Relation of Postharvest Decay Fungi to the Slip-Skin Maceration Disorder of Dried French Prunes

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ABSTRACT

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A postharvest disorder ("box rot") of dried French prunes was studied. Symptoms of this condition are soft, sticky, macerated areas on the fruit and slippage of the skin under slight pressure. When fresh prune fruit were inoculated with *Rhizopus arrhizus* and *R. stolonifer*, more slip-skin maceration (SSM) disorder subsequently developed on dried prunes than when fruit were inoculated with other fungi such as *Aspergillus japonicus*, *Penicillium expansum*, *Mucor circinelloides*, *Cladosporium herbarum*, and *Monilinia* spp. Fresh fruit were commonly found to be naturally contaminated with *Rhizopus* spp. Fresh fruit held in bins at ambient field temperature for 24 hr or more after harvest subsequently developed SSM disorder after drying in proportion to the occurrence of prior *Rhizopus* infection. The following evidence, which implicates *Rhizopus* as the most common cause of SSM disorder, was provided. When fresh fruit were observed to be infected with *Rhizopus* spp. before drying, significant numbers of dried prunes exhibited SSM disorder; a high percentage of fresh prunes inoculated with *Rhizopus* spp. and incubated for 24 hr or more developed SSM disorder on drying; and tissue associated with SSM disorder on dried fruit obtained from commercial sources and from *Rhizopus*-inoculated fresh fruit that were dried contained a pectinolytic enzyme typical of that produced by *Rhizopus* spp. during decay of fresh fruit.

Additional key words: pectinase, prune deterioration, *Rhizopus* rot.

French prunes (*Prunus domestica* L. 'French') are at optimum maturity for harvest when the soluble solids content is at a minimum of 24–25% and the average fruit flesh firmness has decreased to 1.4–1.8 kg (9). Fresh harvested fruit are placed into bins of approximately 454-kg capacity, and usually are held for less than 24 hr before being dried. To prevent confusion, "fruit" always refers to prunes before they are dried, and "prunes" indicates the dried fruit.

The dehydration process (6,12,17) involves moving single layers of fruit in trays (0.9 × 1.8 m) through a tunnel with a parallel flow of hot air moving the same direction as the fruit movement for 12–20 hr. Maximum air temperature in the front end of the tunnel is about 90 C; however, the fruit itself is well below 90 C, which prevents carmelization of sugars. Drying reduces moisture to approximately 20%, and the dried prunes range from 38.4 to 51% reducing sugars (glucose and fructose) and 0.6 to 5.5% sucrose, which inhibits mold growth (16).

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Poor-quality dried prunes result from a number of different causes. Decay of fresh fruit by *Monilinia fructicola* or *M. laxa* is one of the most important causes of poor-quality dried fruit (2). This decay usually starts while the fruit is still on the tree but is stopped by the drying process. El Behadli (5) seldom isolated *Monilinia* spp. from dried prunes and showed that their spores were killed in the dehydrator when exposed to hot air at 72–84 C for 12–19 hr. Storage molds growing on dried fruit of high moisture content also cause poor quality. Miller and Tanaka (8) showed that the *Aspergillus glaucus* group of fungi and *Saccharomyces rouxii* yeast could grow at 20 C on dried prunes in equilibrium with air at a relative humidity (RH) of 76%. *A. niger* and *Penicillium* sp. grew well on prunes equilibrated at 93% RH but failed to grow on those equilibrated at 87%. These fungi are considered less important than *S. rouxii* or the *A. glaucus* group because they did not cause spoilage at RHs below 87%. El Behadli (4) showed that the *A. glaucus* group of fungi was the most prevalent because the dehydration process did not kill their spores, and they were able to grow at low RHs. Finally, an important postharvest disorder on dried prunes referred to by the prune industry as "box rot" (Fig. 1) was due to an unknown cause. The main symptoms of this condition are macerated areas on the dried fruit, which are soft and sticky, and a slipping of the fruit skin under slight pressure. The purpose of this study was to determine which fungi could cause this slip-skin maceration (SSM) disorder of dried prunes. Furthermore, because high levels of pectinase activity were found in some commercially dried prune samples with SSM disorder, attempts were made to determine its significance. A preliminary report of this study was published (14).

MATERIALS AND METHODS

Inoculation study. Ripe fresh French prune fruit from the experimental farm of the University of California, Davis, were placed in sterilized net bags (capacity 50 per bag, six bags per sample), surface-sterilized for 3 min with 0.5% sodium hypochlorite, dipped in a fungal-spore suspension consisting of at least 5,000 spores per milliliter, placed in large plastic bags (six net bags per plastic bag), and incubated at 28 C. The fungi used for individual inoculations were *Alternaria alternata* (Fr.) Keissler, *Aspergillus chevalieri* (Mangin) Thom and Church, *A. japonicus* Saito, *Botrytis cinerea* Pers. ex Fr., *Cladosporium herbarum* (Pers.) Link ex S. F. Gray, *Monilinia fructicola* (Wint.) Honey, *M. laxa* (Aderh. & Ruhl.) Honey, *Mucor circinelloides* V. Tiegh, *Penicillium expansum* Link ex Thom, *Rhizopus arrhizus* Fischer ATCC #11145, *R. arrhizus* isolate #2, *R. arrhizus* isolate #3, and *R. stolonifer* (Ehr. ex Fr.) Lind. Each inoculation treatment consisted of 48 net bags; fruit in 24 of the bags were injured by stabbing approximately 0.5 cm deep with a dissecting needle, and the rest were left uninjured. Six bags of uninjured and injured fruit were immediately placed in cold storage (0 C), and another six bags incubated for 24, 48, and 68 hr at 28 C before cold storage. Injured fruit were kept separated from the uninjured fruit in plastic bags within the incubation chamber. The lots of fruit incubated for 24, 48, and 68 hr were examined daily for decay. They were considered decayed if the skin of the fresh fruit appeared macerated. Samples were left at 0 C for 1–3 days, and they were all commercially dried at the same time. After drying, the prunes from each group of six net bags comprising one sample were bulked into a single plastic bag (300 dried prunes). Subsamples were removed to test for SSM disorder (50 prunes) and pectinase (50 prunes).

SSM disorder was evaluated by prune-industry inspectors who scored any damage to the prune skin, with the exception of cracks, sunburn, scab, mold, or brown rot, as SSM disorder.

Field study. To assess decay development, one standard bin of fresh fruit was harvested from each of three different growing areas in California. Each bin was divided into four sections by wooden partitions, and each section was sampled at different times after harvest (Fig. 2). Fruit from the Tulare area (Southern San Joaquin Valley) was harvested at 1230 hours, 27 August 1980 by shaking three trees, catching the fruit on a catching frame, and conveying the fruit directly into the bin; fruit from the Yuba City area (Central Sacramento Valley) was obtained similarly at 0930 hours, 29 August 1980 by shaking two trees; and fruit from a coastal area near Healdsburg was harvested by knocking the fruit on bare ground first, picking it up 3 hr later, and placing it in the bin at 1100 hours, 30 August 1980.

Fruit maturity at harvest was measured by a flesh firmness tester (D. Ballauf Mfg. Co. Inc., Washington, DC) with a 0.8-cm diameter plunger. These bins were immediately brought to the experimental farm of the University of California, Davis, and held in the open air exposed to direct sun for 3 days.

Net bags containing 25 fresh prunes that had been punctured and inoculated with a mixture of *R. arrhizus* #2 and *R. stolonifer* spores were placed in each section as the bins were filled to determine whether *Rhizopus* spp. would cause decay of surrounding fruit. The bags were placed approximately 10 cm below the upper fruit surface in each bin section. Similarly, a net bag containing 25 punctured fresh fruit inoculated with spores of *M. laxa* and *M. fructicola* was placed close to the edge of each section. The middle

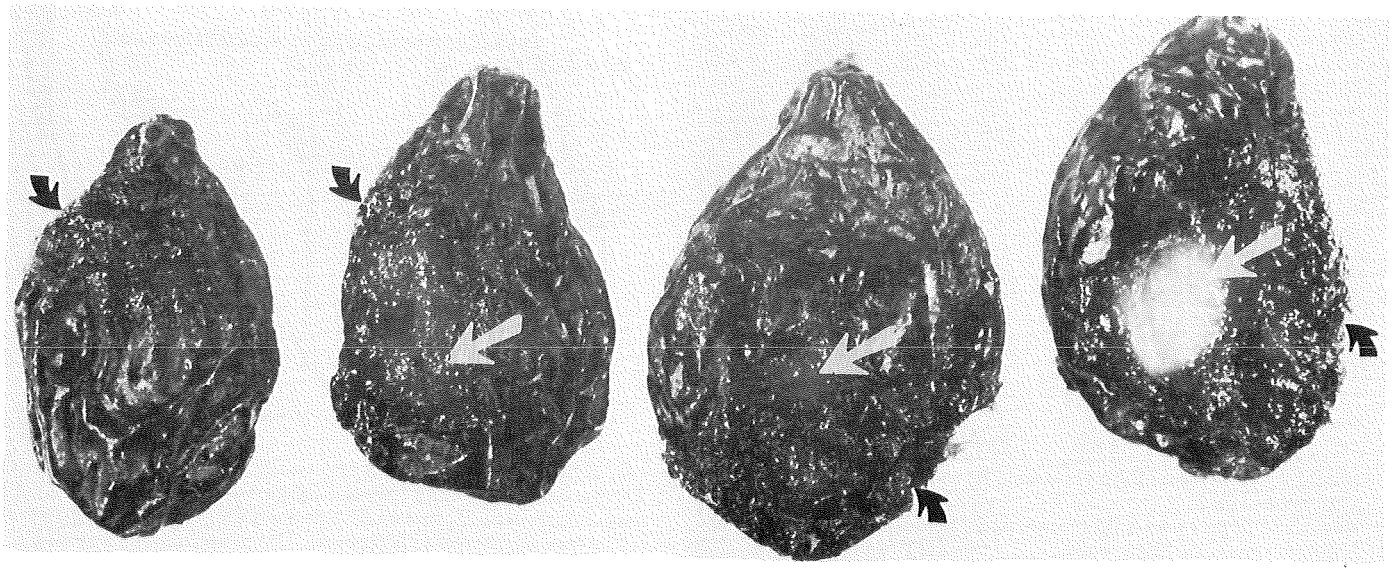

Fig. 1. Prunes from commercial sources showing symptoms of slip-skin maceration disorder. The affected areas indicated by arrows are mushy and sticky. Skin near macerated areas slips with the slightest pressure.

of each section served as the control (Fig. 2). Fruit surrounding the inoculated fruit in each section of each bin were sampled after 24, 48, and 72 hr. Nine hundred fruit were removed from each bin for sampling: 300 surrounding the *Rhizopus*-inoculated fruit, 300 surrounding the *Monilinia*-inoculated fruit, and 300 from the middle of each section. Fruit were examined for visible mold, placed in sterilized net bags (50 per bag), and commercially dried. After drying, prunes were bulked into 300-lot samples each representing 24-, 48-, and 72-hr treatments from which random samples of 50 prunes were removed for evaluation of SSM disorder.

Enzyme study. Fresh prune fruit were vacuum infiltrated for 2 min with a solution containing 1 mg/ml of pectinase (polygalacturonase, E.C. 3.2.1.15, Sigma Chemical Co., St. Louis, MO 63178) to determine whether pectinase could cause SSM disorder. The fruit were dried and tested for the slip-skin condition by applying a slight pressure to the dried skin.

Pectinase was produced from fruit in the following manner: approximately 10 fresh fruit were surface-sterilized with 0.5% sodium hypochlorite for 3 min, placed in 0.5-L jars and punctured with a 0.6-cm diameter glass rod, and finally inoculated with a 10-ml spore suspension of the test fungus. Jars were sealed with filter paper, placed in plastic bags, and incubated at 28 C for 60 hr before the juice was collected by gently squeezing the fruit and straining it through cheesecloth. Pectinase was produced by *Rhizopus* spp. on inoculated, diced, healthy fruit that had been autoclaved for 15 min to be absolutely certain of the enzyme source. Pectinase was obtained from diseased, dried prunes by soaking the fruit for 16 hr in 0.01 *M* phosphate buffer (pH 7.2) and stirring them occasionally. Enzyme filtrates were prepared in the same manner as that used by Strand et al (15).

Pectinase activity was measured initially by the cup-plate test adapted from Dingle et al (3). If any activity was detected, the viscometry method was used. Activity was measured as the initial, approximately linear decrease in viscosity, in centipoise per minute, of 1.4% (w/v) sodium polypectate (Sunkist product 6024) buffered at pH 5.0 with 0.1 *M* sodium acetate. Assay was at 30 C.

The effect of heat on pectinase from fresh fruit infected with *Rhizopus* was compared to the effect of heat on pectinase from dried fruit with the disorder. The various enzymes were heated for 10 min at temperatures of 40, 50, 60, 70, 80, 90, and 100 C, cooled in cracked ice, and stored at 0 C for viscometric analysis.

TABLE 1. The percent decay of fresh prune fruit inoculated with various fungi and incubated at 28 C for 24, 48, and 68 hr and the percent slip-skin maceration (SSM) disorder of the dried prunes

	24 hr		48 hr		68 hr	
Fungus	Decayed[a]	SSM disorder[b]	Decayed	SSM disorder	Decayed	SSM disorder
Rhizopus arrhizus #3	5.00	14.0	25.0	35.0	50.0	94.0
R. stolonifer	0.0	2.0	7.5	25.0	75.0	74.0
R. arrhizus #2	0.3	0.0	4.0	14.0	35.0	48.0
Aspergillus japonicus	0.0	0.0	2.0	8.0	33.3[c]	40.0
R. arrhizus #1	0.0	0.0	3.0	4.0	40.0	38.0
Cladosporium herbarum	0.0	0.0	0.0	2.0	13.3[c]	10.0
Monilinia laxa	0.0	0.0	0.3	0.0	3.3	10.0
Penicillium expansum	0.0	0.0	3.3	4.0	5.7[c]	10.0
Mucor circinelloides	0.0	0.0	0.0	4.0	1.7	6.0
M. fructicola	0.0	0.0	0.0	12.0	4.0	6.0
Botrytis cinerea	0.0	0.0	0.0	2.0	0.3	4.0
A. chevalieri	0.0	0.0	0.0	0.0	1.7	4.0
Alternaria alternata	0.0	0.0	0.0	4.0	3.3[c]	2.0
Noninoculated #1[d]	0.0	0.0	0.0	0.0	0.0	2.0
Noninoculated #2[e]	3.3[c]	10.0	10.0	12.0	17.7	58.0

[a] Number of fruit decayed were estimated by counting the number of fruit with broken skins.
[b] Number of prunes with SSM disorder were rated by industry inspectors from a sample of 50 dried prunes.
[c] This fruit was contaminated with visible *Rhizopus* mycelium.
[d] Noninoculated #1 prunes were surface sterilized before incubation.
[e] Noninoculated #2 prunes were incubated without surface sterilization.

RESULTS

Inoculation study. *R. arrhizus* #3 caused decay of 5% of the fresh fruit within 24 hr, whereas the other genera of fungi tested did not cause appreciable decay (Table 1). When these fruit were dried, 14% of the *R. arrhizus* #3 and 2% of the *R. stolonifer*-infected fruit displayed SSM disorder. Within 48 hr of incubation, both species of *Rhizopus* had caused considerable decay. At this time, fruit inoculated with *A. japonicus* and *P. expansum* began to show some decay. When these fruit were dried, 35% of the *R. arrhizus* #3-inoculated fruit and 25% of *R. stolonifer*-inoculated fruit had SSM disorder. *M. fructicola* caused SSM disorder in 12% of the fruit, and the other fungi caused a lesser degree of SSM disorder. After 68 hr, up to 75% of the *Rhizopus*-inoculated fruit showed evidence of decay. In all cases in which a high percentage of the fruit was decayed, *Rhizopus* mycelium was observed. After the fruit were dried, *R. arrhizus* #3 had caused 94% SSM disorder and *R. stolonifer* 74%. The 40% SSM disorder in the *A. japonicus* sample was in a large part due to *Rhizopus* contamination since 16.7% of the fresh fruit was covered with *Rhizopus* mycelium, whereas in treatments not contaminated with *Rhizopus* only a small percentage of the dried fruit had SSM disorder.

Fruit surface-sterilized and not inoculated remained free of decay, and only 2.0% SSM disorder was observed after 68 hr of incubation at 28 C (Table 1). On the other hand, SSM disorder was observed on 10% of the fruit naturally contaminated with *Rhizopus* after 24 hr and on 58% after 68 hr.

R. arrhizus #3 caused more SSM disorder on injured than on uninjured fruit after 24 hr (Fig. 3). However, injury did not appear to cause higher rates of SSM disorder after 48 and 68 hr. For example, there was less SSM disease of fruit injured and inoculated

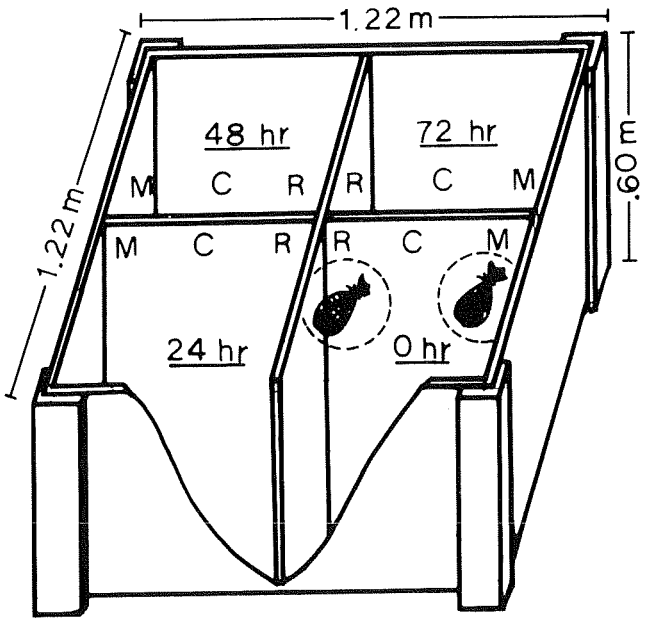

Fig. 2. Schematic of a harvest bin used for incubating prune fruit from Tulare, Yuba City, and Healdsburg for various periods at ambient temperatures. The incubation periods and location of noninoculated fruit (C) or fruit inoculated with *Monilinia* (M) or *Rhizopus* (R) are shown as indicated by the bags. At the end of the incubation periods noninoculated fruit from these three areas were removed for drying and subsequent assessment of slip-skin maceration disorder.

with *R. arrhizus* #3 after 68 hr than of uninjured fruit.

Field study. Flesh firmness of the fresh fruit from the three prune fruit-growing areas of California averaged 1.27 kg for Healdsburg, 2.63 kg for Tulare, and 2.77 kg for Yuba City.

Samples taken immediately after the bins were filled indicated that of the fruit from Tulare and Yuba City, 8.2 and 3.8%, respectively, had SSM disorder, whereas the Healdsburg fruit was free of SSM disorder (Table 2). Part of the 8.2% fruit with SSM disorder in the Tulare lot could possibly be attributed to preharvest *Monilinia* decay because fruit infected with this fungus were observed.

After the fruit were held for 24 hr, fruit from the center of bins where no inoculated fruit had been placed were sampled, examined for decay, and dried. No SSM disorder on the Yuba City fruit was observed after drying, but SSM disorder occurred on 4% of the prunes in the Healdsburg and Tulare bins. After 48 hr, a significant increase (17%) in SSM disorder was observed on fruit in the Healdsburg bin but no increase in the Tulare (4.2%) and Yuba City (0.0%) bins. *Rhizopus* mycelium was observed on the fresh fruit sampled from the control section of the Healdsburg bin. After 72 hr in all three bins, a large increase in SSM disorder of dried prunes was observed in the control section. Mycelium of *Rhizopus* had been observed on this fruit sampled from the control section before drying (Table 2).

In the *Monilinia*-inoculated area of each bin, fruit incubated for 24 hr showed slightly more SSM disorder after drying than observed in the noninoculated control area. After 48 hr, there was a small increase and after 72 hr, a large increase in SSM disorder on prunes sampled from the Tulare (24.0%) and Healdsburg (30.0%) bins. Before drying, *Rhizopus* mycelium was observed on these samples but not on the Yuba City sample, in which only 7.8% SSM disorder of the fruit was observed (Table 2).

The amount of SSM disorder observed in the *Rhizopus*-inoculated area of each bin of fruit incubated for 24 hr varied little from that of the control in all three cases. After 48 hr, large increases in SSM disorder occurred in all three bins. *Rhizopus* mycelium had been observed before drying on fruit samples from the Tulare and Healdsburg bins; these samples contained large numbers of prunes with SSM disorder. After 72 hr, the Yuba City

TABLE 2. Percent slip-skin maceration disorder of noninoculated dried prunes from the same bins with *Monilinia*- and *Rhizopus*-inoculated fresh prunes harvested from three geographical areas

Incubation period before drying (hr)	Tulare			Yuba City			Healdsburg		
	C[a]	M[b]	R[c]	C[a]	M[b]	R[c]	C[a]	M[b]	R[c]
0	8.2[d]	...	...	3.8	...	...	0.0	...	...
24	4.0	6.0	10.0	0.0	2.0	0.0	4.0	6.2	1.9
48	4.2	12.0	34.0[e]	0.0	5.0	18.0	17.6[e]	9.8	38.5[e]
72	28.8[e]	24.0[e]	24.5[e]	11.8[e]	7.8	30.0[e]	28.0[e]	30.0[e]	75.0[e]

[a] C = Control. Each bin section (Fig. 2) was divided into three parts, with the middle noninoculated fruit designated as control.

[b] M = *Monilinia* spp. Twenty-five freshly harvested ripe fruit were inoculated with *M. fructicola* and *M. laxa* and placed in each section.

[c] R = *Rhizopus* spp. Similar to the preceding, except that *R. arrhizus* and *R. stolonifer* were used as inoculum.

[d] This initial slip-skin maceration disorder may have been due to brown rot since some fruit were observed to be naturally infected with *Monilinia*.

[e] *Rhizopus* mycelium was observed on the fresh prunes prior to drying.

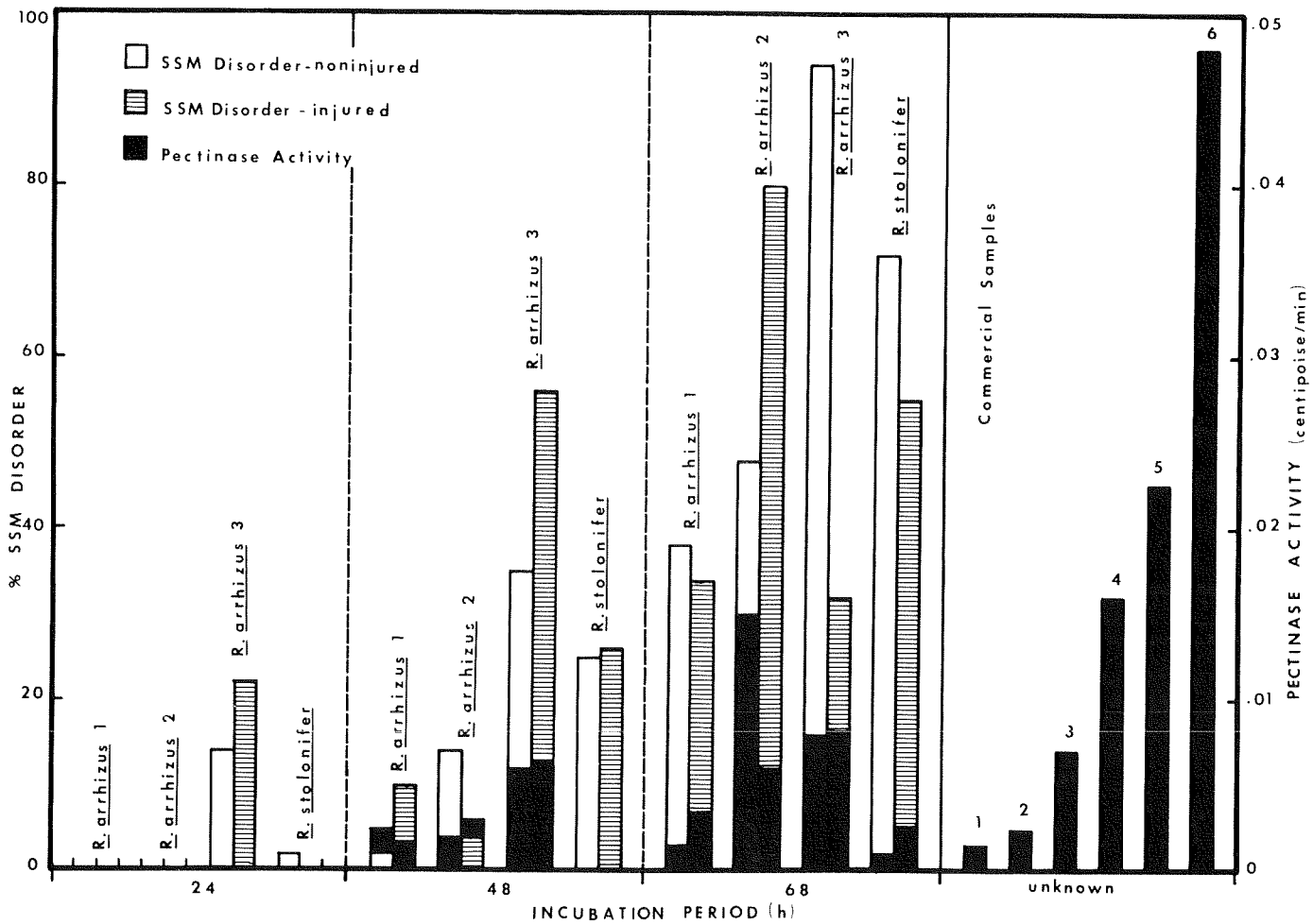

Fig. 3. Slip-skin maceration disorder of dried, injured, and uninjured prunes that had been inoculated with *Rhizopus* spp. and incubated for 24, 48, and 68 hr at 28 C before drying. Pectinase activity of prunes inoculated with *R. arrhizus* after 48 and 68 hr incubation and pectinase activity of prunes inoculated with *R. stolonifer* after 68 hr of incubation. The pectinase activity of six commercial samples with slip-skin maceration disorder are also shown.

and Healdsburg samples contained the largest number of prunes with SSM disorder, 30 and 75%, respectively. The 48-hr sample from the Tulare bin contained more prunes graded as having SSM disorder than the 72-hr sample. This difference may be attributed to sampling method. Where small numbers of *Rhizopus*-inoculated fruit were placed in each of the three bins and held either 48 or 72 hr before drying, significantly higher numbers of adjacent noninoculated fruit became infected and, consequently, larger numbers of dried prunes were found to have SSM disorder (Table 2).

Enzyme study. Absorption of pectinase (purchased from the Sigma Chemical Co.) into fresh prunes by vacuum infiltration produced SSM disorder on prunes upon drying. Thirty-four of 70 prunes exhibited a slip-skin condition, whereas none of the prunes infiltrated with pure water showed this symptom.

A. japonicus was the most prodigious producer of pectinase on fresh fruit (Table 3). The two species of *Rhizopus* studied were also good producers of pectinase, with *R. arrhizus* #2 producing almost as much activity as *A. japonicus*. Enzyme activity was less in *R. stolonifer* than *R. arrhizus* but was about equal to *P. expansum*.

The amount of pectinase detected in prunes with SSM disorder varied in samples taken at various times from a central processing plant (Table 4). The viscometry and cup-plate tests indicated that four of the six samples with SSM disorder had pectinase activity, whereas two had no significant pectinase activity.

Fresh fruit inoculated with *Rhizopus* and then dried displayed pectinase activity (Fig. 3). Samples 3 and 4 with SSM disorder obtained from commercial sources had pectinase activities similar to that of the *R. arrhizus*-infected fruit, and activities of commercially obtained samples 1 and 2 with SSM disorder were similar to that of the *R. stolonifer*-infected fruit after 68 hr of incubation.

Subjecting *R. arrhizus* crude enzyme to temperatures from 40 to 100 C resulted in a heat inactivation curve similar to that found for a preparation from one of the commercially obtained samples (Fig. 4). *R. stolonifer* crude enzyme lost nearly all its activity after being heated to only 50 C.

TABLE 3. Pectinase activity of crude enzyme from fresh fruit inoculated with specific fungi and incubated for 60 hr at 28 C

Fungus	Pectinase activity	
	Cup-plate[a] (mm)	Viscometry[b] (cp/min)
Noninoculated	0	−0.0015 V
Monilinia fructicola	2	−0.0019 V
M. laxa	3	−0.0035 V
Penicillium expansum	9	−0.0392 W
Rhizopus stolonifer	7	−0.0401 W
R. arrhizus #3	12	−0.1141 X
R. arrhizus #2	13	−0.1466 Y
Aspergillus japonicus	15	−0.2364 Z

[a] Diameter of cup subtracted from diamenter of cup plus pectinase induced halo.
[b] Values in the viscometry column not followed by the same letter are significantly different ($P = 0.05$).

TABLE 4. Pectinase activity of crude enzyme from dried prunes with SSM disorder obtained from commercial sources

Sample	Pectinase activity	
	Cup-plate[a] (mm)	Viscometry[b] (cp/min)
Healthy[c]	0	−0.0013 V
SSM disorder #1	0	−0.0012 V
SSM disorder #2	2	−0.0024 V
SSM disorder #3	3	−0.0070 W
SSM disorder #4	5	−0.0158 X
SSM disorder #5	8	−0.0226 Y
SSM disorder #6	9	−0.0479 Z

[a] Diameter of cup subtracted from diameter of cup plus pectinase induced halo.
[b] Values in the viscometry column not followed by the same letter are significantly different ($P = 0.01$).
[c] Dried prunes free of visible defects.

DISCUSSION

Evidence has been provided that links SSM disorder of dried prunes to infection by *Rhizopus* spp. We have shown how the condition can be reproduced within 24 hr by inoculating fresh fruit with *Rhizopus* spp. and drying them in the normal manner (Table 1). Field studies indicated that *Rhizopus* spp. will spread quickly throughout a bin of fresh fruit if left for 48 hr under normal prune harvest temperature conditions before drying (Table 2). Furthermore, we found similar pectinase activity in a crude enzyme extract from prunes with SSM disorder from a commercial source and from fruit we had inoculated with *Rhizopus* (Fig. 3).

Some questions exist regarding the pectinase activity of SSM disorder in dried prunes from natural sources and those produced by inoculating with *Rhizopus*. For example, the prunes with SSM disorder do not always contain appreciable enzyme activity (Fig. 3). We found that although *R. stolonifer* caused 74% of the dried prunes to have SSM disease, pectinase activity was barely measurable. Apparently, most of the pectinase produced by the fungus in and on the fruit is destroyed by heat during dehydration, which explains why it is not detected in all samples. Pectinase produced by *R. stolonifer* was almost completely inactivated by 50 C (Fig. 4). On the other hand, sample #6 with SSM disorder (Fig. 3) had an exceptionally high pectinase activity, indicating that some of the fruit in this sample may have been decayed by *Aspergillus* spp. or *Penicillium* spp., or both, and not dried at a high enough temperature to inactivate all the enzyme. *A. japonicus* produced greater pectinase activity than *Rhizopus* spp. when it was allowed to grow on fresh prunes (Table 3). Another possibility is that this fruit was decayed by *R. arrhizus*, which produces a more heat-resistant pectinase (Fig. 4). Luh et al (7) found that pectinase from apricots rotted by *R. arrhizus* was heat resistant. Thus, more pectinase would remain after dehydration, the amount depending on the effective dehydration temperature.

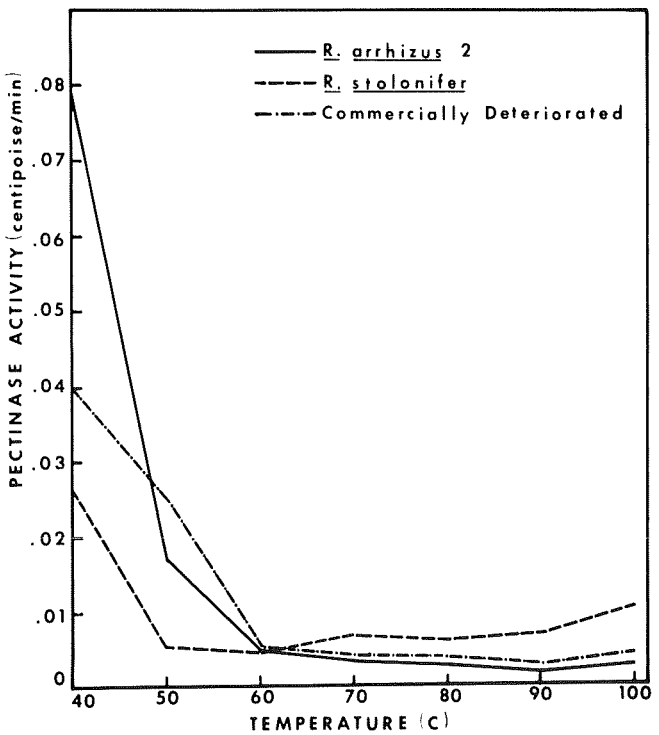

Fig. 4. Effect of heating (10 min at 40–100 C) on activity of pectinase produced by *R. arrhizus* #2 and *R. stolonifer* in fresh fruit, and on that found in commercial sample #5 with slip-skin maceration disorder (Table 4).

Spores of *A. chevalieri* and *A. japonicus* are often found on prunes after drying (5). When we inoculated fruit with *A. chevalieri*, the most common fungus found on dried prunes (4), we detected no decay of the fresh fruit and decay of only dried prunes of high moisture content (25%) after several days of storage (13). This decay was always accompanied by green sporulation or yellow cleistothecia characteristic of *A. glaucus* group fungi of which *A. chevalieri* is a member. *A. japonicus* did cause some decay of the fresh prunes and may be responsible for some fruit with SSM disorder if the prunes are held at high temperature (25–30 C) for three or more days before drying. This fungus is not the primary cause of SSM disorder because of the restricted type of rot it produces on the fruit and the long period of time it takes to cause damage.

Monilinia spp. have been suggested as the chief causal agents of the condition under investigation (2). We were unable to obtain typical symptoms and enough affected prunes by inoculation with these fungi within 24 hr of storage to support this contention. Perhaps when *Monilinia* spp. occurred in combination with *Rhizopus* spp., the two were confused, and the decay in fruit with SSM disorder was attributed to the *Monilinia* spp. Generally, decay caused by *Monilinia* spp. can be distinguished from that caused by *Rhizopus* spp. because *Monilinia* spp. produce sporodochia on the fruit that are evident even after drying.

The amount of fruit decay caused by *Rhizopus* and, subsequently, SSM disorder on dried prunes in a bin, depends on the species of fungus, temperature of the fruit, amount of injured fruit, fruit maturity, humidity of the air surrounding the fruit, and the length of time the fungus grows on the fruit before dehydration. *Rhizopus* spp. can be divided into two groups based on the highest temperature at which they grow. We found that the *R. arrhizus* produced more pectinase than the more common low-temperature *R. stolonifer* (Table 3). This result appears to coincide with an earlier and more extensive decay and subsequent SSM disorder on dried fruit caused by *R. arrhizus* (Table 1, Fig. 3). Temperature of fruit in the bin is important in determining which *Rhizopus* species will cause decay and how fast decay will progress. On potato-dextrose agar, mycelium of *R. stolonifer* grows optimally at 25–27 C with a maximum at 32.2 C and minimum at 4.4 C (11), whereas *R. arrhizus* grows optimally at 35–36 C (18). Fruit injury is important since *Rhizopus* infection initially occurs only on injured tissue (19). Our experiments to demonstrate that more SSM disorder develops on mechanically injured fruit was inconclusive (Fig. 3). Apparently, there was enough injury to the fruit during harvest that this additional injury was not a factor. *R. stolonifer* requires a minimum RH of 92–94% for development, with the optimum RH being 98% under controlled laboratory conditions on nutrient media (10). High RH is almost assured in a bin of mature, respiring fresh fruit. The fungus grows out from single-infection foci, releasing enzymes that act to soften the skin of the infected fruit. The mycelium arising from an infected fruit or from a sporangiospore can penetrate nonwounded healthy fruit in contact with infected fruit (1). The longer *Rhizopus* spp. are allowed to grow, the greater will be the amount of fruit decay and subsequent SSM disorder on dried prunes. Our experiments show that SSM disorder increased over time when the fruit was inoculated with *Rhizopus* spp. (Table 1) and when bins containing *Rhizopus*-inoculated and noninoculated fruit were held at high temperature 48–72 hr before the fruit was dried (Table 2).

LITERATURE CITED

1. Agrios, G. N. 1978. Plant Pathology. Academic Press, New York. 703 pp.
2. Archer, T. E., and Corbin, J. B. 1969. The site and fate of captan residues from dipping prunes prior to commercial dehydration. Food Technol. 23:101-104.
3. Dingle, J., Reid, W. W., and Solomans, G. L. 1953. The enzymatic degradation of pectin and other polysaccharides. II. Application of the 'cup-plate' assay to the estimation of enzymes. J. Sci. Food Agric. 4:149-155.
4. El-Behadli, A. H. 1976. Mold contamination and infection of prunes and their control. Ph.D. dissertation. University of California, Davis. 80 pp.
5. El-Behadli, A., and Ogawa, J. M. 1974. Occurrence and survival of molds on fruit in California prune orchards and dehydrators. Proc. Am. Phytopathol. Soc. 1:43.
6. Gentry, J. P., Miller, W. W., and Claypool, L. L. 1965. Engineering and fruit quality aspects of prune dehydration in parallel and counter flow tunnels. Food Technol. 19(9):121-125.
7. Luh, B. S., Ozbilgin, S., and Liu, Y. K. 1978. Textural changes in canned apricots in the presence of mold polygalacturonase. J. Food Sci. 43:713.
8. Miller, M. W., and Tanaka, H. 1963. III. Relation of equilibrium relative humidity to potential spoilage. Hilgardia 34:183-190.
9. Mitchell, G. F., Sibbett, G. S., and Mayer, G. 1981. Cold storage of French prunes may expand dehydrator capacity. Calif. Agric. 35:20-22.
10. Panasenko, V. T. 1967. Ecology of microfungi. Botan. Rev. 33:189-215.
11. Pierson, C. R. 1966. Effect of temperature on growth of *Rhizopus stolonifer* on peaches and agar. Phytopathology 56:276-278.
12. Rodda, E. D., and Gentry, J. P. 1969. New concepts in fruit dehydrator construction. Am. Soc. Agric. Eng. 12(4):540-541.
13. Sholberg, P. L. 1981. Relation of postharvest decay fungi to the deterioration of dried French prunes. Ph.D. dissertation. University of California, Davis. 53 pp.
14. Sholberg, P. L., and Ogawa, J. M. 1981. Deterioration of dried French prunes related to postharvest decay fungi. (Abstr.) Phytopathology 71:904.
15. Strand, L. L., Ogawa, J. M., Bose, E., and Rumsey, J. W. 1981. Bimodal heat stability curves of fungal pectolytic enzymes and their implications for softening of canned apricots. J. Food Sci. 46:498.
16. Tanaka, H., and Miller, M. W. 1963. Microbial spoilage of dried prunes. Hilgardia 34:171-181.
17. Thompson, J. 1981. Methods of prune dehydration. Pages 150-152 in: Prune Orchard Management, University of California Press, Berkeley. 156 pp.
18. Weimer, J. L., and Harter, L. L. 1923. Temperature relations of eleven species of *Rhizopus*. J. Agric. Res. 24:1-40.
19. Wilson, E. E., and Ogawa, J. M. 1979. Fungal, Bacterial, and Certain Nonparasitic Diseases of Fruit and Nut Crops in California. University of California Press, Berkeley. 190 pp.