Critical studies of severe shot hole symptoms this year revealed for the first time, spur and leaf blighting on diseased trees. Also, examination of diseased leaves, fruits and twigs revealed the presence of shot hole spores and new infections throughout the season on nonsprayed trees.

Brown Rot: Brown rot blossom blight has been effectively controlled with benzimidazole (Benlate and Topsin) sprays during bloom. However, resistant M. laxa was found in apricots in San Benito and Contra Costa Counties. M. Laxa is important on almonds because it causes brown rot blossom blight. But, at this time, resistant M. laxa has not been found on almonds. This is based on an extensive survey conducted in 1982 prior to almond harvest and also less intensive surveys conducted in 1980 and 1981.

In the 1982 survey, a high percentage of M. fruiticola isolated from almond hulls did exhibit resistance to these fungicides. M. fructicola is the second most common cause of almond hull rot and Rhizopus is the most common. M. fructicola rarely causes brown rot blossom blight on almonds. Accordingly, the fact that resistant strains of this fungi have been found in almonds does not affect current control of brown rot blossom blight with Benlate or Topsin.

Investigations on the fitness, or activity, of resistant M. laxa found in apricots are currently underway and indicate that these strains are less fit. Results show that spores from the resistant strains have reduced germination percentages and rates when compared to sensitive strains. Pathogenicity tests conducted on unopened almond blossoms show that higher concentrations of resistant M. laxa spores are required for infection than with sensitive spores.

Continued monitoring for potential benomyl-resistant M. laxa populations in almonds along with results obtained from fitness studies could act as tools in extending the effective use of Benlate and Topsin sprays in controlling brown rot blossom blight of almonds. In addition, work continues on developing alternate fungicides, in the event resistance develops.

Hull Rot: Nitidulid beetles and other insects have been implicated in vectoring the causal agents of the hull rot disease. These insects visit the initially infected hulls (early July) and carry the spores of Rhizopus and Monilinia to newly splitting almond hulls. This process continues during the hull split period and those orchards with the longest hull split period show the greatest disease problem. Spraying an insecticide such as dichlorvos during the hull split period has been planned.

Project No. 82-U2 - Almond Diseases
Ceratocystis Canker Control

Project Leader: Dr. Richard M. Bostock (916) 752-0308 or 752-0301
Department of Plant Pathology
University of California
Davis CA 95616

Objectives: (1) Evaluate chemical/biological agents for protecting fresh bark injuries from infection. (2) Evaluate chemicals/biological agents for their efficacy in eradicating Ceratocystis fimbriata in diseased tissues. (3) Determine relationship between timing of irrigation and disease incidence. (4) Evaluate methods for insect vector control and impact on disease incidence. (5) Initiate basic studies on host resistance mechanisms.

Interpretive Summary: Certocystis canker is caused by the fungal pathogen, Ceratosystis fimbriata, which infects trees through mechanical injuries generated during harvesting operations. Dried fruit beetles and several other insect species are attracted to these wounds and vector the pathogen by carrying and depositing spores in the injuries. Most cankers are of a perennial nature and, if left unchecked, eventually girdle the infected limbs. The objectives of this research are to develop control procedures based on preventing infection or eradicating the pathogen in existing cankers.

Most of the research effort on Ceratocystis canker during 1982 has involved: (1) obtaining experimental materials and developing appropriate methods for studying the disease, (2) evaluating the rate of wound resistance development and (3) conducting preliminary tests with biological and chemical agents for their efficacy in disease control.

In all field experiments, the bark is artificially injured and then the diseased wood is pruned out at the end of the experiment. The natural infection process is mimicked by first contaminating dried fruit beetles with spores of the fungus and then caging them over a bark wound made with a cork borer. Using this method, it was found that injuries in Nonpariel become resistant to infection eight to ten days after injury. This establishes how long a preventive treatment must be effective before the natural resistance mechanisms of the tree can take over. Plans are to expand upon this and determine the variation in wound resistance development among different varieties and during different times of the year.

Other research effort this year has been directed at studies with biological and chemical agents as protectants and eradicants. Four non-pathogenic species from the fungal genus, Trichoderma, were antagonistic to eight isolates of Ceratocystis fimbriata in culture. However, they did not appear to protect injuries from infection in field experiments. Experiments to eradicate the fungus from existing cankers using chemicals were inconclusive. However, further research will continue to explore these approaches.

The only recommendation for canker control at this time is avoid shaker injury to trunks and scaffolds.