ANNUAL REPORT, ALMOND RESEARCH, DECEMBER 31, 1990

Project No. 90-X4 - Continued investigations on almond brownline and stem grooving disorders

Project Leaders: Drs. W. D. Gubler and J. K. Uyemoto
Cooperative Extension and USDA-ARS, respectively, Department of Plant Pathology, University of California, Davis, 95616. All correspondence should be directed to JKU. (916) 752-0309.

Cooperating Personnel: Joe Connell, Janine Hasey, Wes Asai, Wilbur Reil

Objectives

Almond brownline (ABL): 1) Orchard plot (Live Oak). Monitor healthy Peerless and Price trees propagated on Marianna and Lovell rootstocks for incidence and spread of ABL. 2) Maintain and read indicator trees grafted in 1988 and 1989. 3) Continue studies on peach yellow leafroll (PYLR) on almond indicators; graft inoculate ABL on Fay Elberta peach trees.

Almond stem grooving (ASG): 4) Resurvey diseased orchards. 5) Maintain and examine Marianna 2624 rootstocks grafted with diseased and healthy Carmel and Butte scion buds. 6) Graft inoculate almond indicator trees with diseased inocula. 7) Determine symptomatology of almond leaf scorch on almond/Marianna trees.

Interpretive Summary

Almond brown line. The absence of disease apread among our budling orchard trees in a Live Oak site (plus other evidence presented in previous reports) suggest that ABL is not soil borne. ABL may in fact be caused by a mycoplasma. Almond stem grooving. The partial recovery of affected trees that were pruned indicate that there may be a synergistic interaction between the agents causing the union symptom and the root rot.

Experimental Procedures and Results

Almond brownline. In a Live Oak orchard (100 acres), a survey in 1987 revealed an ABL incidence of 15% in each of Carmel, Peerless, and Price/Marianna 2624 trees; at the third-leaf growth stage. By 1989, we had established a 22 replicated trials involving 2 sources of Marianna 2624 (one source was FPMS and the other a commercial nursery in Oregon) and Lovell peach roots supporting budling shoots of Peerless or Price. None showed disease symptoms in 1990. However, two first-leaf nursery replants of Peerless were displaying ABL symptoms. Both trees were removed for graft transmission tests at Davis.

Pathogenicity tests with ABL were initiated in 1988 and continued in 1989. In 1988, 18 trees of Peerless/M2624 were grafted with diseased or healthy Carmel and Peerless inocula. Then, in 1989 more almond/M2624 trees (90) were graft inoculated with additional inocula. None of these trees exhibited disease

symptoms in 1990.

Despite these setbacks, the probable causal agent of ABL was identified. During September 1989, 10 trees each of Carmel, Peerless, and Price/M2624 were grafted with peach buds collected from trees with PYLR. [This disease is caused by a mycoplasma, which is efficiently transmitted from tree-to-tree by various phloem feeding leafhoppers. Leaf symptoms in peach consists of enlarged mid-vein and primary veins.] During the following spring, all grafted almond trees developed yellowed leaves, poor shoot growth, and the trees later declined and died. The unions exhibited deep pits. These symptoms are characteristic for ABL.

If PYLR cause ABL in almond trees grown on Marianna 2624 roots, then what is the likelyhood of infection and its consequence on peach rooted trees? In 1990, we investigated that possibility when two Butte/peach trees showing sparse canopies and producing shrivelled kernels were called to our attention. The symptomatic trees were in their ninth-leaf stage of growth. [I might add that the same grower had removed a couple of Nonpareil/peach trees with shrivelled kernel symptoms in the previous year.] On June 1, two scaffolds on one of the Butte tree were infused with an antibiotic solution. Approximately 4-5 weeks later, leaves on the treated scaffolds turned yellow with necrotic margins, a normal phytotoxic response to the antibiotic. After a couple more months, renewed shoot growth occurred; these averaged 4-6 inches in length. No new growth was evident on the untreated trees. On healthy Butte trees, nearly 2 feet of shoot growth was evident. Also, from May to harvest in September, the kernel developement was monitored. No apparent differences were apparent through mid-July. However, by mid-September it was readily evident that the untreated trees produced all shrivelled kernels, while the treated trees had 70% plump kernels. Healthy trees produced only plump kernels. Nucleic acid extracts of leaf petioles and roots were also done. These preparations were spotted on a nylon membrane and challenged with a radioactive-labelled probes specific for PYLR or cherry buckskin (another mycoplasma disease). Only the PYLR probe hybridized with the preparations.

Almond stem grooving. [A name change to union mild etch is proposed and its useage will begin in all future correspondence. Stem grooving was originally used to describe trees that exhibited longitudinal grooves extending well above and below the union. These were observed in a few trees in the first orchard we had visited in 1987. During the next 3 seasons, however, a different union symptom was more consistently observed on trees exhibiting a similar canopy growth response. The affected unions contained only shallow, short grooves or mild pits, which were occasionally accompanied by necrosis. These symptoms are best seen during late fall. None of the recent trees showed the stem grooving symptoms, which we now suspect may have been an artifact.]

Repeated surveys in orchards located in Esparto and Modesto (in 1988 both had contained ca. 20% diseased Carmel trees and 1% Price trees) produced no new finds. In the Chico orchard, however, and which had 14% diseased Carmel and 4% Butte trees (all but 6 trees were removed and replaced), there were another 7% diseased Butte trees. This was likely a carry over, i.e. trees infected but not expressing recognized symptoms last year, and not evidence of field spread. It is noteworthy to mention that among the trees removed, 40% of them exhibited

rotted roots. Also, during 1990, additional orchards were surveyed. Two Carmel orchards with diseased trees included a third-leaf near Durham with a 27% incidence and a second-leaf near Riverbend with 21%. And a second-leaf Mission planting near Live Oak showed a diseased incidence of 67%.

All graft inoculated indicator trees with diseased buds appeared normal. Attempts to establish infections with almond leaf scorch on almond/M2624 trees failed.

Discussion

Almond brownline. Initially and based on the spatial pattern of diseased trees, a soil borne cause was suspected (see annual report 1987). Later, however, results of our propagation experiments indicated that diseased trees were produced using infected scion buds grafted onto Marianna 2624 understocks (see annual report 1988). Also, yearly surveys done in the Live Oak site (with a 15% disease incidence) showed a decline in numbers of symptomatic trees due to the rigorous tree replacement program (see annual report 1989). During 1990, only two new ABL trees were found and these were recent replants (nursery source).

With kernel shrivel disease, the positive response to an antibiotic and hybridization with a nucleic acid probe specific for PYLR suggests a causal relationship. Learning of these results, the grower promptly removed the diseased trees. However, prior to their removal, diseased limbs were collected and these were used to chip bud into indicator trees at UC Davis. We hope these graft transmission attempts prove successful to continue our studies.

Almond stem grooving. For the most part and due to our failure to transmit the disease agent from diseased orchard trees to healthy almond trees in our nursery, very little is known about the disease. However, some affected trees appear to recover when pruned severely, while others have died. There was evidence of root rot on 40% of the declining trees. It appears that there may be a synergistic interaction caused on the one hand by the union symptoms that imparts stress on the Marianna portion of affected trees and thereby permitting a secondary organism to invade and cause root rotting. We plan to look into this phenomenon.

Also, since PYLR-mycoplasma can induce ABL, we will examine the effects of another biologically distinct mycoplasma disease called cherry buckskin and its relationship to almond stem grooving disease. On peach, buckskin differs from PYLR in that it causes reddening of leaves during mid-summer, tissue chlorosis and necrosis. The necrotic tissues fall out leaving a tattered leaf appearance. The buckskin-mycoplasma does not cause leaf veins to enlarge. The mild etch symptoms on almond trees may be a reflection of a less severe mycoplasma strain.

J.K. UYEMOTO, USDA-ARS
Dept. Plant Pathology
University of California
Davis, CA 95616
752-0309

December 14, 1990

Ms. Susan P. McCloud
Research Director,
Almond Board of California
1900 Point West Way, Suite 111
Sacramento, CA 95815

RECEIVED
DEC 19 1990
ALMOND BOARD

Dear Susan:

Please find enclosed two copies of our annual report on union disorders of almond trees. We were fortunate in developing an important lead on the cause of almond brown line (ABL) disease. Also, the same or closely related mycoplasma was detected in almond trees grown on peach roots. The latter was expected. This is because we had previously demonstrated that diseased scion buds could produce budling trees with ABL, i.e. the same budwood source trees would be used to propagate almond cultivars on peach seedling. The disease incidence on peach seedlings may be a high as 15% in some orchards.

If you have any questions, please contact me.

Sincerely yours,

Jerry Uyemoto
Research Plant Pathologist

pc Douglas Gubler