procedure involves crossing the resistant source to the susceptible source. The progeny is then crossed back to the susceptible one. The resistant plants are classified and saved. This is done for two to six cycles (two or three for trees; six cycles or more for annual plants). At the end of these cycles the resistant plants comprise many traits of the original variety (which was susceptible). Where feasible, the resistant plants are self-pollinated until a true-breeding resistant variety is obtained (2, 5, 16, 30, 31, 33, 42). In the case of trees, cuttings are usually made from the resistant parent tree or trees.

The type of inheritance involved modifies this scheme only in the size of the populations used to detect resistance. When the resistance is Mendelian a relatively small population size is required to ensure advancement with each crossing cycle. In the case of polygenic inheritance, however, a much larger population size is needed to be sure that the most resistant plants are advanced to the next cycle (2, 5, 16, 20, 21, 31, 33, 42).

If an entirely new resistant variety is desired, the effect of type of inheritance remains the same. Resistance that is polygenically inherited requires a much larger population size than Mendelian inheritance to advance resistance through each cycle. Numerous breeding procedures can result in a well-adapted resistant variety. Breeding

schemes such as mass selection of resistant individuals from large heterogenous populations, varietal hybridization and selection, backcrossing, interspecific crosses, hybridizing wild species with susceptible varieties of the cultivated species, and modifications of all these types have resulted in commercially acceptable varieties and cultivars (2, 5, 16, 30, 31, 33, 42).

In breeding for disease resistance, the pathogen should be examined for its potential variability via gene mutation, gene segregation and recombination, heterokaryosis, and parasexualism (30, 31, 42). The reproductive cycles of many pathogens have been investigated to the extent that their genetic variability has been shown to be as diverse as is found in higher plants (4, 5, 8, 9, 15, 20, 28, 42, 50, 52). This leads to the conclusion that pathogens have the capacity to utilize gene recombination, the single most important factor in potential variation. The capacity for genes to recombine rests on how the pathogen reproduces. Pathogens that reproduce asexually are restricted to changes in chromosome structure and possibly chromosome number for their variability. These pathogens are usually fairly stable with new races seldom occurring in nature. Pathogens that reproduce sexually (where nuclei combine) utilize genetic recombination and new races often occur in nature. This information is basic before starting a tree breeding program for disease resistance (5, 42).

We also need to realize the potential variability and the concept that for each gene for pathogenicity (virulence) in the pathogen there is a corresponding gene for resistance in the host and that disease expression results from the interaction of these corresponding genes in the two genetic systems (1, 11, 43). The gene-for-gene concept was first expressed by H.H. Flor for flax rust in 1956 (10).

An old axiom is that the prolonged use of any one source of resistance will ultimately result in a race of the pathogen that can overcome this resistance. The present trend in breeding for disease resistance is to combine Mendelian, polygenic and, where applicable, extrachromosomal inherited resistance into the same variety. Hopefully, this will "tax" the pathogen to the extent that its reproducing potential will be so limited that it is effectively controlled (5, 30, 31, 48).

Trees obviously present unique problems in selection and breeding when compared to annual plants: 1) they take a long time to reach sexual maturity, i.e., produce flowers and fruit; 2) trees have irregularities in flowering and fruiting. For example, we have spring- and fall-flowering elms and magnolias; 3) insects frequently destroy the flowers and/or fruits; 4) there are obvious space limitations in the laboratory, greenhouse and field for mass selection; and 5) interspecific crosses may be difficult or impossible due to different numbers of chromosomes and other factors.

Norman Borlaug, who started his professional career as a tree pathologist-forester, was awarded the Nobel Peace prize about 10 years ago for his role in the "Green Revolution." In speaking at a N.A.T.O. and N.S.F. Advanced Study Institute on Genetic Improvement for Disease Resistance of Forest Trees in 1964 (5), Borlaug stated that in organizing a breeding program for the development of disease-resistant tree species and cultivars there are a number of fundamental questions and considerations that must be brought into harmony:

1) What are the principal diseases that limit productivity? Is it physically and technically possible to design a breeding program that will simultaneously incorporate resistance to all major diseases? One must establish a system of priorities for incorporating disease resistance into a cultivar.

2) What techniques or methods are best for identifying the gene or genes for resistance to the principal diseases?

3) How can the genes for resistance be incorporated into the commercial parental variety (or cultivar) without simultaneously introducing adverse effects on such characters as rate of growth, desirable form and shape, and also incorporating increased susceptibility to other diseases or insect pests that were previously only of minor importance?

4) How can the desired disease resistance be incorporated into a new variety so that once attained, it will be maintained in a useful form for many years? (5)

What Borlaug achieved with wheat working for

20 years in Mexico, would probably take 2,000 years with chestnut, elm, maple, oak, pine, and poplar. It is discouraging but not impossible; short cuts are possible.

There are five steps to get better trees into an urban landscape: 1) mass field selection to discover sources of resistance. This involves testing of many thousands of individuals collected over a wide geographical area; 2) vegetative propagation of likely candidates. But how can one reduce this number quickly to get a breeding program down to a manageable size?; 3) trial plantings over a wide area and under highly variable conditions; 4) evaluation of test selections with certification (if feasible) of the best individuals to build up clone numbers; and 5) distribution to commercial growers (24, 25, 42).

Unfortunately, plant breeders and pathologists can't just focus on improving disease resistance. An urban environment is unnatural or alien to many of our native and introduced trees (12, 13, 38). Adult trees in a city usually die much sooner than they would in a natural habitat. Ruth Foster and Joan Blaine (13), in a study of survival of trees in the city of Boston, found that the average life was 10 years. Seventy-three percent of the young trees on a downtown street had auto wounds and 33 percent were vandalized or had broken branches (12). Foster believes that city engineers are the most common cause of urban tree death. She and Joan Blaine also say "a tree pit in the sidewalk is like a flower pot in the desert." The cumulative effect is that city trees grow less, mature too early, and die too soon (12, 13).

Urban stresses, both biotic and abiotic, often operate in combination. For example, tolerance to many diseases is weakened by adverse site conditions and by air pollution.

Requirements of paramount importance for selecting shade and ornamental trees in urban forestry are resistance or tolerance to: 1) various pests and diseases; 2) resistance to a wide range of chemicals such as air pollutants, de-icing salts, pesticides — including herbicides — and growth regulators; 3) resistance to intense heat or cold — the plants must be hardy and widely adapted; 4) resistance to a water shortage or excess; 5) resistance to soil compaction and the ability to get along with a reduced area for root growth; 6) resistance to storm damage and wind stress — either by virtue of wood quality, crown shape or branch angle; and 7) resistance to continuing construction (12, 13, 45). Besides all this, we demand good tree vigor, desirable shape, size, fruit and leaf color, and suitable growth rate (45).

The ultimate goal of shade-tree improvement in any genus or species is a clone possessing the greatest number of desirable attributes.

From a practical standpoint nurserymen and arborists can not possibly grow and sell all the new shade and ornamental tree cultivars that are being released each year. Nurserymen need trees that (1) are readily propagated from seed, cuttings, or buddings (grafting). This is not possible or easy in some species. Good examples are the chestnuts resistant to chestnut blight which are the result of more than 40 years of breeding and testing, by the Connecticut Agricultural Experiment Station; (2) nurserymen need a rapid growth rate — unfortunately this often brings susceptibility to storm damage or other pest problems; and (3) nurserymen need trees that transplant easily with a high rate of survival.

Before a selection and breeding program for trees can go into effect, a multidisciplinary approach is needed that involves geneticists, plant pathologists, and horticulturists working closely together on a long-range project (5, 6, 42). Administrators must realize that the project needs to be adequately funded for 40 to 50 years or more to bring forth lasting and useful results.

Governmental agencies and the nursery-arborist industry should join forces to provide a much broader array of better street and ornamental trees than are now available. A broadly based educational program to inform potential consumers with which trees do best under specific environmental conditions is vitally needed.

Natural populations of trees have large variations among species, races and individuals as regards stress tolerances and this provides vast possibilities for the selection and breeding of superior types (16, 42). Example: Pollution-tolerant varieties can be developed by detection of variation now known to exist in natural stands of at least 15 tree species (17). We need to select and breed superior races and individuals. Once genetic diversity has been detected, it is possible

to maintain it as a safeguard.

Where are we at present in selecting and breeding disease-resistant shade and ornamental trees? Unfortunately, not very far (16, 42). The first research project in the United States for systematic selecting and breeding shade and ornamental trees for urban planting was started at the National Arboretum near Washington, D.C. in July, 1967. This is a very late start when compared to food and fiber crops and even forest trees (5, 38).

Examples of Tree Breeding Programs

Elms. Frank Santamour, with his research on elms at the U.S. National Arboretum, is currently working on breeding resistant elms in a major way, using advanced techniques (39, 40).

Heybroek, a Dutch scientist, has summarized the development and release of Dutch elm disease-tolerant clones of elms, starting with the work of Christine Buisman in the last 1920's and ending with the release of 'Plantyn' in 1975 and later of 'Lobel' and 'Dodens' (19). The time span developing parents through the F_3 generation is from 1927 to 1975, a period of 48 years. Resistance was developed step-by-step over two generations and three rounds of selection. The variety, later to be named 'Plantyn,' was obtained in 1964 and entered the field test in 1967. By 1975 it had been planted in 24 replicated field tests, each containing many clones. Release of these clones was suspended in 1971. Why? A new and more virulent strain of the pathogen was discovered in Great Britain, believed to be imported there from Canada. Four clones were tested in Great Britain, the Netherlands and the USA in an international cooperative experiment. As a result, two clones were released in 1975, named 'Plantyn' and 'Lobel.' A third was added later and called 'Dodens.' Although these clones are not perfect, they are stated to be better than prevailing cultivars (19). 'Christine Buisman,' released under pressure of administrators in 1936, was the first elm bred with resistance to Dutch elm disease. It has an undesirable bushy growth and shape, plus being susceptible to Nectria canker and cold injury. From the Dutch breeding program then came 'Bea Schwarz' which grew too slowly. The clone 'Commelin,' released in 1961, seems to have no major drawback and is being widely planted as the replacement for the older 'Belgica.' A fourth clone 'Groeneveld,' with a narrow crown and slower growth, was released in 1963 (18, 19).

An elm-breeding program is also going on in Canada. The Japanese elm (*Ulmus japonica*), a hardy Asiatic species with the growth form of the native American elm (*U. americana*), has spawned the 'Jacan' elm. This new cultivar was released by the Morden Experiment Station in 1977. It is tolerant to Dutch elm disease and can be propagated from seed or by budding on Siberian elm (*U. pumila*) rootstock (37).

The Wisconsin elm screening and breeding program has been going on for about 20 years. It aims at producing seed-propagated material from a backcross of Siberian by Japanese elm × Japanese elm. A seedling population of elm that has less than 10 percent of susceptible individuals is regarded as acceptable for disease resistance. This is a practical example of the superiority of a seedling population produced by hybridization in comparison with a clonal population obtained by direct selection (27).

The USDA Nursery Crops Research Laboratory at Delaware, Ohio has developed more than 60 combinations of hybrid elms (44, 45, 47). One of their best and most recent crosses has yielded a hybrid that is remarkably beautiful, fast-growing, and disease-resistant. This cross is between the Siberian elm and a Dutch selection, N 274 × 215 (*Ulmus hollandica vegeta* × *U. carpinifolia*). This cross shows great promise as a source of superb urban clones (45). The USDA program has already released the 'Urban' elm (41). An American elm selection, Delaware No. 2, compares well with several disease-resistant, non-American elm clones (47). This tree is currently being propagated and tested and should be released within the next few years (45).

All elm species except the American elm have a gametic chromosome number of 14; the American elm is a tetraploid with gametes containing 28 chromosomes; which complicates the breeding problem. But we are definitely making progress is producing a DED-resistant elm with the desirable form and other characteristics of an American elm (26, 40).

A major obstacle in all these breeding programs, however, is to convince nurserymen to grow these new elm cultivars and for arborists to buy and recommend them.

Another concern is that in the rush to incorporate Dutch elm disease resistance we may be ignoring other disease problems such as phloem necrosis, various canker and dieback diseases, other wilts, and wetwood.

Maples. At present, there are 150 species of *Acer* being grown and 170 cultivars from these species offer great diversity in size, shape, leaf and fruit characteristics, and leaf coloration. Maples are the most frequently planted group of urban tree species (14, 29, 45). Unfortunately, the 170 cultivars now available have been selected primarily on aesthetic superiority rather than for adaptability and tolerance to urban stresses (25, 29, 44, 45). None has resulted from a systematic program of breeding and selection.

Townsend started an extensive provenance and progeny test of red maple in 1971. In 1973, he found significant variation among half-sib families of red maple in their tolerance to the Verticillium wilt fungus. This was the first reported instance of intra-specific variation in a tree species in tolerance to this important disease, and it indicates the potential for genetically increasing this tolerance through selection and breeding (45, 46).

Chestnuts. The *Endothia* fungus that causes chestnut blight was introduced into the USA at the turn of this century. Breeding against blight began in the late 1920's at the Brooklyn Botanical Garden and later the Connecticut Agricultural Experiment Station in New Haven (1, 5, 23, 51). Crosses to produce hybrid chestnuts have involved many species. The most promising are crosses between the American (*Castanea dentata*), Chinese (*C. mollissima*), and the Japanese (*C. crenata*) chestnuts. These hybrids have shown good disease resistance but few have shown desirable tree form in combination. Many of the hybrids are also susceptible to Nectria twig blight and canker as well as frost injury (51).

Summary

Breeding for disease resistance is but one means of controlling tree diseases. In initiating a program of breeding for disease resistance many things must be considered: variation within the pathogen and host; cataloging of all possible sources of resistance; knowing the mode of inheritance, and the often intricate genetics of plant and pathogen interactions. In examining these considerations, breeding for disease resistance is successful to the extent that pathogens commonly have the capability of ultimately overcoming the sources of resistance used which necessitates a never-ending program of screening and breeding for disease resistance (6).

It is admittedly a long-range program that is presently starving for dollars and teams of dedicated scientists willing to spend a considerable portion of a professional lifetime in the hope of producing several disease-resistant clones that will be widely grown by the nursery-arborist trade and then by the consuming public.

Literature Cited

1. Agrios, G.N. 1978. Plant pathology. 2nd ed. Academic Press, Inc., N.Y.
2. Allard, R.W. 1960. Principles of plant breeding. John Wiley & Sons, Inc., N.Y.
3. Biffin, R.H. 1905. *Mendel's law of inheritance and wheat breeding.* Jour. Agric. Sci. 1: 4-48.
4. Boone, D.M. 1971. *Genetics of Venturia inaequalis.* Ann. Rev. Phytopathol. 9: 297-318.
5. N.E. Borlaug. 1966. Basic concepts which influence the choice of methods for use in breeding for disease resistance in cross-pollinated and self-pollinated crop plants. Pages 327-344 **In** Breeding pest-resistant trees. H.D. Gerhold, E.J. Schreiner, R.E. McDermott, and J.A. Winieski (eds.). Pergamon Press, N.Y.
6. Callahan, R.Z., R.E. Goddard, H.M. Heybroek, C.M. Hunt, G.I. McDonald, J.A. Pitcher, and J.A. Winieski. 1966. General guidelines for practical programs toward pest-resistant trees. Pages 489-493 **In** Breeding pest-resistant trees. H.D. Gernold, E.J. Schreiner, R.E. McDermott, and J.A. Winieski (eds.). Pergamon Press, N.Y.
7. Dickson, J.G. 1956. Diseass of field crops. 2nd ed. McGraw-Hill Book Co., N.Y.
8. Day, P.R. 1974. Genetics of host-parasite interaction. W.H. Freeman Co., San Francisco.
9. Fincham, J.R.S., and P.R. Day. 1971. Fungal genetics. 3d ed. Oxford: Blackwell, Philadelphia.
10. Flor, H.H. 1956. *The complementary genic systems in flax and flax rust.* Advan. Genet. 8: 29-54.
11. Flor, H.H. 1971. *Current status of the gene-for-gene concept.* Ann. Rev. Phytopathol. 9: 275-296.
12. Foster, Ruth S. 1977. Desirable traits for city trees. Pages 21-26 **In** Proceedings 16th Meeting Canadian Tree Improvement Association: Part 2, University of Manitoba, Winnipeg, June 27-30.
13. Foster, Ruth S., and Joan Blaine. 1978. *Urban tree sur-*

vival; trees in the sidewalk. Jour. Arbor. 4: 14-17.
14. Gerhold, H.D., and K.C. Steiner. 1976. Selection practices of municipal arborists. Pages 159-166 **In** Better Trees for Metropolitan Landscapes, Frank S. Santamour, Jr. (ed.). U.S. For. Serv. Gen. Tech. Rep. NE-22.
15. Halisky, P.M. 1965. *Physiologic specialization and genetics of the smut fungi.* Ill. Bot. Rev. 31: 114-150.
16. Hayes, H.K., F.R. Immer, and D.C. Smith. 1955. Methods of plant breeding. McGraw-Hill Book Co., Inc., N.Y.
17. Heggestad, H.E., F.S. Santamour, Jr., and L. Bernstein. 1972. Plants that will withstand pollution and reduce it. Pages 16-22 **In** U.S. Dept. Agric., Yearbook Agric. 1972.
18. Heybroek, H.M. 1966. Aims and criteria in elm breeding in the Netherlands. Pages 387-389 **In** Breeding pest-resistant trees. H.D. Gerhold, E.J. Schreiner, R.E. McDermott, and J.A. Winieski (eds.). Pergamon Press, N.Y.
19. Heybroek, H.M. 1976. Chapters on the genetic improvement of elms. Pages 203-212 **In** Better Trees for Metropolitan Landscapes, Frank S. Santamour, Jr. (ed) U.S. For. Serv. Gen. Tech. Rep. NE-22.
20. Hooker, A.L. 1976. *The genetics and expression of resistance in plants to rusts of the genus Puccinia.* Ann. Rev. Phytopathol. 5: 163-182.
21. Hooker, A.L. 1974. *Cytoplasmic susceptibility in plant disease.* Ann. Rev. Phytopathol. 12: 167-179.
22. Horst, R.K. 1979. Westcott's plant disease handbook. 4th ed. Van Nostrand Reinhold Co., N.Y.
23. Janes, R.A., and A.H. Graves. 1963. Connecticut hybrid chestnuts and their culture. Conn. Agr. Exp.ta. Bull. 657.
24. Jorgensen, E. 1967. Approaches to shade tree research at the University of Toronto. Pages 256-266 **In** Proceedings 43rd Int. Shade Tree Conf.
25. Khalil, M.A.K. 1977. Genetic improvement of trees for the urban environment: challenges and opportunities. Pages 9-19 **In** Proceedings 16th Meeting Canadian Tree Improvement Association: Part 2, University of Manitoba, Winnipeg, June 27-30.
26. Lester, D. 1969. Genetics and breeding of American elm. Pages 9-13 **In** 16th Northeastern Forest Tree Improvement Conference, Macdonald College, Quebec, Canada, Aug. 8-10, 1968.
27. Lester, D.T., and E.B. Smalley. 1972. *Variation in ornamental traits and disease resistance among crosses of Ulmus pumila, U. rubra, and putative natural hybrids.* Silvae Genet. 21: 193-197.
28. Moulton, F.R. 1940. The genetics of pathogenic organisms. Science Press, Lancaster, PA.
29. Mulligan, B.O. 1958. Maples cultivated in the U.S. and Canada. Amer. Assoc. Bot. Gard. and Arbor.
30. National Academy of Sciences. Principles of plant and animal pest control. Vol. 1. Plant disease development and control. Washington, D.C.
31. Nelson, R.R. (ed.). 1973. Breeding plants for disease resistance. Concepts and applications. The Pennsylvania State University Press, University Park.
32. Paddock, W.C. 1967. *Phytopathology in a hungry world.* Ann. Rev. Phytopathol. 5: 375-390.
33. Poehlman, T.M. 1959. Breeding field crops. Henry Holt and Co., Inc., N.Y.
34. Roberts, D.A., and C.W. Boothroyd. 1972. Fundamentals of plant pathology. W.H. Freeman and Co., San Francisco.
35. Robinson, R.A. 1971. *Vertical resistance.* Rev. Plant Pathol. 50: 233-239.
36. Robinson, R.A. 1973. *Horizontal resistance.* Rev. Plant Pathol. 52: 483-501.
37. Ronald, W.G. 1977. Evaluating and breeding urban trees for the prairies. Pages 1-7 **In** Proceedings 16th Meeting Canadian Tree Improvement Association: Part 2, University of Manitoba, Winnipeg, June 27-30.
38. Santamour, F.S., Jr. 1969. Breeding trees for tolerance and stress factors of the urban environment. Pages 627-638 **In** FAO/IUFRO Second World Consult. on For. Tree Breed., Washington, D.C., August 7-16, 1969. No. FO-FTB-69-6/4.
39. Santamour, F.S., Jr. 1972. *Interspecific hybridization with fall- and spring flowering elms.* For. Sci. 18: 283-289.
40. Santamour, F.S., Jr. 1974. *Resistance of new elm hybrids to Dutch elm disease.* Plant Dis. Reptr. 58: 727-730.
41. Schreiber, L.R., and H.V. Main. 1976. *'Urban' elm.* HortScience 11: 517-518.
42. Schreiner, E.J. 1966. Future needs for maximum progress in genetic improvement of disease resistance in forest trees. Pages 455-466 **In** Breeding pest-resistant trees. H.D. Gerhold, E.J. Schreiner, R.E. McDermott, and J.A. Winieski (eds.). Pergamon Press, N.Y.
43. Sidhu, G.S. 1975. *Gene-for-gene relationships in plant parasitic systems.* Sci. Prog., Oxford 62: 467-485.
44. Townsend, A.M. 1975. *Specific crossability patterns and morphological variation among elm species and hybrids.* Silvae Genet. 24: 18-23.
45. Townsend, A.M. 1977. Improving the adaptation of maples and elms to the urban environment. Pages 27-31 **In** Proceedings 16th Meeting Canadian Tree Improvement Assoc.: Part 2, University of Manitoba, Winnipeg, June 27-30.
46. Townsend, A.M., and W.K. Hock. 1973. *Tolerance of half-sib families of red maple to Verticillium wilt.* Phytopathology 63: 673-676.
47. Townsend, A.M., and L.R. Schreiber. 1975. Recent advances in the breeding and selection of elms. Pages 25-28 **In** Proceedings Central States Forest Tree Improvement Conf.
48. Van der Plank, J.E. 1968. Disease resistance in plants. Academic Press, Inc., N.Y.
49. Walker, J.C. 1969. Plant Pathology. 34d ed. McGraw-Hill Book Co., N.Y.
50. Webster, R.K. 1974. *Recent advances in the genetics of plant pathogenic fungi.* Ann. Rev. Phytopathol. 12: 331-353.
51. Winiski, J.A. 1966. Pest-resistance research projects and breeding programs of forest trees in Northeastern United States. Pages 71-75 **In** Breeding pest-resistant trees. H.D. Gerhold, E.J. Schreiner, R.E. McDermott, and J.A. Winieski (eds.). Pergamon Press, N.Y.
52. Wood, F.A. 1966. The current status of basic knowledge of forest tree disease resistance research. Pages 590-606 **In** Breeding pest-resistant trees. H.D. Gerhold, E.J. Schreiner, R.E. McDermott, and J.A. Winieski (eds.). Pergamon Press, N.Y.

Extension Plant Pathologist
University of Illinois
Urbana, Illinois