New findings on pistachio problems

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Ailments commonly thought to be physiological have now been traced to feeding by several insect species

For many years, darkening and collapse of hull tissues and subsequent abortion of immature pistachio fruits was thought to be a physiological disorder. However, in 1983 tests, epicarp lesion symptoms were reproduced when apparently healthy pistachio fruit clusters were exposed to field-collected adult leaffooted bugs, *Leptoglossus clypealis,* for 48 hours in open-mesh cloth bags on the tree (see *California Agriculture,* March-April 1984). This discovery suggested that a number of related bugs might also be able to produce epicarp lesion symptoms.

To find out, we broadened our research emphasis in 1984, examining the chronology of the various symptoms from fruit set in April until harvest in September and studying the effects of feeding by other species of true bugs (Hemiptera). Epicarp lesion is commonly described as a sunken, discolored area with darker zones, which extends over a large portion of the fruit surface; a dried resin exudate is usually present within the lesion boundary. The disorder has also been associated with necrotic spots on the epicarp (skin) or with internal browning of the flesh and shell (mesocarp and endocarp layers).

We began the 1984 program on April 21 with a visit to a pistachio orchard that had fruit clusters with numerous blackened small fruits (less than 4 mm diameter) and typical epicarp lesion symptoms on larger fruits. The shells of affected fruits showed signs of feeding damage suggesting involvement of an insect other than the leaffooted bug.

In subsequent feeding trials in the field, the symptoms were reproduced when previously healthy fruits enclosed in sleeve cages were exposed to several species of true bugs. Examination of the fruits revealed symptomatic patterns of damage by the different species. Control fruits without insects remained healthy. Our results indicated that fruits with typical symptoms were abundant as early as April and that blackening of newly formed fruits was also a form of epicarp lesion.

We observed other fruit symptoms in the orchards, however, that were not associated with insects, such as panicle and shoot blight (necrotic spots of the epicarp), endocarp necrosis (internal browning of the flesh and shell), and stylar-end lesion. Following are descriptions of the pistachio fruit disorders, their incidence, and distribution in California orchards during the 1984 cropping season.

Insect-associated disorder

The term "epicarp lesion" is restricted to fruit symptoms induced by sucking insects. In 1984, incidence of epicarp lesion ranged from 4 to 49 percent in severely affected orchards in the Sacramento and San Joaquin valleys.

Leaffooted bugs (Hemiptera: Coreidae) **Stinkbugs** (Hemiptera: Pentatomidae)

Two species of leaffooted bugs, *Leptoglossus clypealis* Heidemann and *L. occidentalis* Heidemann, and at least four species of stinkbugs in the genera *Thyanta, Chlorochroa,* and *Acrosternum* produced similar external and internal damage to pistachio fruits. External symptoms on fruits 6 to 8 mm in diameter were subepidermal "water-soaked" or dark stained areas at the point of feeding within 48 hours after the bugs fed. As the affected area expanded, the typical necrotic epicarp lesion appeared on the surface of the fruit. These immature fruits shriveled and dropped from the nut cluster within a few days after attack. It is noteworthy to mention that, before the onset of external symptoms, tissue discoloration first developed in the flesh and later extended into the shell and out to the skin.

Internal fruit symptoms from coreid and pentatomid bug feeding were quite distinctive; these symptoms changed as the shell hardened. In immature fruits, a white netting or web pattern appeared in the inner shell tissue within 24 hours after feeding. This was sometimes accompanied by partial collapse or depression of the inner shell surface. Eventually, these fruits also developed external lesions, and most of them dropped from the tree.

As the shell of the maturing nut began to harden, the amount of internal white netting decreased. The white net pattern was sometimes still seen in fruits that had dropped from the tree but retained some green color in the dried shell. As hardening progressed and shells began to lose color, the white net symptoms disappeared and were sometimes replaced by only a small black internal spot on the shell where bug feeding occurred.

At this time, major damage symptoms from leaffooted and stinkbug attack shifted from the shell to the kernel or nutmeat. As the kernel began to enlarge, discolored spongy or punky areas appeared. The damaged tissues in these areas stopped growing, leading to sunken

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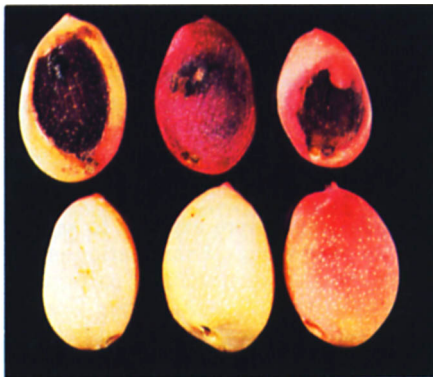
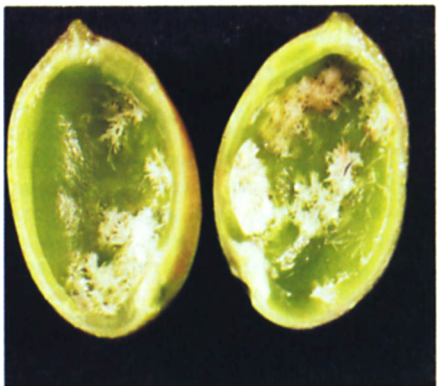
In controlled field tests, epicarp lesion symptoms were induced in pistachios within 48 hours after feeding by true bugs.

"Water-spotting" is an initial symptom of epicarp lesion on small pistachio fruits.

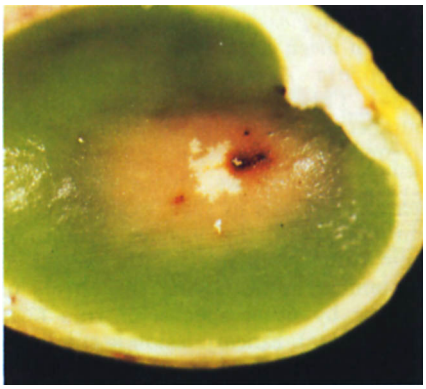
Several different sucking insects can induce external symptoms of epicarp lesion early in the season (blackened small fruit on center branch, above) as well as on more mature pistachios (left).



The leaflike development of the hind legs of adult *Leptoglossus occidentalis* gives the insect its common name, leaffooted bug.

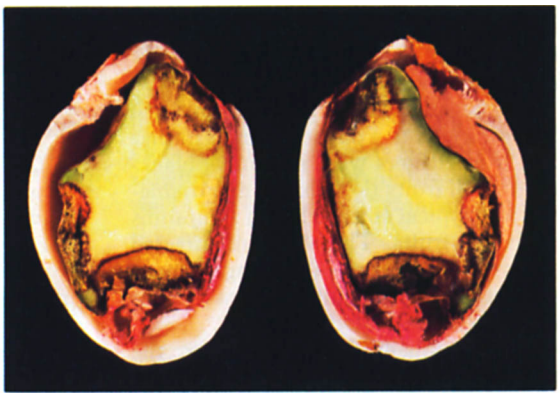


In immature fruits, a white netting or web pattern appeared on the inner shell tissue within 24 hours after insect feeding.



As shells hardened, the white net symptoms were sometimes replaced by a small black internal spot where bugs fed.

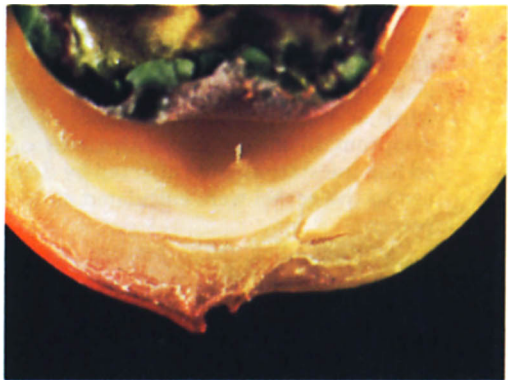
Before external symptoms of epicarp lesion appeared, insect feeding caused the middle flesh layer to become discolored within 24 hours (photo at left).



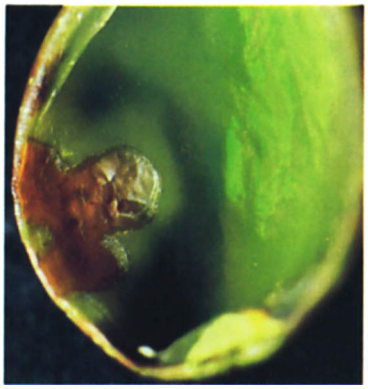
As the nutshell hardens, damage from leaffooted bug and stinkbug attack shifts to the kernel, which becomes discolored, spongy, and punky.

Tissues in areas of pistachio nut damaged by insect feeding stop growing, leading to sunken and distorted nutmeats.

Leaffooted bug feeding can cause extensive late-season damage to kernels of nuts that show no external symptoms.

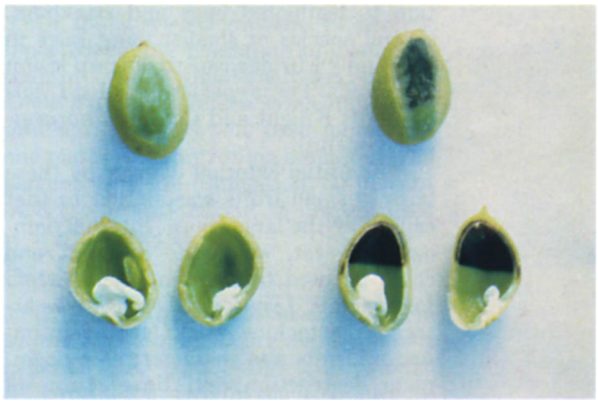


Protruding microscopic stylet sheath or "spike" is a sign of feeding by leaffooted bugs and stinkbugs.



Several species of smaller bugs, such as *Calocoris norvegicus* (left) can cause external epicarp lesion symptoms similar to those of larger bugs, but internal damage differs. Lygus feeding caused pitting (above).

In the initial phase of panicle and shoot blight disease, caused by *Botryosphaeria* fungus, small necrotic spots appear on the fruit surface (above). As the season progresses, the spots enlarge slightly, become darker, and coalesce (above right). Ultimately, blight of the rachis and shoot occurs (right).



Endocarp necrosis (above) and stylar-end lesion (below) both involve darkening of the blossom end of the pistachio fruit. Neither disorder has been associated with a living organism.

and distorted kernels. Black necrotic tissue often occurred extensively in these damaged areas as a result of feeding by leaffooted bugs; it is not clear at present whether the stinkbugs also cause this symptom. In one orchard with a leaffooted bug infestation, nuts affected by damage to the kernel were calculated at 16.5 percent.

Another diagnostic feature is the presence of protruding stylet sheath material produced by leaffooted bugs and stinkbugs during feeding. These microscopic sheaths, or "stylet spikes," were found on the outer hull surface, on the inner hull, on outer shell surfaces, inside the shell cavity, or sometimes on the nutmeat itself. Their presence indicated feeding activity by these bugs when there were no other hull or shell symptoms, particularly after the shells were completely hard and uncolored. However, stylet sheaths were not always found, even on nuts known to be damaged by caged bugs.

Both coreids and pentatomids are capable of penetrating mature shells and causing internal necrosis to nutmeats up to the time of harvest. After shells had completely hardened, however, these nuts did not show external lesion symptoms, nor did they drop as readily from the tree.

Plant bugs (Hemiptera: Miridae)

Our 1984 study showed that, in addition to leaffooted bugs and stinkbugs, several species of smaller plant bugs in the family Miridae caused epicarp lesion symptoms. These species included *Lygus hesperus* Knight and *Calocoris norvegicus* (Gmelin).

The initial external damage by these bugs to small fruits was similar to that caused by the larger bugs. Internal damage, however, was quite different and could be used to differentiate between, for example, *Lygus* and *Leptoglossus* attack on pistachios. Mirid feeding on immature fruits was characterized by complete destruction and liquefaction of shell tissue at the feeding sites within four hours after attack. The dissolved shell material was first green then light brown and was contained within the epidermal membranes, or cuticle, on either side of the shell. There was very little white netting in the shell tissue.

After ingestion of the partially digested shell by a feeding mirid, a distinct, clearly defined cavity or crater was left in the shell. At first, the cavity was covered by the interior cuticular membrane, but this membrane ruptured within 24 to 48 hours and exposed the entire cavity. Cavities were up to 2 to 2.5 mm in diameter and 1 mm deep with perpendicular walls. Only the shell tissue was affected; direct feeding in hull tissues by mirids was not observed, nor was hull tissue pitted. The hulls did develop typical lesion symptoms as caused by the other bugs, and damaged fruits eventually dropped from the nut clusters before shell maturity.

In contrast to mid- and late-season attack by the larger bugs, mirids are apparently unable to penetrate hardening pistachio shells. Consequently, kernel necrosis has not been observed with mirids, and potential crop loss from these bugs is usually over by late May or early June as shells mature. Exposed or prominent stylet sheaths were not seen in association with mirid feeding on pistachio fruits, but greater bleeding or resin exudate was present on hull surfaces. Most *Lygus hesperus* feeding sites (60 to 70 percent) tended to occur on the upper one-third, or shoulders, of a fruit, whereas feeding by leaffooted bugs and stinkbugs appeared to be more random.

Fungus-associated disorder

Symptoms of panicle and shoot blight consisted initially of small dark spots on the fruit surface, which appeared in June. Such spots were closely associated with pores (stomates) present in the epicarp. Affected fruits occurred predominately on the lower branches of trees in orchards with a closed or nearly closed canopy where high-angled sprinkler irrigation equipment was used. As the season progressed, the spots enlarged slightly, became darker, and coalesced. By mid-July, a blight of the rachis (the branched structure supporting pistachio fruits) and shoot occurred.

Isolations from the necrotic spots on the fruit yielded the fungus *Botryosphaeria*. This organism was also recovered from the blighted rachis and shoot, and preliminary results of pathogenicity tests verified it as a pathogen of pistachio (Pemberton, unpublished data). At present, this disease appears to be limited to only a few orchards in the northern Sacramento Valley, where crop losses were estimated at 10 to 40 percent.

Physiological disorders

Because the remaining fruit disorders have not been associated with living organisms, they have been classified as abiotic in nature.

In endocarp necrosis, the blossom end of the shell darkens while the skin and flesh layers remain unaffected. During 1984, fruit with these symptoms first appeared in early June on a Butte County farm. On that farm, we sampled four orchard blocks by collecting fruit clusters at random from a total of three trees per block and removed the hull tissues of 100 fruits per tree to expose the shell. Incidence of endocarp necrosis averaged 1, 4, 10, and 15 percent in these blocks of trees. In five other pistachio orchards in the San Joaquin Valley, the estimated incidence of endocarp necrosis ranged from 0 to as high as 10 percent. Attempts to associate microbes with this disorder failed when tissue pieces of the necrotic margins of the shells were placed on culture medium.

Stylar-end lesion is the necrosis of the skin, flesh, and shell layers on the blossom end of the fruit. This condition appeared in mid- to late May in Kern and Kings counties. In these orchards, symptomatic fruits were in tight clusters, that is, where normal elongation of the rachis did not occur. Incidence of stylar-end lesion among individual trees in a Kern County orchard ranged from 8 to 61 percent. Elsewhere, incidence of this disorder was very low or nonexistent.

Conclusions

All of these fruit disorders either reduce yields or affect nut quality. Therefore, crop management programs are necessary for their control.

A large complex of true bugs (Hemiptera) causes external epicarp lesion and internal shell and kernel necrosis on pistachio nuts. Damage symptoms, which vary with the insect family and maturity stage of the nuts, are diagnostic and can be used to determine which type of insect was feeding on a particular nut. These findings should be helpful in developing management strategies for insect control in pistachio orchards.

Further studies of panicle and shoot blight disease are needed to verify its relation to *Botryosphaeria* and to determine the environmental conditions required for infection. One approach in controlling the disease is to avoid irrigation equipment that discharges water into the tree's canopy. We did not observe this disease in orchards with low-angled sprinkler, flood, or drip irrigation systems.

Because the causes of endocarp necrosis and stylar-end lesion are not known, control measures for these disorders are not currently available. Finding the causes could prove difficult, because we have no clues at this time. It is worth mentioning that fruit with endocarp necrosis did not develop stylar-end lesion.

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