

## ABOVE GROUND FUNGAL DISEASES

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For many years, Verticillium wilt, caused by *Verticillium dahliae* Kleb, was the only fungal disease reported on pistachio trees in California and was the major threat to pistachio production. This disease continues to be a problem in older pistachio plantings where *Pistacia atlantica* was used as the rootstock. However, the development of resistant rootstocks (such as interspecific hybrids PGI, PGII, and UCB1) has solved the Verticillium wilt problem of the pistachios grown in California. In the spring of 1983, however, after a period of heavy and prolonged rains and cool weather, a disease characterized by blighted blossoms and shoots was observed and was described in 1984 as Botrytis blossom and shoot blight, caused by *Botrytis cinerea*. In the summer of 1984, another disease, characterized by blighted panicles and shoots, was first observed in an orchard near the town of Durham in Butte County and later in several orchards in northern California as well in the Chico Tree Improvement Center (now US Forest Service). In subsequent years, the same disease was observed sporadically in orchards in the southern part of the Sacramento Valley and later in central California. Repeated isolations from blighted panicles and shoots revealed the pycnidial stage (a *Fusicoccum* sp.) of the ascomycete *Botryosphaeria dothidea*. In 1985 a fourth fungal disease, Alternaria late blight, caused by *Alternaria alternata*, was first reported. By the late 1990s, Alternaria late blight was considered third in importance after Botryosphaeria panicle and shoot blight and Verticillium wilt, in causing significant losses. Some of the above mentioned diseases have also been reported in other countries besides the United States of America. Summaries of the most important diseases with diagnostic symptoms and signs of each disease and of

sporadic diseases recorded on pistachios in California are given in Tables 1 and 2. An index of pistachio diseases and fungi reported on pistachio in the United States is described in Table 3. Verticillium wilt and root diseases of pistachio are discussed in a separate chapter of this publication, which covers soil-borne diseases.

### ALTERNARIA LATE BLIGHT

The first record of Alternaria blight of pistachio is from Egypt in 1974. The disease has since been reported in California, Italy, and Australia. When serious, it causes shell staining and severe early defoliation, which reduces yield and weakens the tree.

#### Symptoms

Symptoms on leaves appear in midsummer and begin as dark brown to black, angular or circular lesions, 3-7 mm across that develop anywhere on the leaf blade. Lesions enlarge or coalesce to form tan blotches 2.5 - 3 cm across. Later in the season, dense sporulation, especially in the lesion center, can turn the lesion black. Multiple infections on leaf blades cause leaf blight and severe early defoliation. Infections on immature fruit are small black spots, about 1 mm in diameter, and are associated with lenticels. On mature fruit, hull lesions are black, 1-5 mm in diameter, and often surrounded by a reddish purple margin. Lesions along the broken edges of the ruptured hulls of early split fruit or those with hull cracking intensify shell staining. Occasionally, the fungus produces spores on the inner and outer shell surfaces and the inner surface of the hull (Plates 27A,B,C,D,E,F).

#### Causal Organism

Alternaria late blight is caused by *Alternaria alternata* (Fr.:Fr.) Keissl. Several strains have been isolated from pistachio, some of which

**Table 1.** The most important diseases of pistachio (*Pistacia vera*) in California

Disease common name	Fungal pathogen	Diagnosis: Main symptoms and signs
Panicle and shoot blight	<i>Botryosphaeria dothidea</i> ( <i>Fusicoccum</i> sp.)	Panicle, leaf, shoot, and bud blight; black angular or circular lesions on green fruit and immature leaves; large black lesions on fruit, large brown lesions on mature leaves with light beige margins.
Verticillium wilt	<i>Verticillium dahliae</i>	After sectioning, shoots show discolored xylem tissues very characteristic for trees infected by <i>V. dahliae</i> ; shoot wilting of certain portions of trees; and defoliation and sudden death of trees.
Alternaria late blight	<i>Alternaria alternata</i> , <i>A. tenuissima</i> , and <i>A. arborescens</i>	Small black spots on leaves or necrotic large areas with sporulation in the center; leaf blight; black lesions surrounded by red-colored halo on epicarp of fruit; fruit blight.
Blossom and shoot blight	<i>Botryotinia fuckeliana</i> ( <i>Botrytis cinerea</i> )	Blossom and shoot blight; cankers initiated from male inflorescences common on '02-16' and '02-18' cultivars, blight and flagging of tender shoots of both male and female trees.

**Table 2.** Sporadic diseases recorded on pistachios in California<sup>1</sup>

Disease common name	Fungal pathogen	Main symptoms and signs	Year recorded
Phomopsis blight	<i>Phomopsis</i> sp.	Shoot & leaf wilting (flagging)	1985
Sclerotinia blight	<i>Sclerotinia sclerotiorum</i>	Shoot and leaf wilting and blight (flagging)	1985
Root and crown rots	<i>Phytophthora</i> spp.	Black lesions on the crown of scion but not extending into the rootstocks.	1989
	<i>Armillaria mellea</i>	Decay of roots; white mycelial plaques and distinct black rhizomorphs.	1990
Hull and kernel molds	<i>Aspergillus niger</i>	Soft water-soaked hull covered with black sporulation on the surface touching the shell.	1995
	<i>Penicillium expansum</i> and other <i>Penicillium</i> spp.	Disintegration of hull covered with bluish, greenish powdery sporulation. Greenish, bluish sporulation leading to soft disintegration of the hulls.	1995
Anthrachnose hull rot	<i>Colletotrichum gloeosporioides</i>	Black and pinkish well-developed lesions on the hull. Entire surface of hull covered with bright pink and powdery sporulation.	1997 & 1998
Shoot blight	<i>Botryosphaeria rhodina</i>	Typical <i>Botryosphaeria</i> shoot blight symptoms. Affected shoots become dark brown to almost black	2000 & 2001

<sup>1</sup> These diseases were recorded throughout the State of California during 1985-2005.

**Table 3.** Index of fungi recorded on pistachio (*Pistacia vera* L.) in the United States<sup>1</sup>

Common name	Causal agent	State reported
Leaf necrosis; root rot	<i>Phytophthora nicotianae</i> var. <i>parasitica</i>	California
Phytophthora root rot	<i>Phytophthora</i> spp. <i>Pythium</i> sp.	California
Panicle and shoot blight	<i>Botryosphaeria dothidea</i>	California, Arizona
Armillaria root rot	<i>Armillaria mellea</i>	California
Thread blight	<i>Pellicularia koleroga</i>	Texas
Wood rots	<i>Pleurotus ostreatus</i>	California
Sapwood rot	<i>Schizophyllum commune</i>	California
Leaf and fruit spot	<i>Alternaria alternata</i> <i>A. tenuissima</i> , <i>A. arborescens</i> .	California
Blossom and shoot blight	<i>Botrytis cinerea</i>	California
Various hull and kernel decays	<i>Aspergillus</i> spp., <i>Aspergillus flavus</i> , <i>A. parasiticus</i> , <i>A. niger</i> , <i>Cladosporium</i> spp. <i>Fusarium oxysporum</i> , <i>Fusarium</i> spp., <i>Penicillium</i> sp., <i>Eurotium</i> spp., <i>Phomopsis</i> , <i>Pestalotiopsis</i> sp.	California
Root rot	<i>Phymatotrichopsis omnivorous</i>	California, Texas
Verticillium wilt	<i>Verticillium dahliae</i>	California
Leaf spots	<i>Hyalotia pistacina</i>	Maryland
Phomopsis blight	<i>Phomopsis</i> sp.	California
Leaf spot	<i>Phyllosticta lentisci</i>	Texas
Septoria leaf blight	<i>Septoria pistaciarum</i> <i>Septoria pistaciae</i>	Arizona California
Sclerotinia shoot blight	<i>Sclerotinia sclerotiorum</i>	California

<sup>1</sup> Source: Farr, D.F., Bills, G.F., Chamuris, G.P., and Rossman, A.Y. 1989. Fungi on Plants and Plant Products in the United States. APS Press. St. Paul, Minnesota.

sporulate better than others. Sporulating cultures are greenish black or rusty brown. The optimum temperature for growth and sporulation is 27-30°C (81-86°F). *A. arborescens* E. Simmons and *A. tenuissima* (Kunze:Fr.) have also been isolated from pistachio and proved to be pathogenic.

### Disease Cycle and Epidemiology

High relative humidity and dew favor the disease. The disease starts on leaves of the exposed shoots on the top of trees and eventually moves in the lower parts of the tree. Blight is worse in late summer because longer periods of dew and higher relative humidity above 95% are recorded in August and September than in June and July. At the same time, aging leaves and fruit become more susceptible. Leaves stressed by crop load appear susceptible to *Alternaria*, for infections

occur sooner and are more severe on leaves of fruit-bearing shoots than non-bearing shoots. Thus, *Alternaria* leaf blight is generally more severe in on crop years than in off crop years. Severe *Alternaria* leaf blight is most likely in orchards with higher humidity, such as ones located close to rivers, those with cover crops, or those irrigated by sprinklers or flooding when compared to drip irrigation systems.

The fungus is common in nature and survives and develops on crop debris and senescing leaves and weeds. The necrotic areas of leaves with Peter's scorch (Plate 27G), fruit damaged by sunburn or birds, and early split fruit are often colonized by *Alternaria* spp., probably including the pathogen causing *Alternaria* late blight. Spores are present on developing fruit and leaves from early April until harvest. In early August, there is a rapid three-fold increase in propagule number, and populations on leaves and fruit

remain high through September and October. The period of sudden rise in spore population is the critical period for disease development and management.

### Control

*Alternaria* late blight of pistachio, like other *Alternaria* diseases, is difficult to control, and requires a combination of fungicide treatment and good irrigation practices. Strobilurin fungicides (azoxystrobin, pyraclostrobin, and trifloxystrobin, which are registered in California pistachios) applied two or three times from June to early August can reduce disease if resistant *Alternaria* strains to strobilurin fungicides have not developed. Resistance to azoxystrobin has been reported in several orchards with *Alternaria* late blight in 2004. In addition, chlorothalonil, iprodione, and tebuconazole can control the disease if applied several times, but these fungicides are not registered. Curtailing irrigation during the critical period in early August lessens disease, and irrigation systems that allow the ground surface to dry quickly are recommended. Orchards with no cover crop or those irrigated by drip, micro-sprinkler, or sub-surface systems have less severe *Alternaria* late blight than flood- or sprinkler-irrigated orchards. Delayed harvest should be avoided to minimize fruit infection and the consequent shell staining. All commercial cultivars grown in California are susceptible.

### ANTHRACNOSE HULL ROT

Although species of *Colletotrichum* have frequently been isolated from washings of pistachio fruit and leaves in California, fruit with the characteristic pink sporulation (Plate 27H) of species of *Colletotrichum* have only been found once in a commercial pistachio orchard in Tulare County in 1998. The fungus was identified as *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz. (sexual stage *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk. *C. acutatum* J. H. Simmonds has been found causing a leaf spot and hull rot in pistachio in Australia. This species was shown to cause anthracnose of almonds in California. Although Koch's postulates have not yet been completed

on pistachios in California, this is the first report of *C. gloeosporioides* causing hull rot in California pistachio.

### Symptoms

Black/pinkish well-developed lesions were observed in the hulls. In some fruit the entire surface of the hull was covered with pink and powdery sporulation (Plate 27I). Lesions on leaves were not observed. Not much is known about the life cycle of this disease on pistachio and its sporadic occurrence does not necessitate any specific treatment.

### Control

No specific control measures are recommended. However, fungicide control of *Alternaria* late blight and/or *Botryosphaeria* blight may reduce Anthracnose hull rot.

### ASPERGILLUS FRUIT ROT

This disease is sporadic, but in very hot, dry years can cause some damage, occasionally as much as 2% loss.

### Symptoms

The early symptom of *Aspergillus* blight is a water-soaked discoloration of the hull which becomes soft and light beige. At harvest, infected fruit dry and their hulls become papery, densely wrinkled, and bright yellow, which also stains the shell bright yellow. All tissue under the skin of the epicarp is converted to black powdery spore masses of the pathogen. After washing off the spores, bright brown or yellow stains are seen on the shell surface. This staining differs from the lighter yellow staining caused by delayed harvest and from the uniform brown staining of hull infections caused by *A. alternata*. (Fr.:Fr.) Keissl (Plate 27J).

### Causal Organism

The pathogen is *Aspergillus niger* van Tiegh or other *Aspergillus* spp. in Section *Nigri*. *A. niger* grows quickly on all substrates. On acidified potato dextrose agar, colonies are white initially or sometimes with a yellow felt, then develop a compact layer of brown or black conidiophores are cream to yellow on the reverse side. Large cream to buff, globose or oval sclerotia are

occasionally produced. Optimal temperature for growth is 25-35°C.

### Disease Cycle and Epidemiology

The fungus attacks early split fruit through the ruptured hull or normal fruit with insect or mechanical damage. The incidence of *A. niger* in early split fruit is higher (up to 30%) than that in injured normal fruit, and about three times greater in older early split fruit than in those formed during the two weeks preceding harvest. Sources of inoculum include sporulation of *A. niger* inside early split fruit, on male inflorescences wetted by rains or irrigation water, or pistachio residues or seeds of weeds that have dropped to the ground.

### Control

No specific control measures are recommended. However, fungicide control of Alternaria late blight and/or Botryosphaeria blight may reduce Aspergillus blight.

### AFLATOXIN CONTAMINATION

Aflatoxins are naturally occurring toxins produced by specific molds while growing on certain commodities. Although the highest risk for aflatoxin contamination is in corn, peanuts, and cottonseed, other crops including tree nuts are sometimes also contaminated. Aflatoxin contamination of pistachio nuts grown in California is typically low with incidences of contamination about 1 in 10,000 nuts. Nevertheless, the tolerances for aflatoxin contamination established by governments are usually very low, typically 20 parts per billion (ppb) or lower.

### Symptoms and Associated Fungi

Aflatoxins are odorless and colorless chemicals, so they cannot be observed directly in pistachio nuts. Nonetheless, aflatoxin contamination develops during the growth of aflatoxin-producing fungi, and signs of these fungi can be observed. In California, aflatoxins can be produced by three closely related species of fungi, *Aspergillus flavus* Link ex Fries., *A. parasiticus* Spear, and *A. nomius* Kurtzman & Hesseltine. Typically, both *A. flavus* and *A. parasiticus* are present in commercial pistachio orchards with *A. flavus*

being more common than *A. parasiticus*, while *A. nomius* is only very rarely observed. All three species produce abundant yellowish-green spores, resulting in greenish-appearing colonies on the surface of kernels and on other substrates (Plate 27K). In addition to using the presence of these fungi to identify aflatoxin-contaminated nuts, the appearance of a nut can be used to indicate the likelihood of aflatoxin contamination. For example, nuts with dark brown discoloration on the shell exterior are more likely to be contaminated with aflatoxin, even though the aflatoxin-producing fungi do not cause shell discoloration. Although many aflatoxin-contaminated nuts have shells with extensive dark discoloration, some contaminated nuts will have little discoloration, including nuts that have a characteristic discoloration restricted to along the shell suture. Also, small nuts are more likely to be contaminated than large nuts.

### Disease Cycle and Epidemiology

The aflatoxin-producing fungi can grow on a wide variety of substrates but cannot grow well on living plant tissue. Therefore, besides decaying pistachio kernels, these fungi are usually found in pistachio orchards growing on plant debris on the orchard floor. The debris can be from weeds, cover crops, or the pistachio trees themselves. Examples of debris colonized by aflatoxin-producing fungi are dead leaves, male inflorescences, and fallen immature nuts. The aflatoxin-producing fungi grow very well at high temperatures (75°-105°F) but not at low temperatures (less than 55°F). Therefore, they are most active during the summer and have little or no growth during the winter. These fungi typically produce abundant spores (conidia), which are spread by wind and probably by certain cultural practices such as disking or mowing. At this time, it is not clear to what extent, if any, the spores are spread by insects and orchard birds.

As the pistachio nuts mature in the orchard, the kernels become susceptible to decay by aflatoxin-producing fungi. However, the intact hull serves as an effective barrier limiting the decay of the kernel. Nonetheless, the hull of the pistachio nut can become ruptured, which exposes the kernel to invasion by fungi and



insects. An important example of this is the early split nut, which is a nut that has the hull split along the shell suture where the shell has split (Plate 27L). Early split nuts are the main source of aflatoxin contamination in pistachio nuts. Early split nuts are typically about 2–3% of the nuts on the tree, although some orchards have over 10%. Early split nuts form throughout summer beginning in mid-July and continuing to harvest.

Navel orangeworm (*Amyelois transitella*) seems to play an important role in the aflatoxin contamination of pistachio nuts. Most of the aflatoxin found in pistachio nuts in California is in nuts damaged by navel orangeworm larvae. The reason for the association of navel orangeworm with aflatoxin contamination is not understood at this time but it seems likely that the navel orangeworm larva removes parts of the testa (seed coat) thereby exposing the kernel and enabling the aflatoxin-producing fungi to grow better.

Aflatoxin contamination is considered to occur preharvest and not postharvest. However, aflatoxin will continue to be produced during transport to the processor. The heat of the drying process probably kills the aflatoxin-producing fungi in the nuts but has little effect on the aflatoxin already present. Even if the heat does not kill these fungi, they are not able to grow and produce aflatoxin at the low moisture levels of the dried nuts. And as long as the dried nuts are stored at low moisture, these fungi are not able to grow and produce more aflatoxin.

### Control

The three main ways to reduce aflatoxin contamination are to control navel orangeworm, minimize early split nut formation, and avoid harvesting late. Ways to control navel orangeworm are presented elsewhere in this manual and will not be covered here. The best way to minimize the number of early split nuts seems to be to ensure that the trees receive sufficient water in late spring. Studies have shown that drought stress in May substantially increases the numbers of early split nuts at harvest. Rootstock also is a factor in early split

formation. Trees on *P. atlantica* tend to have more early split nuts than trees on other rootstocks, and trees on UCB1 tend to have the fewest early split nuts. Delaying harvest allows more time for aflatoxin-producing fungi to produce more aflatoxin in already colonized nuts besides infecting additional nuts. Also, delaying harvest can result in substantial increases in navel orangeworm damage, an important factor in aflatoxin contamination. So, late harvests can result in relatively high levels of aflatoxin contamination. In the last several year, experiments are being done using atoxigenic strains of *A. flavus* to control aflatoxin contamination in pistachio.

### BOTRYOSPHAERIA PANICLE AND SHOOT BLIGHT

Botryosphaeria blights of woody plants have been known since the early 1900's. The pathogen attacks more than 50 plant species representing 34 genera and 20 families that include almond, avocado, walnut, giant sequoia, coastal redwood, incense cedar, and willow. Botryosphaeria panicle and shoot blight of pistachio was first described in California in 1984 and now threatens the industry. The disease also occurs in Greece (misidentified and reported there as Camarosporium blight), Italy, and South Africa (although in South Africa two species of *Botryosphaeria* have been reported in causing stem cankers in *Pistacia* spp.).

### Symptoms

Symptoms appear in mid to late spring as black, 1-2 mm circular spots on shoots, rachises, and leaves. Shoots originating from contaminated or partially infected buds develop black lesions at the base. Lenticel infections in 1-year-old shoots are circular, black lesions of 5 to 7 mm in diameter, and usually do not enlarge in size. In mid May, leaves on infected shoots wither in 3-5 days and brown blighted shoots and leaves soon are distinct among the healthy dark green foliage (Plate 27M). Lesions on petioles kill individual leaflets, and entire leaves die (Plate 27N) when the petiole is infected and drop beginning as early as July. Early leaflet infections are somewhat elongated spots that aggregate along the midrib. Blade infections appear next and are small round black spots. Blade and midrib lesions enlarge as the summer progresses and become irregular

brown spots, up to 15 mm in diameter, and surrounded by a diffuse slightly chlorotic halo (Plate 27O). The lesions and halos often coalesce creating blotches of various shapes that ultimately dry to tan. The leaves die and fall from the tree. Most defoliation happens in late summer and can be severe (Plate 27P).

Rachis infections occur at the base or at branching points, and part or the entire rachis collapses depending upon the location of the lesion (Plate 27Q). Initial fruit infections appear in midsummer and are tiny, pin-sized round black spots, usually associated with lenticels. There may be a hundred or more such lesions, most of which develop no further, on each fruit. On one to several fruit in a panicle, a lesion enlarges, the fruit turns black, the infection moves into the peduncle, rachis and eventually the shoot on which the fruit cluster is borne killing part or the entire cluster (Plates 27R,S,T). Most of the blighted panicle is light tan, the consequence of girdling; only infected fruit are black, and these turn a silver gray in fall when the fungus produces pycnidia (Plate 27U). The naked dead black rachises, which remain on the tree for 3 or 4 years, are visible all year long and are a good diagnostic symptom.

Infections on shoots, or those that move into shoots from rachises or leaves, create cankers (Plate 27V). Sunken cankers develop around infected leaf and bud scars, range from 1 - 10 cm in length, and do not enlarge in subsequent years. *B. dothidea* (Moug.:Fr.) Ces & DeNot. sporadically causes cankers that are up to 30 cm in length and covered with dark exudates on trunks or extending into the main scaffolds. The infection is limited to the bark and does not kill trees. Gumming may or may not accompany *B. dothidea* infections and is not a diagnostic characteristic. Some cankers are associated with pruning wounds.

### Causal Organism

*Botryosphaeria dothidea* (synonym *B. ribis*; pycnidial stage a *Fusicoccum* sp.) produces black, asymmetrical pycnidia that are solitary or arranged in groups of 5-8, each with an apical ostiole through which the conidia extrude in a gelatinous matrix. In cross section the contents are shiny-white. Conidia are hyaline, nonseptate, fusiform, and measure 15-29 × 5-8

µm (Plate 27W). Isolates of *B. dothidea* from pistachio grow well on regular and acidified potato-dextrose agar at 20-36° C (68-96°F) with optimum at 27-30° C (81 - 86°F). Initially colonies are white, later changing to light gray, then almost black (Plate 27X). Many isolates do not produce pycnidia in culture and some produce reddish pigments. Two morphologically distinct strains (a gray and a black) were recorded since 1994 from infected pistachio fruit, but the importance of these two different strains is not known at this point. Some of the strains also produce a diffusible brown to reddish pigment in the culture medium. Characterization of *B. dothidea* isolates by means of molecular analysis is under investigation. In recent studies, cluster analyses of RAPD markers comparing 15 pistachio isolates of *B. dothidea* from three locations in California (north, central, and south) and known *B. dothidea*, *B. ribis*, and *Fusicoccum luteum* isolates indicate that the pistachio isolates are very uniform but unique in their molecular structure, and were separated as a taxonomic clade between *B. dothidea* and *B. ribis*. Based on recent taxonomic studies of the *Botryosphaeria* spp., the *Fusicoccum* sp. that causes panicle and shoot blight may be a species of *Botryosphaeria* different from *B. dothidea* and *B. ribis*. Only the pycnidial stage has been found on pistachio; however, the perfect stage (ascospore stage) was found on other hosts, such as almond, avocado, olive, pyracantha, California blackberry (*Rubus ursinus*), and walnut. Many conidial isolates from various hosts and ascospore isolates from blackberry routinely infected pistachio when inoculated.

In 2000 and 2001, shoot blighted due to panicle and shoot blight disease were collected from several pistachio orchards in California. The fungus isolated from these pistachios was *Lasiodiplodia theobromae*, the pycnidial stage of *Botryosphaeria rhodina*. Koch's postulates using five distinct isolates of *B. rhodina* from pistachio showed that they are pathogenic to current growth shoots. The disease cycle and epidemiology of *B. rhodina* has not been studied in pistachio.

### Disease Cycle and Epidemiology

Conidia released from pycnidia present on last year's blighted buds, shoots, petioles, rachises, and fruit cause the primary infections in spring

and summer (Plates 27U, 27X,Y,Z). New pycnidia in current season infections develop in late summer and fall and contribute inoculum for late season infection. In addition to these, pycnidia in old *B. dothidea* cankers produce conidia for at least 6 years. The pathogen also as a saprophyte colonizes dead wood, including old Botrytis cankers, and pycnidia produced here are another source of inoculum. Buds are infected as soon as they develop (Plate 27AA).

Conidia are spread mainly by rain but also by insects, birds, and water from sprinkler irrigation (Plate 27BB). Fall and winter rains spread conidia to leaf and bud scars and buds. Some buds are killed, some partially damaged, while others remain healthy but contaminated. Rain anytime during the growing season moves inoculum to growing tissues. The pathogen grows best at relatively high temperatures, thus the disease becomes severe in late spring to summer when temperatures rise. Some spring infections apparently remain latent until summer. The factors that trigger their development are unknown, although nutritional and drought stresses have been reported to predispose trees to the infection by this pathogen.

Airborne ascospores have been trapped in low numbers in pistachio orchards even though pseudothecia (structures producing ascospore inoculum) have not been observed there. Ascocarps were found in almond, avocado leaves, dead olive shoots, cankers on pyracantha, blighted blackberry canes close to pistachio orchards, and walnuts. Pycnidial isolates from these and several other hosts as well as ascospore isolates from blackberry induced blight on pistachio after inoculation, suggesting that other hosts can also serve as sources of inoculum for Botryosphaeria blight of pistachio. Ascospores could function in long distance dissemination, but pycnidia are the major sources of inoculum for the destructive epidemics in orchards.

### Control

Botryosphaeria panicle and shoot blight is extremely difficult to control, especially if allowed to increase over several years. The best approach employs fungicides, pruning, and irrigation management. Two to three

applications of strobilurins (azoxystrobin, pyraclostrobin, or trifloxystrobin) during summer will control the disease, although in orchards with high pressure, sprays can start at bloom and continue during summer. Pruning of infected parts 5 cm below the blighted margins in late summer and fall reduces disease the following year. In orchards irrigated by sprinklers, lowering the sprinkler angle so that water does not reach the tree canopy or shortening the duration of irrigation from 48 to 24 hours is recommended. Irrigating only during daytime for 12+12 hours on two consecutive days also is helpful. A monitoring system (BUDMON) based upon the percentage of buds harboring spores during the dormant season and another technique (Overnight Freezing Incubation Technique) that detects latent infections of the pathogen provide disease prognosis bases for making disease management decisions. Because the pathogen infests buds, for success in grafting, grafting material should be gathered only from trees in healthy orchards. Buds infested by *B. dothidea* when grafted on rootstocks usually do not develop.

### BOTRYTIS BLOSSOM AND SHOOT BLIGHT

Botrytis blossom and shoot blight of pistachio was first reported in California in 1984. The disease is sporadic but can cause significant damage in cool wet springs. Even though direct loss to Botrytis usually is minor, the colonization of old Botrytis cankers by *Botryosphaeria dothidea*, a more serious pathogen of pistachio, elevates the importance of Botrytis blossom and shoot blight.

### Symptoms

The disease occurs in spring and begins with the wilting and death of tender shoots on male and female trees (Plate 27CC). The dead shoots, with leaves attached, are bent in a characteristic shepherd's hook shape. The bases of blighted shoots become brown and swollen, and mycelium is sometimes present in the shoot pith. Infections of inflorescences on Peters trees cause blossom blight but do not extend into older wood. Expansion into older wood of 02-16 and 02-18 males often results in cankers up to 25 cm in length and death of 2-year-old wood (Plate 27DD). In very wet years, blossom infections on



female trees occasionally move into the previous-year's wood, girdling twigs thereby blighting young panicles. Diseased blossoms and basal portions of shoots are generally covered by the buff-colored sporulation of the pathogen (Plate 27EE). Infections on leaflets at the mid rib and upper part of the blade cause characteristic V-shaped lesions (Plate 27FF), whereas those on leaf blades away from the midrib are large distinct, circular lesions with wide chlorotic margins (Plate 27GG). Hail injury on leaves can be infected. The disease does not cause defoliation. Infections on the rachis can kill parts of developed fruit. Seedlings of rootstock nursery plants can also be infected, killing the tender tips (Plates 27HH, II,JJ,KK,LL).

### Causal Organism

*Botrytis cinerea* Pers.:Fr., the conidial stage of the ascomycete *Botryotinia fuckeliana* (deBary) Whetzel, produces smooth, lemon-shaped, light beige conidia that measure 11.7 x 9.3 µm. Two strains are commonly isolated from pistachio shoots. One produces abundant conidia and few large or no sclerotia, the other produces sparse conidiophores and conidia and abundant small black sclerotia on agar media. Sclerotia form on shoots infected the previous year then pruned and left on the orchard floor and, occasionally, on those remaining in the tree. Sclerotia incubated over wet sand at temperatures below 20°C (68°F) produce conidiophores and conidia, and presumably perform similarly in nature. Apothecia have not been observed on sclerotia in culture or in pistachio orchards.

### Disease Cycle and Epidemiology

Inoculum sources include 1-year-old infected shoots, dead bud scales, previous and current season infected female and male inflorescences, conidia produced on leaf lesions and blighted fruit clusters, sclerotia, and dead and senescing plant parts on the orchard floor. Conidia are airborne and also may be blown in from outside sources. Infections start in early spring when trees bloom and shoots emerge, and continue for about 3 - 4 weeks or until weather turns warm and dry. In California, the disease is prevalent during cool wet springs. Losses usually are insignificant, but in very wet years (such as

1998 and 2005) killing of fruit clusters may reduce yields (Plates 27HH,II,JJ). Frequently though, excessive killing of current-season shoots can reduce next season's fruiting wood, and blight of male flowers creates pollination problems.

### Control

One application with thiophanate-methyl during bloom lessens *Botrytis* blossom and shoot blight. New fungicides such fenhexamid also provide good control. Pruning and removing blighted shoots from the orchard reduces inoculum and perhaps also disease. Pruning blighted and cankered shoots to prevent colonization by *B. dothidea*, is important. Most cultivars are susceptible, and the male cultivars 02-16 and 02-18 are more susceptible than the commonly used Peters pollinator. Also the cultivar Kerman can suffer significant damage in excessively wet years.

### KERNEL DECAY

The severity of kernel decay varies from year to year and averages 1-2%. Increased concern for contamination by mycotoxins, especially aflatoxins, has raised the importance of kernel decay (for more details see aflatoxin contamination section). Decay by *Aspergillus flavus* and *A. parasiticus* (the fungi producing aflatoxins) is rare and occurs with decreasing frequency in pistachio, walnut, pecan, and almond.

### Symptoms and Associated Fungi

Common saprophytic fungi including species of *Alternaria*, *Aspergillus*, *Cladosporium*, *Eurotium*, *Fusarium*, *Penicillium*, *Trichothecium*, *Ulocladium*, *Epicoccum*, and *Rhizopus* decay kernels of pistachio. Kernel decay is associated with dark brown discoloration or "staining" of the shell exterior but most shell discoloration is probably caused by rupture of the hull and not by fungal decay. If decay is extensive, the kernel is discolored beneath the seed coat. The shells of fruit infected by *A. niger* are stained a characteristic bright yellow (Plate 27MM).

### Disease Cycle and Epidemiology

Little is known of the infection biology of fungi associated with kernel molds. Most are common saprophytes that live on dead organic matter in the orchard and some develop on senescing

leaves of weeds. Spores are probably spread by wind and blowing dust. Based on observations, kernel mold severity varies by orchard and season, and is often highest if significant rains occur in spring or just before and during harvest. Nearly all kernel decay and aflatoxin contamination occurs before harvest and is associated with early split fruit or those damaged by navel orangeworm (*Amyelois transitella*) larvae. Improper storage conditions that allow moisture to build up can result in significant contamination by several *Aspergillus* species, including *A. flavus*.

### Control

Control of navel orangeworm and irrigation to avoid drought stress in May can minimize early splitting. Also harvest as soon as the nuts are mature in order to avoid increased levels of kernel molds. Pioneer Gold I (PGI) and UCB1 rootstocks result in lower incidence of early splits than *P. atlantica* and PGII and thus can contribute in reducing kernel infections by *Aspergillus* fungi.

### PHOMOPSIS BLIGHT

Phomopsis blight occurs sporadically on pistachio trees in northern California and in Greece and causes little or no economic damage. Symptoms are similar to those caused by Botrytis blossom and shoot blight and Sclerotinia blight. Shoots turn brown in late spring and are distinctly visible among healthy dark green foliage (Plate 27NN). *Phomopsis* spp. were also isolated from diseased fruit and cankers caused by *Botrytis cinerea* or *Botryosphaeria dothidea*. Leaf and petiole wilting occurred within one week, and shoot wilting within 3 weeks after inoculation in April of current-season shoots. Current-season shoots inoculated with *Phomopsis* in late July remained symptomless but the pathogen was recovered the following March from the inoculation sites. White or light beige to brown isolates of a *Phomopsis* sp. were recovered from pistachio and native plants including wild blackberry (*Rubus ursinus*), elderberry (*Sambucus caerulea*), and willow (*Salix* sp.) grown close to

pistachio orchards plants. Pycnidia with  $\alpha$  (elliptical) and  $\beta$  (filiform) pycnidiospores can be found in blighted pistachio. Pycnidiospores are splashed-dispersed by rain or sprinkler water and contaminate buds. The fungus may also be spread by ascospores of the *Diaporthe* perfect stage, which has not been found on pistachio. Although mycelium of *Phomopsis* sp. may develop inside the shell cavity before shell splitting in insect-punctured fruit, only circumstantial evidence implicates hemipteran insects in the spread of this pathogen.

### POWDERY MILDEW

Powdery mildew is a minor disease and occurs wherever pistachio is grown.

### Symptoms

Powdery mildew attacks hulls, rachises, petioles, the undersides of leaves, and young stems (Plates 27OO,PP). In California pistachios, the fungus is active only in spring and produces colonies usually smaller than 2 cm in diameter. A brown scar or russeted look develops on infected tissues as the fungal mycelium dies (Plate 27QQ).

### Causal Organism

Powdery mildew fungi reported on pistachio include *Uncinula verniciferae* P. Henn. on *Pistacia chinensis* and an *Oidium* sp. on *P. vera*. Identification of the powdery mildew on pistachio in California has been difficult because cleistothecia have not been observed. *Phyllactinia guttata* (Wallr.:Fr.) has been reported on *Pistacia atlantica* in Greece and has erect conidiophores 50-350  $\mu$ m long which produce singular, clavate or somewhat rhomboid conidia. One or more germ tubes usually occur on the side of the conidium with an unlobed appressorium. The cleistothecia are scattered bearing 3-15 equatorial appendages. Appendages have a distinct bulbous swelling at the base and are 1-1.5 times as long as the cleistothecial diameter. They may contain 6 - 30, broadly clavate to slender asci with two elliptical ascospores. Few studies have investigated the host range or possible biological specialization of this fungus.

### Disease Cycle and Epidemiology

The epidemiology of the powdery mildew on pistachio has not been examined. As with other powdery mildews, the conidia are readily wind disseminated. Cleistothecia develop within the external hyphae and form appendages late in their development. For *P. guttata*, the uneven wall thickness and bulbous nature of the appendages allows for the entire cleistocarp to be dislodged from the host. When dry, the appendages flex like a spider's legs and allow the cleistocarp to be dislodged and carried by wind. These fungi may overwinter as cleistothecia or through buds infected the previous season.

### Control

The disease occurs sporadically on the pistachio cultivar Kerman but is more widespread on Trabonella and Red Aleppo cultivars (Plate 27DD). The disease is not serious enough to warrant control.

### SCLEROTINIA BLIGHT

Sclerotinia blight is a minor blight of pistachio and occurs primarily in early spring in pistachio orchards in northern California.

### Symptoms

The symptoms, similar to those caused by *Botrytis cinerea*, are wilting of tender shoots, and flagging, shriveling, and drying of the leaves (Plate 27RR). The base of the shoot turns a dark gray, and unless the sporulation of *B. cinerea* is present on infected tissue, correct diagnosis requires isolation.

### Causal Organism

*Sclerotinia sclerotiorum* (Lib.) de Bary, grown on potato-dextrose agar, has white mycelium with large, black sclerotia developing on the colony surface mainly around the edge of the Plate. Sclerotia are not found on infected pistachio tissues. Apothecia, which arise from the sclerotia, are brown, relatively flat, up to 10 mm in diameter, and produce ascospores. The fungus does not produce conidia.

### Disease Cycle, Epidemiology and Control

*S. sclerotiorum* survives as sclerotia in the soil and can be disseminated in contaminated soil, organic amendments, or water. The development of apothecia is favored by moderately cool (14°C) (57°F) temperatures and wet conditions. Once established, the fungus grows between 5 and 30°C (41 and 86°F) and best between 15 and 20°C (59 and 68°F). Ascospores, which are actively ejected into the air from apothecia at the soil surface in the orchard, are the only inoculum and can travel considerable distances before they land on a susceptible host. Inoculum probably originates in nearby vineyards and prune orchards where sclerotia and apothecia have been observed. Sclerotia have not been found in pistachio orchards. The infection court is not known but it is believed that ascospores infect buds as they emerge. Wilting is observed 2 days after infection while shoot death may take up to 7-10 days. The disease is infrequent and sporadic and control measures are not recommended.

### SEPTORIA LEAF SPOTS

Septoria leaf spots are caused by three species of *Septoria*. *Septoria pistacina* and *S. pistaciarum* are common pathogens of pistachio in Mediterranean countries and the Middle East. *S. pistaciarum* has been reported on *P. vera* but not *P. atlantica* and *P. terrebinthus* in Texas and Arizona in the United States. *S. pistaciae* is extremely rare and has been reported in France, Italy, and California. Severe epidemics of *Septoria* spp. can cause premature defoliation and reduce tree vigor.

### Symptoms

*S. pistaciarum*, which occurs in Arizona, first causes angular brown necrotic lesions, 1-2 mm in diameter and limited by the smaller veinlets of the leaf, on both leaf surfaces (Plate 27SS). Up to 20 pycnidia form in the center of the spots which often merge becoming large necrotic areas (Plate 27TT). Similar spots are produced on fruit.

*S. pistaciae*, recorded in one orchard in California, produces numerous, subcuticular

brown spots, 0.5 - 1.5 mm, on both sides of the leaf. Numerous small black pycnidia are aggregated and immersed in the spots. In California, *S. pistaciae* causes distinct grayish to light-brown fruit lesions, 1-4 mm in diameter, surrounded by a bright, distinctly reddish halo and mostly located near the peduncle (Plate 27UU). Leaf infections have not been observed.

### Causal Organisms

*Mycosphaerella pistaciarum* Chitzanidis (pycnidial stage *Septoria pistaciarum* Caracciolo) produces black ascocarps, with 50-60 cylindrical, clavate, and short stipitate asci in each ascocarp. The ascospores, eight in each ascus, are oblong and bi-cellular. Pycnidia have a typical ostiole and form on both sides of the leaf. Conidia are hyaline, filiform, and irregularly curved with 2-5 and occasionally 9 septa. Spermogonia produce hyaline, rod-shaped spermatia. Pycnidia of isolates of *S. pistaciarum* from Arizona pistachio trees bear curved to falcate conidia with 3-9 septa.

*S. pistaciae* Desmaz. pycnidia have a peridium complete to the top forming a typical ostiole. Pycnidiospores are linear, straight or curved, and most have one septum, though some have two or three. The teleomorph has not been identified.

*Septoria pistacina* Allescher (perfect stage *Mycosphaerella pistacina* Chitzanidis) produces pycnidia (160-200 x 96-160 µm) that develop on both the upper and lower surfaces of the leaf. Conidia are 31.8-47.0 x 3.6 -4.8 µm, hyaline, filiform, and curved, with one septum at the middle. Spermogonia measure 80-118 x 77-106 µm, and spermatia are hyaline, rod-shaped, and 3.9-5.6 x 1.0-1.4 µm. The *M. pistacina* produces black pseudothecia of 90-112 x 80-106 µm, each bearing about 20 clavate, short-stipitate asci. Each ascus has eight ascospores, measuring 26.2-40 x 3.2-4.8 µm; ascospores are hyaline, two-celled, and have a constriction at the septum. This species has not been encountered in USA. It is common in Turkey and Greece (Plate 27VV).

### Epidemiology and Disease Cycle

*S. pistaciarum* (and *S. pistacina*) overwinter in fallen leaves that were infected while on the tree in previous seasons. Pseudothecial primordia appear on fallen leaves early and young asci develop from then until February to early March. Most ascospores are mature and ready for discharge from late April through May, and are released during or after rain. The optimum temperature for the development of pseudothecia and asci of *S. pistaciarum* is 10°C (50°F). Infections due to ascospores of *S. pistaciarum* have an incubation period of about 10 days. Pycnidia with viable conidia of *S. pistaciarum* have been found on fallen leaves throughout winter and up to July, but their role in the disease is unknown. Secondary infections are caused by *S. pistacina* conidia, which spread by rain or sprinkler water, and may continue until late fall. Spermogonia production begins on leaves on the tree in mid-September and continues on fallen leaves until December. Their role is unknown. Nothing is known about the epidemiology of *S. pistaciae*.

### Control

Septoria leaf spots are controlled with preventive fungicide sprays. Dithiocarbamates (zineb, mancozeb) are recommended. Also chlorothalonil and copper fungicides are also effective, but must be applied after fruit have reached 1 cm size to avoid phytotoxic damage to very young fruit. Benzimidazole fungicides are also effective. Applications should begin when the first leaves unfold and repeated if necessary monthly until early June. None of these fungicides are registered for California pistachios.

### STIGMATOMYCOSIS

The term Stigmatomycosis is the general name for a disease that occurs in a number of crops, such as cotton, soybean, pecan, pomegranate, citrus, and pistachio in the United States. It has been reported on pistachio in Greece, Iran, Russia, and is frequently a problem in California pistachio orchards severely infested by hemipteran insects. In a 1989 survey in California, fruit with stigmatomycosis were



found in 90% of samples collected from late June to mid-September and from all growing areas.

### Symptoms

Stigmatomycosis is characterized by a wet, smelly, rancid, slimy kernel. Kernels with stigmatomycosis can be 1) small, dark green and partially developed with a brown funiculus, 2) well-developed, dark green and rancid (Plates 27WW,YY,XX), or 3) full-sized but abnormal, being white or light yellow and jelly-like, with a lobbed appearance. In contrast, symptoms of kernel necrosis (see epicarp lesion), which is caused by large hemipterans, are dry, punky, brown areas in the kernel. Sometimes, kernel necrosis and stigmatomycosis symptoms can be present in the same fruit.

### Causal Organisms

*Eremothecium coryli* (Peglion) Kurtzman (syn. *Nematospora coryli* Peglion), an ascomycete yeast, and *Aureobasidium pullulans* (de Bary) G. Arnaud cause stigmatomycosis. *E. coryli* grows on potato dextrose agar as yeast-like oval or spherical budding cells either isolated or in short chains and has few hyphae which are septate at maturity. In addition to buds, the yeast produces many asci (or sporiferous sacs or sporangia) that are cylindrical to naviculate, with two to eight needle-like ascospores arranged lengthwise. Ascospores are apiculate to fusiform, with a distinct septum at or near the center and the upper cell slightly broader at the septum, and after liberation are held together in a mass by long appendages. *E. coryli* colonies are creamy and perfectly round. The yeast grows at 10-37°C (50-99°F), with an optimum range of 30-35°C (85-95°F). More asci form at 15-20°C (59-68°F) than 25-35°C (77-95°F).

*A. pullulans* on potato dextrose agar produces smooth, faint pink yeast-like colonies that are covered with a slimy mass of spores. Older colonies change to black due to chlamydospore production. Primary conidia are hyaline, smooth, ellipsoidal, one-celled, and variable in shape and size; secondary conidia are smaller. Conidiophores are undifferentiated, intercalary or terminal, or arising as short lateral branches. Endoconidia are produced in an intercalary cell and released into a neighboring

empty cell. Hyphae are hyaline, smooth, thin-walled, with transverse septa. The fungus grows at 10-35°C (50-95°F) with optimum growth at 30°C (86°F).

### Disease Cycle and Epidemiology

The fungi causing stigmatomycosis are associated with hemipteran insects of the stinkbug families Pentatomidae and Coreidae. Hemipterans are common pests in pistachio orchards, and up to ten species have been found in California pistachio orchards. Three common stinkbug pests, *Thyanta pallidovirens*, *Chlorochroa uhleri*, and *C. ligata*, and a leaf-footed bug, *Leptoglossus clypealis* (Coreidae), experimentally transmitted *E. coryli*, which caused typical symptoms of stigmatomycosis in pistachio kernels. Symptoms first appear in late June after pistachio shells have hardened, but the disease becomes frequent in July through September, a period that coincides with kernel development. Smaller hemipterans, such as *Lygus* and *Calocoris* spp., may carry but not transmit the pathogens because they are unable to puncture the firm fruit pericarp after the second part of May. Stigmatomycosis and kernel necrosis are worse in orchards irrigated by sprinklers than in those irrigated by drip, microjets, or flood. This suggests either greater humidity requirements for infection, more activity of hemipterans in sprinkler-irrigated orchards, or more abundant pathogen propagules.

### Control

Fungicides do not control stigmatomycosis but insecticides reduce hemipteran vector populations and the incidence of stigmatomycosis.

### DISEASES REPORTED IN OTHER COUNTRIES

The following diseases have been reported in pistachios in countries other than USA.

- 1) Eutypa dieback (or gummosis), caused by *Eutypa lata* (Pers.:Fr.) Tul. & C. Tul., asexual stage: *Libertella blepharis* A. L. Smith, has been reported in Greece.
- 2) Gum canker, caused by *Cytospora terebinthi* Bres. has been reported in Italy and Iran.



- 3) Rust, which attacks *Pistacia vera*, *P. terebinthus*, *P. palaestina*, and *P. lentiscus* and caused by *Pileolaria terebinthi* Castagne (synonym *Uromyces terebinthi* (DC.) G. Winter), occurs commonly in the Mediterranean countries.
- 4) Stem canker caused by *Botryosphaeria obtusa* (Schwein.) Shoemaker (pycnidial stage *Sphaeropsis sapinea* (Fr.:Fr.) Dyko & Sutton) in South Africa pistachios.
- 5) Dieback and canker occurs in the Kerman province of Iran. The suspected pathogen is *Paecilomyces variotii* Bainier but pathogenicity tests have been inconsistent.

## NONINFECTIOUS DISORDERS

### EPICARP LESION AND KERNEL NECROSIS

Epicarp lesion puzzled pistachio growers for many years and was thought to be a genetic or nutritional disorder until it was associated with feeding by hemipteran insects. It occurs wherever pistachio trees are grown and hemipterans are present. Losses can be significant.

#### Symptoms

It is important to differentiate symptoms of epicarp lesion from those of *Botryosphaeria* or *Alternaria* fruit infections. In young fruit, the first symptoms of epicarp lesion appear in late April and during May as a light brown diffuse area with dark brown zones on the outer surface of the green fruit, with or without a black spot where the lesion originated. The affected area varies in size but may eventually cover the entire surface. Generally only a portion of the fruit surface is affected (Plate 27YY). In many cases, resin is found in the center of the lesion on the insect puncture. The lesions darken and dry as they age, remain soft in texture, and sink as the underlying tissues shrink. The discoloration generally extends into the shell, which is often thin and poorly developed in that area. Many affected fruit fall. On more mature fruit, epicarp lesion is an extended dark brown to black area on the hull but the fruit does not fall. Lesions are on the side of the fruit, occasionally just on the tip, and can extend to the base of the pedicel.

Although external symptoms are alike, internal symptoms vary with the species of hemipteran insect that caused them. In immature fruit, mirids cause internal pits 1-3 mm in diameter with blackened rims while larger hemipterans cause a white netting within 24-48 hours after insect feeding. As the fruit matures, and after late May, the smaller hemiptera can no longer puncture the lignified shell or cause epicarp lesion, but larger hemiptera can feed on maturing fruit through harvest. As the shell hardens, the white net symptom is sometimes replaced by a small black internal spot (Plate 27ZZ) where bugs feed with or without a protruding microscopic stylet sheath or "spike." As the shell hardens, damage from attacks by leaf-footed bugs and stinkbugs shifts to the kernel causing kernel necrosis. Such kernels are discolored, spongy, and punky. In commercial orchards where there are successions of various hemipteran insects of the families Miridae, Pentatomidae, and Coreidae, all symptoms are found.

#### Causal Agents

Among the insects known to cause epicarp lesion are *Leptoglossus occidentalis*, *Thyanta pallidovirens*, *Phytocoris relativus*, *P. conspurcatus*, *Calocoris norvegicus*, *Neurocolpus longirostris*, *Lygus hesperus*, *Psallus vaccinicola*, *Boisea rubrolineata*, *Liorhyssus hyalinus*, *Nezara viridula*, *Chlorochroa uhleri*, *C. ligata*, and *Acrosternum hilare*. The squash bug *Anasa tristis*, species of *Brochymena*, and harlequin *Murgantia histrionica* are found on pistachio but do not cause epicarp lesion or kernel necrosis.

#### Epidemiology and Control

Symptom type clearly corresponds to the predominant insect population present in the orchard at a specific period of time and both small and large hemiptera need to be controlled to reduce epicarp lesion and kernel necrosis. Control measures include insecticide application, biological control, and cultural practices. Cover crops planted along an orchard border can be sprayed to kill the insects that cause epicarp lesion. Insecticide sprays should be applied earlier than the time when epicarp lesions appear.

## HULL RUPTURE OF PISTACHIO

A normal pistachio fruit splits its shell while on the tree prior to harvest, but the hull remains intact and acts as an important physical barrier protecting the kernel from insect and fungal invasion. The hulls of a small percentage of fruit rupture before harvest. These are usually poor quality nuts because their kernels are frequently moldy and insect damaged and their shells discolored. Early splitting and hull cracking are distinct types of hull rupture.

### Early Splitting

The hull of an “early split” pistachio fruit splits along the shell suture (Plate 27L). Because both the hull and shell are split in the same place, the kernel is exposed and is frequently moldy and insect infested. In most orchards, approximately 2-3% of the fruit will be early splits, although the percentage can range from less than 1% to more than 10%. Early splitting begins in late July and continues into September. Moldy kernels are more likely in fruit whose hulls rupture in early summer. Early split fruit weigh less than normal fruit and have smaller shells that are stained yellow brown or dark brown, especially along the shell suture (Plate 27AAA). Early splits with rough, shriveled hulls have substantially more aflatoxin, moldy kernels, and insect infestation than those with smooth hulls, which split later. The cause of early splitting is unclear but irrigation is an important factor. Expansion or growth of the kernel in a shell cavity that is normally smaller than the size of the developing kernel is a possible cause of early splitting. Early splitting is increased substantially by insufficient irrigation during shell formation in late spring (May) and slightly by excessive irrigation in late August.

### Cracking of the Hull

Cracking occurs anywhere on the hull except along the shell split and exposes the shell. Typically fewer than 5% of fruit have cracked hulls at harvest. Because hull cracking begins later in the season than early splitting, fruit with cracked hulls are less likely to have moldy kernels and their shells are not as

severely discolored as are those of early split fruit. Expansion or growth of the hull while part is still attached to the shell is a possible cause of hull cracking. The relationship of irrigation to hull cracking is not known.

### Immature Nut Splitting

Immature nut splitting occurs sporadically in spring or early summer before the kernel has developed (Plate 27BBB). Although the cause is unknown, sudden changes in temperature and moisture may be involved.

## SHELL STAINING

Various degrees of shell stain result from decay or natural deterioration, injury, and oxidation of hulls and reduce the quality of pistachio fruit. Decaying hulls stain part or the entire shell surface light to dark brown, depending on the extent of hull decay. A severe brown shell stain usually develops in fruit infected by *Alternaria alternata*, particularly in orchards with sprinkler or flood irrigation. *Aspergillus niger* infections discolor shells a bright yellow. Natural deterioration of the hull produces a diffuse light yellow color and affects more fruit in late-harvested orchards than in those harvested early. Even in fruit harvested early, the initial signs of staining due to hull deterioration are present as 2-4 mm long blemishes near the stem side of the fruit. Failure to remove hulls from fruit within 24 hours of harvest leads to extensive and high incidence of oxidative yellow shell staining.

Harvesting as soon as fruit are mature and immediate hulling reduce shell staining. Converting irrigation systems to subsurface irrigation reduces relative humidity and shortens the period of dew formation on leaves, resulting in less *Alternaria* blight and thus less shell staining.

### PETER'S SCORCH

This disorder is very common in the male cultivar Peters, less so in other males, and only occasional in female trees. Because female trees are seldom affected, its potential for economic damage is undetermined. It has been reported from Arizona and California.

The first symptoms appear in late June and during July when air temperatures have increased. Initially, one to few small, black

water-soaked, angular lesions up to 5 mm in diameter appear in each leaflet blade, usually with exuded black gum in the center. These lesions become light green and diffuse then brown and necrotic (Plate 27CCC,DDD,EEE). if rain, dew, or high humidity prevails in summer, these lesions may be colonized by *Alternaria alternata* or *Cladosporium* spp. and their sporulation turns the lesions black or dark green (Plate 27G). 'Peters' trees showing scorch symptoms defoliate earlier than non-scorched trees. Colonization of scorch lesions by *A. alternata* can serve as an early source of *Alternaria* inoculum and also increases defoliation. Occasionally lesions caused by *B. dothidea* and Peter's scorch may occur on the same leaf.

The cause of Peter's scorch is unknown. The effects of climate, soil, and cultural practices on scorch are not known, and control measures are not available.

### PIN-SIZED LESION

Pin-sized lesion occurs infrequently on pistachio trees in California and causes no economic damage. In spring and summer, small chlorotic spots 2 - 3 mm in diameter and surrounded by a yellow halo appear on the foliage of both male and female trees (Plate 27FFF). At the center of these lesions is a pin-sized puncture, which usually penetrates the leaf (Plate 27GGG).

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### REFERENCES

- Ahimera, N., Driever, G. F., and Michailides, T. J. 2003. Relationships among propagules numbers of *Botryosphaeria dothidea*, latent infections, and severity of panicle and shoot blight in pistachio orchards. *Plant Disease* 87:846-853.
- Ahimera, N., Gisler, S., Morgan, D. P., and Michailides, T. J. 2004. Effects of single-drop impactions and natural and simulated rains on the dispersal of *Botryosphaeria dothidea* conidia. *Phytopathology* 94:1189-1197.
- Aminae, M. M., and Ershad, D. 1989. Die-back of young shoots of pistachios in Kerman. Page 79 in: *Proceedings of the 9th Plant Protection Congress of Iran*, Mashaad, Iran.
- Ash, G. J. and Lanoiselet, V. M. 2001. First report of *Colletotrichum acutatum* causing a leaf spot and hull rot of pistachio. *Australasian Plant Pathol.* 30:365-366.
- Ash, G. J. and Lanoiselet, V. M. 2001. First report of *Alternaria alternata* causing late blight of pistachio (*Pistacia vera*) in Australia. *Plant Pathol.* 50:803.
- Assawah, M. W. 1969. *Pileolaria terebinthi* Cast. on pistachio in U.A.R. (Egypt). *Phytopath. Medit.* 8:157-161.
- Bolkan, H. A., Ogawa, J. M., Rice, R. E., Bostock, R. M., and Crane, J. C. 1984. Leaf-footed bug implicated in pistachio epicarp lesion. *Calif. Agric.* 38(3 & 4):16-17.
- Bolkan, H. A., Ogawa, J. M., Rice, R. E., Bostock, R. M., and Crane, J. C. 1984. Leaf-footed bug (Hemiptera: Coreidae) and epicarp lesion of pistachio fruits. *J. Econ. Entomol.* 77:1163-1165.
- Bolkan, H. A., Ogawa, J. M., and Teranishi, H. R. 1984. Shoot blight of pistachio caused by *Botrytis cinerea*. *Plant Dis.* 68:163-165.
- Carter, M. V. 1991. The status of *Eutypa lata* as a pathogen. *Phytopathological Paper No. 32*. International Mycological Institute, London.
- Chen, W.-Q., Ntahimpera, N., Morgan, D. P., and Michailides, T. J. 2003. Mycoflora of *Pistacia vera* in the Central Valley, California. *Mycotaxon* 63:147-158.
- Chen, W.-Q., Morgan, D. P., Felts, D., and Michailides, T.J. 2003. Antagonism of *Paenibacillus lentimorbus* to *Botryosphaeria dothidea* and biological control of panicle and shoot blight of pistachio. *Plant Disease* 87:359-365.
- Chitzanidis, A. 1956. Species of *Septoria* on the leaves of *Pistacia vera* L. and their perfect states. *Annls Inst. Phytopathol. Benaki* 10:29-44.
- Corazza, L., Granata, G., Avanzato, D. and Chilosi, G. 1991. Main phytopathological aspects of pistachio nut in Italy. (In Italian; English summary). *Rivista di Frutticoltura* 4: 37-40.

15. De Sousa da Camara, E. 1948. *Mycetes aliquot lusitaniae*. Agronomia lusitana: 294.
16. Doster, M. A., and Michailides, T. J. 1994. *Aspergillus* molds and aflatoxins in pistachio nuts in California. *Phytopathology* 84:583-590.
17. Doster, M. A., Michailides, T. J., Goldhamer, D. A., and Morgan, D. P. 2001. Insufficient spring irrigation increases abnormal splitting of pistachio nuts. *Calif. Agriculture* 55(3):28-31.
18. Doster, M. A., and Michailides, T. J. 1994. Development of *Aspergillus* molds in litter from pistachio trees. *Plant Dis.* 78:393-397.
19. Doster, M. A., and Michailides, T. J. 1994. Relationship between shell discoloration of pistachio nuts and incidence of fungal decay and insect infestation. *Plant Dis.* 83:259-264.
20. Doster, M. A. and Michailides, T. J. 1995. The development of early split pistachio nuts and their contamination by molds, aflatoxins, and insects. *Acta Hort.* 415:359-364.
21. Doster, M. A. and Michailides, T. J. 1995. The relationship between date of hull splitting and decay of pistachio nuts by *Aspergillus* species. *Plant Dis.* 79:766-769.
22. Ershad, D., and Barkhordary, M. 1974. Host range and vectors of *Nematospora coryli* Peglion in Kerman of Iran. *Iran. J. Plant Pathol.* 10 (3,4):34-39.
23. Ershad, D., and Barkhordary, M. 1976. Investigations on stigmatomycosis (massu disease) of pistachio. *Iran. J. Plant Pathol.* 12 (1,2):17-23.
24. Goldhamer, D.A., Michailides, T. J. and Morgan, D. P. 2002. Buried drip irrigation reduces fungal disease in pistachio orchards. *California Agriculture* 55(4):133-138.
25. Granata, G. and Pennisi, A. M. 1984. "In vitro" activity of some fungicides against *Cytospora terebinthi*, agent of a pistachio decline. (In Italian; English summary). *Notiziario sulle malattie delle piante* 105 (32), 130-135.
26. Granata, G., Sidoti, A. and Corazza, L. 1993. New acquisitions on control of *Septoria pistaciae* Desm. and *Cytospora terebinthi* Bres. Pages 64-65 in: Proc. 9th G.R.E.M.P.A. Meeting Pistachio. Italy, May 20-21, 1993, 64-65.
27. Grigoriou, A. C. 1992. First detection of spermagonia, aecia and basidia of *Pileolaria terebinthi* (D. C.) Cast. [Syn. *Uromyces terebinthi* (D. C.) Winter] on pistachio trees. Page 72 in: Summaries of Invited Research Papers, 6<sup>th</sup> National Phytopathological Congress, Rio Patra, October 6-8, Hellenic Phytopath. Soc., Greece.
28. Holtz, B. A., Michailides, T. J., Ferguson, L., Hancock, J. G., and Weinhold, A.R. 1996. First Report of *Rhizoctonia solani* (AG-4) on Pistachio Rootstock Seedlings in California. *Plant Disease* 80:1303.
29. Korf, R. P., Dumont, K. L. 1972. *Whetzelinia*, a new generic name for *Sclerotinia sclerotiorum*. *Mycologia* 64:248-251.
30. Kouyeas, H. 1979. "Stigmatomycosis" of the nuts of the pistachio tree (*Pistacia vera* L.). *Annls. Inst. Phytopath. Benaki* 12:147-148.
31. Ma, Z., Boehm, E. W. A., Luo, Y., Michailides, T. J. 2001. Population structure of *Botryosphaeria dothidea* from pistachio and other hosts in California. *Phytopathology* 91:665-672.
32. Ma, Z., and Michailides, T. J. 2002. Characterization of *Botryosphaeria dothidea* isolates collected from pistachio and other hosts in California. *Phytopathology* 92:519-526.
33. Ma, Z., and Michailides, T. J. 2002. A PCR-based technique for identification of *Fusicoccum* sp. from pistachio and various other hosts in California. *Plant Dis.* 86:515-520.
34. Ma, Z., Morgan, D. P., Felts, D., and Michailides, T. J. 2002. Sensitivity of *Botryosphaeria dothidea* from California pistachio to tebuconazole. *Crop Prot.* 21:829-835.
35. Ma, Z., Luo, Y., and Michailides, T. J. 2003. Nested PCR assays for detection of *Monilinia fructicola* in stone fruit orchards and *Botryosphaeria dothidea* from pistachio in California. *J. of Phytopathology* 151:312-322.
36. Ma, Z., Felts, D., and Michailides, T. J. 2003. Resistance to azoxystrobin in *Alternaria* isolates from pistachio in California. *Pesticide Biochemistry and Physiology* 77:66-74.
37. Ma, Z., and Michailides, T. J. 2004. An allele-specific PCR assay for detecting azoxystrobin-

- resistant *Alternaria* isolates from pistachio in California. J. Phytopathology 152:118-121.
38. Ma, Z., Luo, Y., and Michailides, T. J. 2004. Spatiotemporal changes in the population structure of *Botryosphaeria dothidea* from California pistachio orchards. Phytopathology 94:326-332.
  39. Ma, Z., and Michailides, T. J. 2004. Characterization of iprodione-resistant *Alternaria* isolates from pistachio in California. Pesticide Biochemistry and Physiology 80:75-84.
  40. Ma, Z., and Michailides, T. J. 2004. A real-time PCR assay for the detection of azoxystrobin-resistant *Alternaria* populations from pistachio orchards in California. Crop protection 23:1259-1263.
  41. Ma, Z., and Michailides, T. J. 2005. Genetic structure of *Botrytis cinerea* from different host plants in California. Plant Disease 89:1083-1089.
  42. Ma, Z., and Michailides, T. J. 2005. Advances in understanding molecular mechanisms of fungicide resistance and molecular detection of resistant genotypes in phytopathogenic fungi. Crop Protection 24:853-863.
  43. Michailides, T. J., Morgan, D. P., and Felts, D. 2002. First report of *Botryosphaeria rhodina* causing shoot blight of pistachio in California. Plant Disease 86:1273.
  44. Michailides, T. J., Morgan, D. P., Ma, Z., Luo, Y., Felts, D., Doster, M. A., and Reyes, H. 2005. Conventional and molecular assays aid diagnosis of crop diseases and fungicide resistance. California Agriculture 59(2):115-123.
  45. Michailides, T. J. 1989. The 'Achilles hell' of pistachio fruit. Calif. Agric. 43(5):10-11.
  46. Michailides, T. J. 1991. First report of Septoria spot in California pistachios. Page 93 in: California Pistachio Industry Annu. Report Crop Year 1990-91. Fresno, CA.
  47. Michailides, T. J. 1991. Pathogenicity, distribution, sources of inoculum, and infection courts of *Botryosphaeria dothidea* on pistachio. Phytopathology 81:566-573.
  48. Michailides, T. J. 1991. Susceptibility of pistachio male cultivars to Botrytis blossom and shoot blight caused by *Botrytis cinerea*. Plant Dis. 75:410-415.
  49. Michailides, T. J., and Morgan, D. P. 1990. Etiology and transmission of stigmatomycosis disease of pistachio in California. Abstr. Phytopathology 80:973.
  50. Michailides, T. J., and Morgan, D. P. 1992. Effects of temperature and wetness duration on infection of pistachio by *Botryosphaeria dothidea* and management of disease by reducing duration of irrigation. Phytopathology 82:1399-1406.
  51. Michailides, T. J., and Morgan, D. P. 1993. Spore release by *Botryosphaeria dothidea* in pistachio orchards and disease control by altering the trajectory angle of sprinklers. Phytopathology 83:145-152.
  52. Michailides, T. J., and Morgan, D. P. 1994. Occurrence of Sclerotinia and Botrytis shoot blights on pistachio in California. Plant Dis. 68:163.
  53. Michailides, T. J., and Morgan, D. P. 1995. Spread of *Botryosphaeria dothidea* in pistachio orchards irrigated with drip or microjets. Calif. Pistachio Industry Annu. Rep. Crop year 1994-95, Fresno, p. 102-106.
  54. Michailides, T. J., and Morgan, D. P. 1996. Principles for the control of Alternaria late blight of pistachio caused by *Alternaria alternata* in California. Pages 56-63 in Proc. of the 9th G.R.E.M.P.A. Meeting – Pistachio, Caruso, T., E. Barone, and F. Sottile, eds. May 20-21, 1993, Bronte-Schiaccia, Italy.
  55. Michailides, T. J., Morgan, D. P., and Felts, D. 1997. Spread of *Botryosphaeria dothidea* in central California pistachio orchards. Acta Hortic. 470:582-591.
  56. Michailides, T. J., Morgan, D. P., and Goldhamer, D. A. 1995. Using subsurface drip irrigation to reduce Alternaria late blight of pistachio caused by *Alternaria alternata* (second year report). Calif. Pistachio Industry Annu. Rep. Crop year 1995-96, Fresno, p. 129-136.
  57. Michailides, T. J., Morgan, D. P., and Krueger, W. Chemical control of Botryosphaeria panicle and shoot blight of pistachio in California. Pages 73-76 in: California Pistachio Industry Annu. Rep. Crop Year 1994-95, Fresno, CA.
  58. Michailides, T. J., and Morgan, D. P. 2004. Panicle and Shoot blight of pistachio: A major threat to the California Pistachio Industry.



- January 2004, APSnet Feature:  
<http://www.apsnet.org/online/feature/pistachio/>
59. Michailides, T. J., Morgan, D.P., and Weinberger, G. 1995. Chemical control of Botrytis shoot blight with benomyl by ground or air application. Page 77 in: California Pistachio Industry Annual Report, Crop Year 1994-95. Calif. Pistachio Ind. Ed., Fresno.
  60. Michailides, T. J. and Ogawa, J. M. 1987. Botrytis blight of pistachio: New findings. California Pistachio Industry Annual Rep. Crop Year 1986-87. Pp. 108-112.
  61. Michailides, T. J., and Ogawa, J. M. 1987. Botrytis shoot blight of pistachio: New findings. Pages 108-112 in: California Pistachio Industry Annual Report, Crop Year 1986-87. Calif. Pistachio Ind. Ed., Fresno.
  62. Michailides, T.J., Rice, R. E., and Ogawa, J. M. 1987. Succession and significance of several hemipterans attacking a pistachio orchard. J. Econ. Entom. 80:398-406.
  63. Michailides, T. J., Teviotdale, B. L., and Weinberger, G. 1999. Botryosphaeria Blight of Pistachio: Identification and Control Manual. California Pistachio Commission, Fresno, 53 pp.
  64. Mila, A., Driever, G. F., Morgan, D. P., and Michailides, T. J. 2005. Effects of latent infection, temperature, precipitation, and irrigation on panicle and shoot blight of pistachio in California. Phytopathology 95:926-932.
  65. Munkvold, G. P. and Marois, J. J. 1994. Eutypa dieback of sweet cherry and occurrence of *Eutypa lata* perithecia in the central valley of California. Plant Dis. 78:200-207.
  66. Ntahimpera, N., Driever, G. F., Felts, D., Morgan, D. P., and Michailides, T. J. 2002. Dynamics and pattern of latent infection caused by *Botryosphaeria dothidea* on pistachio buds. Plant Dis. 86:282-287.
  67. Pantidou, M. L., 1973. Fungus-Host Index for Greece. Benaki Phytopathological Institute. Athens, Greece. 382 pp.
  68. Parmeter, J.R. Jr. 1970. Biology and Pathology of *Rhizoctonia solani*. University of California Press, Berkeley.
  69. Pearson, T. C., Doster, M. A., and Michailides, T. J. 2001. Automated detection of pistachio defects by machine vision. Applied Engineer. in Agric. 17:729-732.
  70. Pryor, B. M., and Michailides, T. J. 2002. Morphological, and molecular characterization of *Alternaria* isolates associated with Alternaria late blight of pistachio. Phytopathology 92:406-416.
  71. Rice, R. E., Uyemoto, J. K., Ogawa, J. M., and Pemberton, W. M. 1985. New findings on pistachio problems. California Agriculture 39:15-18.
  72. Rumbos, I. C., 1986. Isolation and identification of *Eutypa lata* from *Pistacia vera* in Greece. J. Phytopath. 116:352-357.
  73. Simmons, E. G. 1967. Typification of *Alternaria*, *Stemphylium*, and *Ulocladium*. Mycologia 59:67-92.
  74. Sneh, B., Burpee, L., and Ogoshi, A. 1991. Identification of *Rhizoctonia* Species. APS Press, 133 pp.
  75. Swart, W. J., and Bates, W. M. 1995. First report of stem canker caused by *Botryosphaeria obtusa* on pistachio. Plant Disease 79:1036-1038.
  76. Swart, W. J., and J. T. Blodgett. 1998. First report of *Botryosphaeria dothidea* basal canker of pistachio trees in South Africa. Plant Disease 82:960.
  77. Tzavella-Klonari, K. 1979 Studies on the biology, epidemiology and pathogenesis of the disease caused by the fungus *Septoria pistaciarum* Caracciolo on pistachio nut tree. (Ph. D. thesis) Sc. Ann. School Agric. and For. Arist. Univ. Thessaloniki, Vol. KA, 72 pp (in Greek).
  78. Wasfy, E. H., Ibrahim, I. A., and Elarosi, H. M. 1974. New *Alternaria* disease of pistachio in Egypt. Phytopath. Medit. 13:110-111.
  79. Young D. J. and Michailides, T. J. 1989. First report of Septoria leaf spot of pistachio in Arizona. Plant Dis. 73:9.