These results suggest that there is a slight adverse effect when seedlings have more than 25 heat units during the first 5 days after planting. Since the optimum temperature for germination in controlled environments is not obtained with 25 heat units, some other factor is probably responsible for the slight decrease in emergence at heat units above 25.

The National Oceanic and Atmospheric Administration (NOAA) provides a weather forecast service. The Bakersfield office has participated the last two years by providing the 5-day heat unit forecast. It is anticipated that other NOAA offices in the San Joaquin Valley will provide the same service in the future. Cotton growers will be able to make sound planting decisions by obtaining warm and cool germination percentage data on seed lots for planting, then refining planting decisions based upon quantifiable weather data.

Conclusions

If less than 10 heat units are predicted for 5 days, planting would not be recommended. If heat units are predicted to be between 11 and 15, planting should proceed only if germination testing verifies that the seed is of superior quality. If large numbers of acres are to be planted and a grower feels planting must proceed with 11 to 15 heat units, planting rates could be adjusted upward to obtain the desired plant stand. When heat units are predicted to be greater than 16, conditions are favorable for stand establishment with all but poor-quality seed lots.

Planting rates should be adjusted for the planting conditions and to a lesser extent for the quality of seed (tables 1 and 2). Seeding rate changes very little when 16 or more heat units are predicted for 5 days across all ranges of seed quality. When warm plus cool germination percentage is 140 or more, there is also very little adjustment in seeding rate for higher quality planting seed. With 14 or fewer heat units predicted during the 5 days following planting, large adjustments must be made in seeding rates, especially if the seed quality is poor.

Unless growers have no choice, it is recommended that planting seed have at least a warm plus cool germination percentage of 140. With seeds of lesser quality, plants will take longer to emerge (fig. 1), and these seedlings will be exposed to seedling diseases for a longer time. Best yields will be obtained with good-quality seed that can emerge rapidly in an environment promoting fast early growth.

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The two cherries at right show typical symptoms of buckskin disease of sweet cherries, named for the pebbly, pale color of diseased fruit. Symptoms may vary, depending on the rootstock and the strain of the disease.

Buckskin disease of cherry

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Serious sweet cherry tree losses occur in California as a result of "cherry decline," a collection of diseases caused by fungi, mycoplasma-like organisms, viruses, and other unidentified agents. A major cause of cherry decline is cherry buckskin disease, also called X-disease of cherry. Buckskin disease was first reported in 1931 on sweet cherry in California and within 20 years eliminated the sweet cherry industry in Napa and Sonoma counties. The disease now threatens cherry production in San Joaquin County.

Symptoms

The name buckskin disease derives from the pebbly, leathery-skinned, pale fruit of diseased trees. In California, two strains of buckskin, "Napa Valley" and "Green Valley," have been described in sweet cherry. Each strain produces distinct symptoms, depending on the rootstock on which the tree is grafted.

Trees grown on sweet cherry (*Prunus avium*) 'Mazzard' rootstock and having the Green Valley strain produce small-sized, conical-shaped fruit with short, thick stems. The skin of dark-colored cherry varieties may remain light in color. In addition, leaves on severely diseased trees are smaller, sparser, more yellow, and more erect than normal leaves, giving the tree a "see-through" look. New terminal growth on twigs is usually reduced or absent, and the ends of twigs or branches may die back each year. In contrast, the Napa Valley strain on sweet cherry rootstock induces small, but normal-shaped fruit with normal stem length.

Trees grown on 'Mahaleb' rootstock (*P. mahaleb*) and having either the Green Valley or the Napa Valley strain may not develop fruit symptoms but instead suddenly wilt and collapse above the graft union. This reaction is thought to be caused by very rapid killing of the rootstock cambium tissues at the graft union when they are contacted by the buckskin disease agent. The rapid death of the cambium tissue prevents the disease agent from spreading into the rootstock. When sweet cherry is grafted high on Mahaleb rootstock on several separate limbs, a single limb may become infected and die without affecting the remainder of the tree. This limb then can be removed and the remainder of the tree reworked.

Cause and spread

Buckskin appears to be caused by mycoplasma-like organisms found in the nu-

trient-conducting phloem cells of affected trees. In an electron microscope, the pathogens appear as minute tubules, spherical-shaped bodies, and oval-shaped bodies. They are similar in size and makeup to very small bacteria but lack the rigid cell walls of bacteria. Their small size and flexibility allow them to move into plant and insect tissues that would normally exclude most bacteria.

Cherry buckskin disease may be transmitted by grafting, but under field conditions it is most often spread by leafhoppers, which acquire the pathogen when they feed on diseased trees. During the following three to five weeks, the pathogen multiplies inside the insect, invades its blood system, and finally passes into the salivary glands. At this time, the pathogen can be transmitted to healthy trees by the feeding of infected leafhoppers.

Several species of leafhoppers in California can transmit the buckskin pathogen. The most abundant vector is the mountain leafhopper (*Colladonus montanus*), which feeds on numerous kinds of plants, including cherry. Cherry is not a preferred plant, however. Adult mountain leafhoppers can overwinter in sugarbeet fields. When the beet fields are harvested in late spring, the leafhoppers disperse into adjacent areas, which may include cherry orchards with patches of curly dock (*Rumex crispus*) and clover (*Medicago* and *Trifolium* spp.). Mountain leafhoppers can breed on curly dock and clovers, but not on sugarbeets. Experiments, which we conducted in 1984-85 using plants growing in greenhouses, indicate that sugarbeets and curly dock are not susceptible to infection by the disease agent. Some of the clover species can be infected, however.

The mountain leafhopper efficiently transmits the buckskin pathogens to cherry, but normally it does not feed for long or survive well on cherry. In 1978, we conducted experiments in which mountain leafhoppers were exposed to the foliage of buckskin-diseased cherry trees for one week in the field and then tested individually for their ability to transmit the buckskin agent to celery, a sensitive indicator plant. From April through most of July, there was very little transmission from cherry, but in August and September, about 20 to 25 percent of the surviving leafhoppers transmitted the disease. This is the time that the mountain leafhopper is often most abundant in cherry orchards, so control of the insect during this period is important in preventing buckskin disease spread.

A buckskin vector capable of living and even reproducing on cherry is Flor's leafhopper (*Fieberiella florii*), perhaps the most important leafhopper in central California for transmitting the buckskin disease agent from tree to tree within an orchard. Although Flor's leafhopper isn't common in cherry orchards, it has been associated with explosive outbreaks of buckskin disease near its favored breeding plants — ornamental shrubs that retain their leaves year round. These include privet (*Ligustrum* spp.), boxwood (*Buxus* spp.), firethorn (*Pyracantha* spp.), myrtle (*Myrtus communis*), and *Viburnum* spp. None of these ornamentals are considered hosts of the disease agent, but they serve as overwintering sites for immature Flor's leafhoppers. During the spring, the leafhoppers develop into adults and then may disperse in late spring through fall to nearby cherry orchards.

Control strategies

In the state of Washington and in the eastern United States, cherry buckskin disease control has centered around the removal of chokecherry, a weedy shrub that is a source of the pathogen. The sudden appearance of buckskin disease in California cherry orchards suggests that other plants are important sources of the buckskin pathogen, since chokecherry is not found near commercial cherry orchards here. Removal of diseased trees therefore appears to be an important management strategy in the control of buckskin. Diseased tree removal was an important component of the buckskin eradication program in Washington during 1948-52.

The mountain leafhopper disperses widely. It prefers to feed on herbaceous plants and survives poorly, at best, when confined to cherry trees. Its role in the spread of buckskin thus may be to introduce the disease agent into healthy orchards. Flor's leafhopper, on the other hand, can thrive and reproduce on cherry but does not migrate as extensively as the mountain leafhopper. We believe that Flor's leafhopper may be the primary vector that transmits buckskin disease from tree to tree. Because Flor's leafhopper prefers to breed on ornamental shrubs such as privet, boxwood, firethorn, myrtle, and viburnum, its populations can be reduced by the removal of nearby ornamental shrubs, timely applications of insecticides, or both.

Leafhopper populations within cherry orchards should be closely monitored with yellow sticky-board traps, since experiments conducted from 1982 through 1986 have shown a direct relationship between the number of leafhoppers trapped in an orchard and the incidence of disease. If either species of leafhopper moves into an orchard, the orchard should be treated with an effective insecticide such as Diazinon or Pydrin (fenvalerate). In 1986 leafhopper control studies, we found that Diazinon provided good immediate control but no continuing control. Pydrin provided good control for up to six weeks, but spider mite outbreaks have been observed after its use.

In summary, an effective buckskin control program should include: (1) removal of buckskin-diseased cherry trees (if trees are removed during the growing season, they should be treated first with an insecticide), (2) removal of or treatment with an insecticide any ornamental hosts of Flor's leafhopper near a cherry orchard (again, the ornamental host should be treated with an insecticide before removal), and (3) monitoring of orchards and host ornamentals for the presence of vector leafhoppers and treatment with an insecticide if one mountain leafhopper per trap per week or any Flor's leafhoppers are found.

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Buckskin disease is spread by Flor's leafhopper (above) or the mountain leafhopper (below), which pick up the pathogen when they feed on diseased trees.

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