ARMILLARIA ROOT ROT IN PEACHES IS CONTROLLED BY METHYL BROMIDE

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Abstract. Armillaria root rot, probably incited by Armillaria tabescens (Scop.) Dennis, Orton and Hara, but possibly by A. mellea (Vahl.: Fr.) Kummer, was prevented from recurring in peach [Prunus persica (L.) Batsch.] replants, for 3 to 5 years following 1 or 2 lbs of methyl bromide per 100 sq ft soil surface treatment under plastic. Treatment was made following removal of roots over ½ inch diameter to a depth of 12 inches and in October when soil was dry. Replants set the following January were budded trees of various genotypes on either Okinawa or Flor-daguard peach seedling rootstocks.

Armillaria root rot (ARR), known as oak root fungus, oak root rot, or mushroom root rot is found throughout the United States. The northern and western phase is incited primarily by A. mellea and the southeastern phase is incited primarily by A. tabescens. The presence of these fungi overlap in the southeastern U.S. and symptoms are similar with both showing white to cream strands of the fungus in and under the bark near the ground line. The two fungi are said to be separated visually by A. mellea frequently having dark brown to black strands (rhizomorphs) along the surface of diseased roots (Philip and Davis, 1936), but separating symptoms were not clearly evident to us after field demonstration by Pathologist C. N. Clayton in a North Carolina orchard nearly 30 years ago. Furthermore, A. mellea, unlike A. tabescens, has an annulus on the mushrooms, but these fruiting bodies are not usually present in our peach evaluation plots the first year after dying when trees are removed.

Both fungi kill peach trees in similar fashion, show similar symptoms, and have similar hosts and recommendations for control (Philip and Davis, 1936; Sharpe, 1966; West, 1949). All commercial peach rootstocks have been rated as susceptible for A. mellea including Lovell, Nemaguard, Flordaguard, and Okinawa (Beckman et al., 1998; Norton et al., 1963) and no resistance in commercial peach stocks in north Florida to central Georgia is known for A. tabescens. In fact, so many different kinds of susceptible plants have been reported as infected that probably no woody plant is immune. However, some species appear tolerant (West, 1949), but peaches are extremely susceptible (Thornton, 1940). Control measures for ARR include avoiding land that had red oaks (Philip and Davis, 1936; Sharpe, 1966) just prior to clearing and planting (especially turkey oak and laurel oak—Quercus laevis Walt. and Q. laurifolia Mich x., respectively), fallowing land for a minimum of 3 to 5 years (Rhodes, 1950; West, 1949), or removal of infected roots followed by fumigation (Philip and Davis, 1936; West, 1949). Furthermore, it has been observed in north Florida and Georgia that peaches following tung nut (Aleurites Fordii) and pecan (Carya illinoinensis) usually suffer high mortality with in 2-3 years indicating these two crops are resistant carriers.

Fumigation with carbon disulfide has been used in California with good success for controlling oak root fungus in drier sandy soils, especially when combined with removal of infected roots, but not in heavy wet soils (Philip and Davis, 1936). Fumigation of infected sites with methyl bromide in clay soils at Griffin, Georgia did not control oak root fungus (J. W. Daniell, pers. comm.), but the high moisture retention of the fumigant may have prevented deep penetration of the fumigant. The purpose of our research was to determine if methyl bromide could control oak root fungus in sandy soils if applied in the dry autumn season.

Materials and Methods

Peach seedlings from the University of Florida variety breeding program were planted in the mid 1960s onto newly cleared land on which a mixture of oak and pine grew. Seedling trees succumbed in a scattered pattern to ARR over the following years and when all seedlings were evaluated the land was replanted with new seedlings until 1976. Identification of ARR was made by cutting into bark near the ground line to visually show mycelial strands/mats of the fungus be-
neath the bark. Usually there was a 50% or greater loss of these replant trees in the second or third year in the general area where previous trees had died from ARR.

Grafted trees were replanted in 1976 from selected genotypes for variety evaluation and spaced 10 feet apart within rows 20 feet apart. An orchard map was begun to record each tree by row location where losses to ARR occurred in the future. Trees were fruited and removed upon the decision to discard the genotype (usually in year 3 to 5), and trees dying from ARR were recorded and also removed. In 1983, at each location where a tree had died an area 10 x 10 feet was dug and roots to 1 ft depth and greater than half inch diameter were removed. These tree plots were treated in October 1984, after the rainy season had stopped and the soil had dried, with 2 lbs of methyl bromide under a 1 x 10 ft. plastic tarp. Methyl bromide was released under the tarp into an evaporation pan. Peach trees of selected genotypes from the breeding program were replanted in January 1986 and grown for five growing seasons until evaluated for fruit and tree characteristics at which time they were removed. Again, the tree losses from oak root fungus were recorded and removed during these 5 growing seasons.

In autumn 1990, we repeated the 1983-84 method of root removal and fumigation for locations where trees had died from ARR since planting in January 1986. Peach trees were again planted in January 1992, but fumigation treatment was at 1 lb methyl bromide for each 10 x 10 ft. location. Trees were grown until June 1997 when all trees were removed. Tree losses to ARR during these 5½ years were also recorded.

Results and Discussion

Grafted trees were planted in 1986 and 1992 in previous ARR infected locations following root removal and fumigation with methyl bromide at 1 or 2 lbs/100 sq ft. No locations fumigated in 1984, and replanted in January 1986, had suffered any losses to ARR by time of tree removal in July 1990. However, during that 5 year period an additional 26 trees in different locations died from ARR. Tree losses probably would have been higher, but some genotypes were discarded from the breeding program and trees pulled in June during the third and fourth growing seasons. Generally, the new tree losses were immediately adjacent in the row to the sites where a tree had died previously from ARR, indicating that infected roots had spread to the adjacent location by the time of tree death and prior to methyl bromide treatment at 2 lbs/100 sq ft. All remaining trees were pulled in June 1991 and the 26 locations where trees had died from ARR were fumigated in October at 1 lb methyl bromide/100 sq ft. The entire block was replanted in January 1992. Again, by the middle of the fifth growing season in June 1996, we had observed no tree loss to ARR where the 26 previous trees had died, but an additional 21 trees had died and again most were located adjacent to sites of previous tree losses to ARR.

In summary, we have had no repeat tree losses to ARR on sites following root removal and fumigation under tarp treatment with methyl bromide at 1 or 2 lbs/100 sq ft in dry soil in October following the rainy season. However, trees located adjacent to methyl bromide treated sites suffered the majority of subsequent losses to ARR. Thus, we conclude that specific site treatment with methyl bromide in dry sandy soils will control ARR in that site until reinoculated.

Literature Cited


