

Plant Pathology: A 55-Year Retrospective*

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Plant pathologist George Zentmyer, Professor Emeritus in the Riverside branch of the University of California, has devoted much of his life to the benefit of avocado growers worldwide. He is widely known in our industry, respected and revered. Dr. Zentmyer has for forty-eight years enlightened members of the California Avocado Society through his annual contributions to the Yearbook. In this article, his admirers have an opportunity to look back, with him, on his long, distinguished—and still active—role in the avocado industry.

Over the past nearly sixty years I have fortuitously been involved in three of the major plant diseases of the 20th century: White Pine Blister Rust (WPBR); Dutch Elm disease (DED); and *Phytophthora cinnamomi* root rot, of over 1000 hosts (especially of avocado and eucalyptus). This chapter provides a retrospective of my research on the world epidemics of these and other plant diseases.

My interest in plant pathology was based on pure chance—on the occasion of a summer vacation in northern Washington, in the little town of Northport on the Columbia River, when I was 15. An invitation from a wise uncle initiated this fateful decision. Uncle Arthur Strahorn was a soil scientist with the USDA on a several-year assignment studying smelter damage to the coniferous forests in northern Washington from a huge smelter at Trail, British Columbia.

Working with my uncle was a young forest pathologist with whom my uncle and I went trout fishing on weekends and evenings, and who kindly showed me some of the unique tree diseases he was working on. He patiently explained to me aspects of the mysterious field of plant diseases, fungi, disease spread and control—a totally new world to a young high school junior!

Then and there, with the love I have always had for mountains and the outdoors, my future was decided: Forest Pathology was to be my goal! Nine years later I accepted a full-time position in the Division of Forest Pathology, USDA, headquartered in San Francisco, to work on forest disease problems in northern and central California, Oregon, and Utah. A year after that, (1938) I completed my Ph.D. requirements from the University of California, Berkeley, in Plant Pathology (Forest Pathology).

PREPARATION

En route to my visualized profession of forest pathology I received excellent undergraduate training at UCLA, especially in the sciences—botany (plant anatomy, cytology, taxonomy, physiology, morphology), chemistry, physics, and geology. There were at UCLA then very capable and provocative professors who sparked my interest in biology, including Flora Murray Scott, Carl Epling, O. L. Sponsler, and O. A. Plunkett, who introduced me to the world of fungi.

My graduate studies were at the University of California, Berkeley, the parent of what became later the mammoth nine-campus U.C. system. Berkeley had a vigorous and thriving plant pathology department in the 1930s. The situation in Berkeley then was ideal, with the stimulating climate—both intellectual and environmental—and the idyllic campus. Graduate studies in Plant Pathology were inspiring and challenging, with close contact with a unique faculty that included James T. Barrett, Max W. Gardner (Chairman), H. N. Hansen, T. E. Rawlins, William C. Snyder, Ralph Smith (the first plant pathologist in California, just retired), Harold Thomas, Harvey Thomas, and C. E. Yarwood. My first plant pathology course was with a wonderful and knowledgeable professor, James Barrett, and a young, enthusiastic assistant professor Bill Snyder was in charge of the laboratory course.

My major professor was H. N. (Nick) Hansen of Hansen & Snyder *Dual Phenomenon* fame (4), who in addition to his beloved mycological studies taught Forest Pathology. I was willingly drafted to assist in the course, which was taught for the first time that year. Professor Hansen was an uncompromising taskmaster who insisted on nothing but the best from his students in both courses and research. He was a rugged perfectionist with his own definite and original thoughts and ideas. This was fine training and helped my career immeasurably. Once one got beneath the gruff exterior the twinkle in the eye and the friendship were wonderful. I owe a great deal to Nick Hansen's high ideals and guidance. Ken Baker sagely commented in his prefatory chapter (1) that "Graduate studies should be one of life's most pleasant and rewarding experiences." How true this was in my case!

Another helpful aspect of my educational experience through high school and the university resulted from my fascination and involvement with journalism as editor and sports editor of the high school newspaper and yearbook, and at UCLA as sports editor of the Yearbook and sports editor of the Daily Bruin. This has served me well in considerable writing and editorial responsibilities in my career (25 years as Associate Editor of the Annual Review of Phytopathology, for example).

In fact, I nearly became a sports writer, but now realize that my chosen career was really ideal, because I combined my love of writing with a fascinating and important field of science. I have always been interested in sports and have actively played baseball, basketball, badminton, handball, and tennis. Not making any team at UCLA I made a four year mini-career of writing for the Daily Bruin of the exploits of UCLA football and basketball teams (under two very human and inspiring coaches, Bill Spaulding and Caddy Works). My sports interests were vicariously satisfied by covering the teams' league games, including trips north by train to Palo Alto, Berkeley, Corvallis, and Seattle

as "foreign correspondent" for the Daily Bruin, sending dispatches to the L. A. Times, with appropriate bylines!

FOREST PATHOLOGY

My first position in forest pathology in San Francisco, in 1937, provided an unparalleled opportunity for contact with the pioneer forest pathologist in the country E. P. Meinecke, who was trained in his native Germany under the father of forest pathology, Robert Hartig. Dr. Meinecke had just retired when I joined the office, but the contacts with him over the next few years were priceless.

White Pine Blister Rust

I was very fortunate to be involved in research on two of the major forest and shade tree diseases in the world, white pine blister rust and the Dutch elm disease, when they were actively spreading, even exploding into new areas of the United States. In my graduate studies at Berkeley, I wrote a comprehensive paper on the white pine blister rust in 1936. What an experience then in 1937 to be part of a Forest Pathology research team that monitored the spread of *Cronartium ribicola* from Oregon into northern California, by scouting through the forests of the northern Sierra Nevada looking for white pine blister rust leaf symptoms on the alternate host species of *Ribes*.

In relation to the white pine blister rust epidemic that our Forest Pathology team was studying in 1937-40, a comment by Dr. Meinecke in 1931 (7) was very prophetic. He stated, "The greatest menace from killing epidemics has come through the accidental introduction of foreign diseases. It is a common experience that certain diseases, harmless enough in their native habitat, may assume formidable proportions when they are transferred to new surroundings where they find more congenial conditions and more susceptible hosts." WPBR is one of the many deadly examples of this.

The sad story of WPBR in the United States begins with the strange management procedures followed in European forests. The European forest industry relies not on indigenous pines but on plantations of imported species. Our eastern white pine, *Pinus strobus*, was introduced into Europe from the United States in 1705 because of its superior growth and fine timber production. *P. strobus* soon became one of the primary trees in forest nurseries and plantings in much of Europe. (Much later it was found to be very susceptible to WPBR.) WPBR was first reported in the Baltic Provinces of Russia in 1854. It is thought to have originated in Asia on *Pinus cembra*. The disease then spread rapidly through western Europe, mostly on *P. strobus*, and currants and gooseberries.

In 1906, the dreaded WPBR appeared in Geneva, New York. The disease was ironically traced to seedlings of *P. strobus* imported back to their native land from nurseries in Germany and France. This disastrous maneuver developed because the seedlings could be grown much more cheaply in Europe than in the U.S.! Infections on the Pacific Coast from similar imported *P. strobus* from France in 1910 started the disease cycle on *P. monticola* in forests of British Columbia, Washington and Oregon that finally reached California in 1936.

Our Forest Pathology team scouting in 1937 was in the northern Sierras in the midst of an aerial onslaught of aeciospores from infected white pine (*Pinus monticola*) in southern Oregon. In 1937, we found infection on *Ribes* leaves in the Montgomery Creek drainage of the Sierras, east of Redding, over 150 miles south of known infections on Oregon western white pine. This was part of a vivid demonstration of the efficient aerial attack by the aeciospores of that classic dioecious fungus, *Cronartium ribicola*, as it traveled rapidly to the southern limits of the white pine forests of the Sierra Nevada mountains some 400 miles farther south. Blister rust provides a significant example of the difficulty in controlling a fungal disease of this type with aeciospores from its multiple arsenal of spores floating through the air with the greatest of ease, and utilizing two hosts as it outwits the weary *Ribes* eradicators and inexorably moves south.

DUTCH ELM DISEASE — CONNECTICUT

A few years later I moved to the Connecticut Agricultural Station in New Haven to work with one of the great figures in plant pathology in the 20th century, James G. Horsfall. There I was involved in studies of another devastating forest and shade tree pathogen, *Ophiostoma ulmi* (syn. *Ceratocystis ulmi*), cause of the Dutch elm disease. At that critical time (1940) the disease was spreading like wildfire into the magnificent elm trees of New England.

The disease had been found in New York only ten years previously and was spreading rapidly, not only into New England but to the north and west, facilitated by its bark beetle vectors, *Scolytus multistriatus* and *Hylurgopinus rufipes*. It was obviously a threat to millions of valuable elms throughout the nation. No American tree has a wider range than the American elm, embracing the entire area east of the Great Plains except for southern Florida. Vivid memories of the destruction wrought by chestnut blight, first found in New York in 1904, roused fear that this new disease would similarly wipe out the American elms. It was soon apparent that the American elm (*Ulmus ulmi*) is the most susceptible of any elm species to this Dutch elm disease pathogen.

The initial emphasis in New York and New England on control was aimed at total eradication. Millions of dollars were spent on this approach in the early years, rather than concentrating on research on control. By 1940, it was obvious that quarantine measures were not working, and they were finally abandoned.

Chemotherapy

In the new position at New Haven we studied many aspects of Dutch elm disease, especially development, spread, and control (13). There I had my first exposure to two of the three major "C" words in my career: Chemotherapy and Chelation. Dr. Horsfall, with characteristic energy, intuition, and foresight, initiated the first chemotherapy project on tree diseases in the United States in 1940. I was the investigator with him and found the unique project fascinating but frustrating.

This early chemotherapy research involved primarily American elm seedlings planted in rows at the Experimental Station's farm in Mt. Carmel, Connecticut. Most of the early tests were by trunk injection, with a single hole bored into the trunks of the 1- to 3-m tall (4 to 8 ft) elm trees near the base and a container with test chemicals suspended by

tubing above the injection point. For containers we used inverted 250 ml flasks for early trials, but soon found a more satisfactory and cheaper container: a Cremo Ale bottle or can produced in quantity by a local New Haven brewery. The ale was of only modest quality but the containers were superb. This initiated our unique "Cremotherapy" experiments. When we expanded our "Cremotherapy" tests to New Haven street trees or woodlot elms, some of the local citizenry questioned our sanity upon seeing elm trees with a number of Cremo Ale cans attached to their trunks.

For our tests we selected 97 chemicals of very different types. They were each tested in four different types of experiments, for their effect on (a) the pathogen in agar and in nutrient solution, (b) the healthy tree, (c) toxin production by the fungus in vitro, and (d) disease development when injected into the tree before (immunization) and after (therapy) inoculation (plus some tests by soil application) (13). Results varied somewhat in the different categories. For the bottom line, the chemicals most effective in immunization and therapy were 8-hydroxyquinoline sulfate, hydroquinone, p-nitrophenol, and benzoic acid.

Results with injection of larger trees were not as definite, though in some cases retardation of disease was obtained with the same chemicals effective with smaller trees. Difficulties with the larger elms were the result of: inadequate distribution of chemicals by the current methods of treatment; plugging of the vascular system with gums and tyloses by the time the disease was noted; and, most importantly, the lack of effective systemic chemicals in the 1940s. We were 20 years too early for the truly systemic fungicides of the 1960s, 1970s, and 1980s.

In an early epidemiological study we assayed the progress of the pathogen in mature woodlots of Connecticut. Plots were established around single, isolated, diseased trees, and the number of infections spread out from these trees was determined over a two-year period. The incidence of disease decreased rapidly as distance from the original source increased. The relationship was linear, showing that the probability of infection decreased with the logarithm of the distance of the distance from the source of the inoculum. It was soon evident that distance was acting as a dosage factor in the spread of the fungus and the beetle vector (13).

Chelation

Another very satisfying aspect of my research career in Connecticut involved a second "C" word, *chelation*. Looking through the literature in the Yale Science Library one day, the idea occurred to me that various chelating agents, 8-hydroxyquinoline for example, that inhibit metal catalysis, might have an effect on fungi by virtue of their chelating ability. Laboratory experiments confirmed this theory-chemicals that chelated metals also reduce growth and sporulation of several fungi, and adding metals, e.g. zinc, to the system counteracted this effect.

It was also demonstrated that at lower pH levels metal complex formation does not take place with 8-hydroxyquinoline, and growth of the test fungus (in this case, a *Penicillium* sp.) was normal at low pH, in the presence of an otherwise inhibitory concentration of the chemical. As further corroboration of the theory, several other chelating agents had fungistatic activity, including EDTA (ethylene-diaminetetraacetic acid), TMTD (tetramethylthiuram disulfide), and Cupferon (ammonium nitrosophenyl-hydroxylamine).

Thus, the chelation theory was born, before chelation was in vogue in the biological and agricultural sciences. This research was published in *Phytopathology* in 1943 and *Science* in 1944 (12), with the conclusion: "The evidence strongly supports the view that 8-hydroxyquinoline owes its fungi-static action to the phenomenon of forming inner complex salts with metal ions, and thus rendering them unavailable to microorganisms." In 1944, Adrien Albert in Australia independently came to the same conclusion regarding chelation and bacteria.

PHYTOPHTHORA ROOT ROT: CALIFORNIA

In 1944, an intriguing position became available at the University of California Citrus Experiment Station in Riverside after the death of William T. Horne. Professor Horne had attained an international reputation for his research on diseases of the avocado and other subtropical crops. The four years at New Haven had provided a very positive and stimulating experience for me, and the Riverside position seemed to offer a new and challenging opportunity. This did develop very quickly into a fascinating field of research on the genus *Phytophthora*, with emphasis on another devastating pathogen (*P. cinnamomi*) and a unique host, the avocado, *Persea americana*.

In 1944, a disease known as "decline," "water injury," and "melanorhiza" was causing alarm in the avocado groves of southern California. *Phytophthora cinnamomi* had recently been isolated from avocado roots in California and was emerging as a prime suspect in this disease syndrome. This pathogen, relatively unknown at the time, had been described in 1922 by USDA pathologist R. D. Rands in Sumatra (10) as the cause of a stripe canker of cinnamon trees (*Cinnamomum burmanii*). Since then this fungus has made its way into or been described from approximately 70 countries on over 1000 hosts.

It soon became evident that the California avocado "decline" was indeed a *Phytophthora* root rot problem, with a severe rot of feeder roots under excess moisture conditions in the soil, in the presence of *P. cinnamomi*. This became a major threat to the California avocado industry, and to avocados in many countries.

My research at Riverside since 1944 has emphasized *Phytophthora*, especially *P. cinnamomi*, and several other species of *Phytophthora*, including *P. palmivora* on cacao (*Theobroma cacao*), other diseases of avocado, and diseases of other subtropical and tropical woody crops. An APS Monograph, "*Phytophthora cinnamomi* and the Diseases It Causes" (16) covered much of the *Phytophthora* research on such aspects as the biology, life cycle, origin of the pathogen, taxonomy, sporulation factors, physiology, environmental influences, basis for pathogenesis, prevention of disease, and disease control by means of fungicides, biological control, resistance, and integrated control.

Chemotaxis

The research at Riverside brought another "C" word and two "P" words to the fore as cornerstones of my research: *Chemotaxis*, and *Phytophthora* and *Persea*.

In regard to chemotaxis, in the late 1950s, my laboratory tests were aimed at developing a rapid screen for avocado resistance to *P. cinnamomi* and to learn more about the infection process—including inoculation of seedling stems, small roots, and

leaves, which yielded nothing positive. Another test involved small excised feeder roots placed in a petri dish containing actively swimming zoospores. This was an instant success. An almost instantaneous attraction of zoospores occurred to the vigorous white roots (which do not have root hairs). Attraction was striking to the region of elongation; this was chemotaxis at its best! (14) The zoospores soon encysted on the root surface, germinated, and penetrated the roots, and within 24 hours a brown lesion appeared in the region of elongation, identical to lesions observed in infection of intact plants. Positive attraction of the germ tubes of zoospores to the avocado roots was also evident in spores that settled to the bottom of the petri dish. As another aspect of the infection process, we found that aspartic and glutamic acid are the primary chemicals involved in root exudates that attract zoospores to roots. As an aside, we postulated that saturating the soil with various amounts of the attractive amino acids might counteract the infection process by confusing the zoospores. This was not effective, however.

This active chemotaxis and chemotropism of zoospores of *P. cinnamomi* was reported in 1961 (14). Some initial evidence of selective attraction of zoospores to different plants and varieties of the avocado occurred; however, this did not develop into a rapid screen for resistance. Variation between roots of the same variety of avocado was found to be substantial, depending on the vigor of the root and the amount of root exudate. Some research in Australia and other locations is now reviving this chemotaxis test as a possible indication of resistance.

Goode (2), in the late 1950s, first indicated the occurrence of chemotaxis in *Phytophthora*, in an interesting study on *P. fragariae*. Our work with *P. cinnamomi* and the avocado corroborated this and provided more information on chemotaxis as one of the principal features of the infection process with *Phytophthora*. Chemotaxis was the subject of much research with zoospores on *Phytophthora* and *Pythium* at several laboratories in the 1960s. Many different species of *Phytophthora* and a variety of crops were studied. Reactions in general were similar to those noted above with *P. cinnamomi*; but there were few indications of specificity of the reaction.

Resistant Rootstocks

In 1944 nothing was known about the susceptibility to *P. cinnamomi* of various rootstocks in use in California. In 1946 I began the search for root rot resistance that has developed into a comprehensive international project involving collections not only in California but in 25 countries largely in Latin America. I became an agricultural explorer/ botanist, which led to a fascinating nearly 50-year saga of exploring Latin America and collecting varieties of avocado and species of *Persea* related to the avocado indigenous in many countries of this hemisphere, as well as researching other diseases of avocado and other subtropical and tropical trees (cacao, coffee, Cinchona, Grevillea, for example).

The avocado (*Persea americana*) is an evergreen subtropical tree producing a fruit that is a unique and nutritious contribution of the Americas to the world's food supply. The tree is native from Mexico through Central America and into northern South America; the fruit has been known to natives of this area for centuries as a valuable food source. Aztec pictographs depict the avocado tree, and the Aztec name, *ahuacaquahuitl*, is the

forerunner of our common English name. The Spaniards (after the conquest) shortened the Aztec name to *ahuacate* or *aguacate*. The remarkable Inca civilization in South America knew this fruit, as evidenced by artwork on their pottery.

The avocado has a remarkably short history as a cultivated crop. It was imported into Florida in 1833 and into California in 1856, but interest in this strange new fruit was minimal for many years. The avocado industry worldwide is now sizeable, considering that it is a specialty crop recently developed from a tree in Mexico. The large California industry originally developed primarily from an attractive and savory green fruit (named the Fuerte in California) collected in 1911 for F. O. Popenoe's nursery in Altadena, California. World production is over 3 billion pounds, and it is a multimillion dollar industry. In California the 1990 value of the avocado crop was nearly \$240 million, from approximately 70,000 acres of avocado trees. Mexico is now the leading producer worldwide, with the U.S. (largely California) second, followed by some 60 other countries. Other subtropical or tropical producers include the Dominican Republic, Brazil, South Africa, Australia, Spain, and Israel.

TROPICAL EXPLORATION The genus *Persea* (family Lauraceae) is largely native to the Americas, with approximately 80 species. The avocado is the most important member of the genus commercially. The search for a rootstock resistant to *Phytophthora* root rot began in the 1940s in the herbarium of the New York Botanical Garden in the Bronx. There I took detailed notes from their excellent collection of dried plant specimens of *Persea* from Latin America. Fortunately, a gracious botanist, Caroline Allen, an expert on *Persea* and the Lauraceae, was present on my first visit to the herbarium and was very helpful in relation to my subsequent field trips, as was later Lucille Kopp, who developed a Monograph on the genus *Persea* in 1966 (6). We used the Kopp Monograph extensively in later years.

I made similar visits to herbaria in other major botanical gardens and institutions in the U.S.: Chicago Natural History Museum, California Academy of Sciences, Gray Herbarium, Missouri Botanical Gardens, University of California, Berkeley, and the U.S. National Arboretum in Washington., D.C. Herbaria in other countries visited for this purpose were; Kew Herbarium in Kew Gardens, England; Escuela Agricola Panamericana in Honduras; and herbaria at the University of Costa Rica and the Costa Rica Museum.

Armed with these records of past *Persea* and avocado collections, I began to visit possible collection sites in Latin America, beginning with Mexico in 1951; Costa Rica, Honduras, and Guatemala in 1952; Guatemala, El Salvador, and Costa Rica in 1953, etc. The search has continued with many Latin American trips over the next 40 years up to an October 1993 trip to Costa Rica searching for an elusive Aguacate de Anis, one of Wilson Popenoe's early exotic collections there in the 1920s.

This project has involved nearly every country in Central and South America and several in the Caribbean. I have had fine cooperation from many scientists and native inhabitants of these Latin American countries. Collections have been made in diverse locations—cloud forests, volcanic slopes, rain forests, swamps, montane highlands, and hot humid lowlands—involving some spectacular scenery and some remarkable forms

of plant, animal, insect, and human life. The travels have involved many different forms of transportation, from the old reliable DC3 to dugout canoes to Boeing 747s.

WILSON POPENOE One remarkable man was the inspiration for much of my avocado exploration and collecting in Latin America: Wilson Popenoe, noted, respected, and beloved agricultural explorer, horticulturist, educator, and administrator over 60 years in Latin America. His first exposure to the avocado was in his father F. O. Popenoe's nursery in California that imported the Fuerte avocado from Mexico in 1911 and initiated the California avocado industry. He began collecting in 1915, searching for superior edible fruit for the U.S. Office of Plant Introduction with David Fairchild. Many of Popenoe's early dried avocado specimens are in the herbarium of the National Arboretum in Washington, B.C., and have been the basis for some of our subsequent rootstock collections.

My first encounter with Wilson Popenoe was in 1952 on visiting the Escuela Agrícola Panamericana, south of Tegucigalpa, Honduras. He had founded this excellent agricultural school in the 1940s, funded by the United Fruit Co., and was the Director for 20 years. A pleasant evening in his Spanish style house initiated the first plans for *Persea* and avocado collections, which began the next morning. We hiked up Mt. Uyuca, a volcanic mountain near the Escuela, and collected fruit of a new species of *Persea*. This was from a gigantic tree recently described by Wilson and Louis Williams, botanist at the Escuela. This wonderful and profitable relationship continued for over 20 years, with Wilson's well-considered advice in the following years, especially on long evenings before the fire in his famous Spanish colonial residence, the "House in Antigua", Guatemala.

The search for a resistant rootstock has involved several approaches, in California as well as Latin America—testing many different seedling types, recovering the rootstocks from apparently healthy trees growing in soils infested with *P. cinnamomi*, and collecting a wide variety of germ plasm from Latin America, including wild and semicultivated avocado trees and variants. Other species of *Persea* were also collected, as well as five other genera in the Lauraceae: *Aiouea*, *Beilschmedia*, *Nectranda*, *Ocotea*, and *Phoebe*.

This program has resulted in collections of nineteen species of *Persea* from eighteen Latin American countries, in hundreds of collections of the avocado and its varieties and variants from Latin America and California, and the five other genera in the Lauraceae. For the first 20 years of the project I was the principal collector. Since 1971, Eugenio Schieber, a very knowledgeable plant pathologist, botanist, and fine friend in Guatemala, has been of great assistance with many of the collections, including several new species of *Persea*. His knowledge of Guatemala and several other Central American countries has been priceless.

ORIGIN OF *PHYTOPHTHORA CINNAMOMI*

Another aspect of research at Riverside concerns the origin of species of *Phytophthora*, especially *P. cinnamomi*. In the course of the collections of avocado rootstock material described above, root and soil samples were also collected from trees in undisturbed soil in indigenous situations in Latin America as well as trees under cultivation. *P.*

cinnamomi has not been isolated from any of this indigenous material. Similar samples were also taken from avocado trees in commercial groves or small plantings in Latin America, some of them affected with root rot. Nearly 400 samples have been collected from indigenous and cultivated groves from Argentina, Brazil, Chile, Columbia, Cuba, Costa Rica, Ecuador, El Salvador, Guatemala, Haiti, Honduras, Mexico, Peru, Puerto Rico, St. Croix, Trinidad, and Venezuela. *P. cinnamomi* was isolated from avocado trees in twelve of these countries, but only from cultivated groves (15).

Similar samples were taken from host trees for *P. cinnamomi* in undisturbed locations along the Pacific Coast in the United States, in California, Oregon, and Washington, from Coast redwood, Douglas fir, and Monterey Pine. None of these was positive for *P. cinnamomi*. Many samples were also taken from the native vegetation in southern California, in areas above any avocado or other cultivation. *P. cinnamomi* has not been recovered from any of these samples, which were taken from moist areas.

To date, based on these samples, it seems unlikely that *P. cinnamomi* is indigenous to the Americas (15). There are some indications of an Asian origin, and also possibly of a South African origin for the A1 mating type. Von Broembsen (11) isolated the A1 type of *P. cinnamomi* from streams draining undisturbed mountainous areas in the southern Cape Province of South Africa.

The matter of origin has considerable practical significance, for example, if an area in which a host susceptible to a pathogen (*i.e.*, *P. cinnamomi*) is to be planted is free of the pathogen, every effort should be made to exclude the fungus by using clean nursery stock and taking every precaution to prevent any entrance of the pathogen into the area. Also, areas of origin of a crop should be fruitful areas in which to search for resistance to a pathogen of that crop.

Dr. Zentmyer's published retrospective moves from this point to discussions of potato late blight disease in Mexico, Jarrah dieback of eucalyptus forests in Australia, and black pod of cacao—all three diseases attributable to Phytophthora species. To conserve space those discussions have been excised in this abridgement, as has an equally informative section dealing with international agricultural research centers.

EPILOGUE

It is an awesome and challenging task to try to summarize nearly 60 years of research and academic life into a meaningful prefatory chapter. This is a summation of research on world epidemics of tree diseases of varied types, and on other miscellaneous disease problems and interesting international projects that have attracted and captivated me over the years. It has been a privilege to be involved in the wondrous field of plant pathology, one of the most challenging and satisfying fields in agricultural sciences. My deep appreciation to many wonderful colleagues and friends all over the world who have helped me to make my career one of continual interest, inspiration, satisfaction, and fun! Thank you all.

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