Lee M. Hutchins Award

The Lee M. Hutchins Fund was established in 1979 by means of gifts from the estate of Dr. Lee M. Hutchins. The award, consisting of a certificate and income from the invested funds, is made for the best contribution to basic or applied research on diseases of perennial fruit plants (tree fruits, tree nuts, small fruits, and grapes, including tropical fruits but excluding vegetables). The results of the research must have been published in an official journal of the Society.

Gaylord I. Mink

Gaylord I. Mink was born in Lafayette, IN, September 23, 1931. He earned a B.Sc. degree in 1956, M.Sc. degree in 1959, and a Ph.D. degree in 1962 at Purdue University. He was appointed assistant professor and remained at Purdue until August 1962 at which time he began his research post at the Irrigated Agriculture Research and Extension Center, Prosser, WA, with an academic appointment in the Department of Plant Pathology, Washington State University, Pullman. He was appointed professor of plant pathology and plant pathologist at the university in 1973. He maintains strong ties with the tree fruit industry, serving on many state, regional, national, and international committees and working groups concerned with tree fruit virus research.

Dr. Mink is honored with the Lee M. Hutchins Award for his research on the etiology, epidemiology, and control of cherry rugose mosaic disease of *Prunus* spp. caused by various strains of prunus necrotic ringspot virus (PNRSV). Important contributions from his long-standing research program on tree fruit viruses have been published since 1959. The major publications on cherry rugose mosaic disease were published in 1980, 1982, 1984, 1987, and 1988 (*Plant Disease* 64:691-694, 68:207-210, 68:378-381, 71:91-93, 72:636-640; *Phytopathology* 72:1542-1545, 74:1320-1324).

PNRSV causes virus diseases in many perennial plants, particularly *Prunus*. The virus, which is distributed worldwide, consists of many strains and is naturally transmitted by seed and pollen, the latter playing a significant role in intraorchard spread. One of the most severely affected areas of sweet cherry production is the Pacific Coast region of North America (southern British Columbia, Washington, Oregon, and northern California). Here, sweet cherry trees often exhibit symptoms of cherry rugose mosaic disease caused by strains of PNRSV. Since the principle symptom is failure of the fruit to ripen and infected trees do not recover, trees must be removed. However, control of the disease by tree removal is impossible unless infected trees can be identified and removed before they flower and produce infectious pollen. Mink demonstrated in 1980 that a serological assay (ELISA) of dormant flower buds during the winter months was superior to testing of summer leaf tissues and that rugose mosaic-diseased trees could be distinguished from those infected with ordinary PNRSV strains by relative absorbance in ELISA, thus providing a method of identifying rugose mosaic-infected trees for removal before the trees bloomed.

A consequence of the dormant tissue testing was the establishment of a self-supporting ELISA laboratory at Prosser, which annually tests samples of cherry for PNRSV and other viruses on a fee basis for growers in several western states. ELISA testing has been extended to annual testing for PNRSV and prune dwarf virus (PDV) of more than 10,000 registered *Prunus* seed and scion source trees maintained by certified fruit tree nurseries in Washington, Idaho, Montana, and California. An important observation by Dr. Mink and his colleagues culminated in a paper published in 1987 that demonstrated that three different serotypes of PNRSV could be isolated from trees exhibiting identical rugose mosaic symptoms. Moreover, a symptomless biotype of PNRSV was identified in some Washington orchards, which was serologically identical to many disease-causing strains. This symptomless biotype was found to be associated with decreased spread of rugose mosaic disease. Inoculation of this symptomless biotype to cherry prevented subsequent infection or disease expression by rugose mosaic biotypes. A recent publication suggests that cross protection can provide effective control against many of the PNRSV strains that induce rugose mosaic disease.

Significant contributions have been made by Dr. Mink in other aspects of tree fruit research. He and his colleagues have studied the presence of infective PNRSV and PDV on sweet cherry pollen taken from commercial beehives. They were the first to demonstrate that these viruses, while readily detected by ELISA on pollen collected shortly after deposition in beehives, could not be detected in the same hives after 7–8 weeks. A factor(s) that degraded these viruses, probably an enzyme, was isolated and partially characterized from bee-collected pollen but not from pollen collected by hand. Results that bees, emerging from hives transported from southern California to Washington, contain viable, virus-contaminated pollen on their bodies suggested that foraging bees from commercial hives may be involved in long-range distribution of PNRSV and PDV as well as in intraorchard spread.