Wind Scab of French Prune: Symptomatology and Predisposition to Preharvest and Postharvest Fungal Decay

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ABSTRACT

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Severe scabbing, termed wind scab (WS), of French prune (*Prunus domestica* 'French') was caused by developing fruit rubbing against other fruit, leaves, and shoots during strong wind gusts prevailing from north to northwest. WS occurred only during years in which north or northwest winds exceeded 20 km/hr for at least 10 days within 3 wk after full bloom. The affected areas developed several layers of cutinized cells, the outer layers showing deep fractures that retained moisture and facilitated germination of fungal spores and penetration by their germ tubes. Both incidence and severity of WS on mature fruit correlated positively (R^2 = 0.72–0.91 and 0.94, respectively) with the incidence and severity of WS on dehydrated fruit. Surfaces of wind-scabbed areas acted as traps in collecting fungal propagules. The incidence of decay caused by *Phomopsis cinerascens* was significantly higher in fruit with WS. Although germinated spores of *P. cinerascens* could penetrate directly through the epidermal layers of unwounded ripe and overripe prune fruit, wounds or cracks on wind-scabbed fruit facilitated infection. WS resulted in yearly average percentages of off-graded prunes equivalent to those of russet scab, as determined by commercial inspectors.

Additional keywords: environment, wind bruise

Prunes (*Prunus domestica* L.), mostly of the French variety, are produced in California and a few other areas around the world (1). Over 95% of the prunes produced in California are dried for domestic and foreign markets. Prune fruit ideal for drying have a smooth, waxy skin that does not have any blemishes or other defects and is transformed to a wrinkled, shiny, dark brown to purplish surface after dehydration. When blemishes from insect feeding, russet scab (RS) (8), end and side cracks (4), or wind scab (wind bruise) occur, fruit are downgraded and used for juice. In two prune orchards in 1988, we observed that an unusually high number of fruit decayed by *Phomopsis cinerascens* (Sacc.) Bubák in Bubák & Kab. had associated wind scab (*unpublished*).

Both plum (*P. domestica* 'Friar') (5) and prune seem to be relatively sensitive to winds. Scars caused by wind rubbing fruit against other fruit, leaves, small twigs, or branches create russeted areas that become very scabby and resemble RS after dehydration (8). Typical symptoms of RS of immature prune fruit originate as shiny, lacy areas on the stylar end of fruit and become brown and scabby on ripe fruit (8). RS has been most severe when rainfall has occurred during and 1 wk after full-bloom stage (8). In years with rains in March (scab years), RS can cause significant losses (2). In this study, symptoms of wind bruise damage of prune fruit are referenced to "wind scab" (WS) to differentiate the condition from RS.

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WS caused considerable economic losses to California's prune industry in 1984, 1988, and 1991. Especially in 1988, we noticed an unusually high number of prunes decaying on the tree and found that this decay was associated with wind-scabbed areas. Dark brown decay lesions usually developed at the margins of wind-scabbed areas or sometimes in cracks or russet-scabbed areas. Because WS can be easily confused with RS, the purpose of this study was to characterize WS in detail and determine specific symptoms for diagnosing it and distinguishing it from RS. In addition, we investigated the predisposition of wind-scabbed fruit to postharvest decay by various fungi.

MATERIALS AND METHODS

Symptomatology and light microscopy. Young and ripe fruit were photographed to illustrate the damage on prune fruit created by wind. For light microscope studies, healthy fruit and fruit with WS symptoms were sectioned in pieces 5 × 5 × (3–5) mm (including the outer surface), fixed in 2% glutaraldehyde, washed, dehydrated, infiltrated with paraffin oil, sectioned at approximately 10 μm thickness with a microtome, and stained with Sudan IV, using methods described elsewhere (8). The sections were mounted in a solution of 0.2% phenol and 50% glycerol, observed at 320×, and photographed.

Scanning electron microscopy (SEM). Both green and ripe healthy prune fruit and fruit with WS symptoms were sectioned into 15- to 20-mm^2 sections (including the outer surface) and prepared for SEM using methods described elsewhere (8).

Relationship of WS before and after dehydration. To determine the relationship between symptoms of WS on mature fruit and symptoms after dehydration, samples of 50 prune fruit each were harvested from 24 trees not sprayed with chemicals or water and separated according to the length of wind abrasion into five severity categories, on a scale of 0–4 (Fig. 1A), where 0 = healthy (no abrasions), 1 = abrasions 2–4 mm long, 2 = abrasions 5–8 mm long, 3 = abrasions 9–12 mm long, and 4 = abrasions ≥13 mm long. After evaluation, samples were placed in plastic net bags and dried in a commercial dehydrator at 80 C for 18 hr, then evaluated according to the severity scale (Fig. 1B). The experiment was repeated in a second unsprayed prune orchard with samples of 100 mature prunes harvested from each of 41 trees selected arbitrarily. Data were analyzed by the REG procedure of SAS (11).

Effects of winds on WS and decay of prune fruit. Wind speeds and directions were retrieved (CIMIS Weather Service) for 1982 through 1991 from a weather station (Davis.A; Stn. Code CAYOAAA2) adjacent to the experimental plots. In 1984, 1988, and 1991, three to six samples of 100 ripe fruit were collected in September from the north or the south side of three to six replicate trees in two rows of three orchards (orchards 1, 2, and 3) located in Davis, California, and evaluated for incidence and severity of WS before and after dehydration. The percentage of fruit with decay initiated from WS was also recorded in samples collected from orchards 1 and 3. To identify the fungi present in tissues damaged by WS, surfaces of fruit collected from orchards 1 and 2 were disinfested in 0.084% NaOCl for 1 min, and isolations were made from decay lesions associated with WS, stylar-end cracking, and areas with no macroscopically obvious wounds. Isolations were made on acidified (2.5 ml of a 25% [v/v] lactic acid solution per liter of medium) potato-dextrose agar (APDA)

(five isolations per dish), and incubated at 23 ± 1 C for 5–7 days.

Mycoflora associated with wind-scabbed and healthy fruit. The surface mycoflora associated with healthy fruit and fruit with WS were determined in a commercial orchard in Fresno County. Ten fruit per each of six samples, selected arbitrarily, were placed in a plastic bottle with 200 ml of deionized water and shaken for 2 hr in a two-cycle shaker. Aliquots of 100 μl were plated on each of five APDA dishes and incubated at 23 ± 1 C. Colonies of fungi that developed were counted after 5–6 days. The experiment was repeated.

In another experiment, 16 disks (1 cm diameter) were cut with a cork borer from eight healthy and eight wind-scabbed fruit selected arbitrarily and placed in a 2.5 × 20 cm test tube to which 15 ml of sterile deionized water was added. The disks were cut from the scabby areas on the wind-scabbed fruit. Each test tube was vortexed for 1 min, then 100 μl of the washings was plated on five APDA dishes. The dishes were incubated at 23 ± 1 C, and fungi that developed were identified and recorded after 5 days of incubation. There were three test tube replications of eight fruit for each category of fruit, and the experiment was repeated.

To determine the mycoflora colonizing wind-scabbed areas, 30 healthy and 30 symptomatic fruit were surface-disinfested in 0.084% NaOCl solution for 1 min, rinsed in sterile water, and allowed to dry on sterile paper towels. Ten pieces, 2–4 mm^2 per fruit and including the epidermal layers, were removed with fine tweezers from both wind-scabbed and healthy areas and placed in APDA dishes. Fungi that developed from plated epidermal tissues were identified and recorded after 5 days of incubation at 23 ± 1 C. The experiment was repeated twice.

Isolations were also made from prune fruit collected from orchard 1 with decay lesions associated with WS and stylar-end and side cracks. Three replicates each of 10 fruit were used in each experiment.

Inoculation of healthy fruit and wind-scabbed fruit with *P. cinerascens*. To determine whether WS predisposed fruit to infection by *P. cinerascens*, the fungus most often isolated from decaying fruit, healthy and wind-scabbed (severity 3 and 4) fruit were inoculated in the laboratory. All picked fruit were surface-disinfested by dipping them for 3–4 min in a solution containing 0.084% NaOCl. Fruit with WS were allowed to dry, placed on a waxed wire screen in plastic containers (23 × 32 × 10 cm) (24 fruit per container), and inoculated on the margins of wind-scabbed areas with 40 μl of a 10^5 spores per milliliter suspension of *P. cinerascens*, prepared from a 10-day colony grown on PDA. In addition, healthy ripe and overripe fruit were inoculated after being wounded with a sharp glass rod 2 mm in diameter. Healthy ripe and overripe unwounded and inoculated fruit served as controls. To maintain a saturated atmosphere, 300 ml of distilled water was placed in each container. Seventy-two hours after inoculation with *P. cinerascens*, fruit were prepared for SEM observation as previously described (8). There were four replications for each treatment, and the experiment was repeated twice.

Pathogenicity tests with other fungi commonly associated with WS. Pathogenicity of six fungi commonly isolated from prunes (especially from decay associated with wind-scabbed areas) was tested on mature, ripe, and overripe French prunes harvested from a commercial orchard in Fresno County on 1 and 16 September and 2 October, respectively. After harvest and surface-disinfestation in 0.084% NaOCl for 3 min, fruit were allowed to dry, were placed in three plastic containers over waxed wire-mesh (25 fruit per container), and were punctured with a sharp glass rod 2 mm in diameter. Fruit were then inoculated in each wound with 30–40 μl of a suspension containing (concentration of spores per milliliter of fungus) 2×10^4 of *Monilinia fructicola* (G. Wint.) Honey, 10^5 of *P. cinerascens*, and 1.5–2 $\times 10^5$ each of *Aspergillus melleus* Yukawa, *Cladosporium herbarum* (Pers.:Fr.) Link, *Penicillium expansum* Link, and a *Fusarium* sp. in each wound. Other sets of fruit were prepared and inoculated similarly but were not wounded. Because of the hydrophobic, waxy surface of unwounded prunes, drops of inoculum were held on the unwounded fruit in small wells cut from Tygon tubing (5 mm i.d., 3–4 mm high). Inoculated fruit were incubated at 23 ± 1 C. The experiment was repeated once. The percentage of infected fruit and the diameter of decay lesions were measured 3–8 days after inoculation. Inoculated fruit were compared with a pairwise *t* test.

RESULTS

Symptomatology and light microscopy. On green fruit, WS usually occurred on the side of the fruit, appearing as longitudinal, scabby areas with the largest dimension along the axis (stem to stylar end) of the fruit (Fig. 1). In contrast, RS usually occurred in an irregular zone surrounding the stylar end of the fruit (8). In addition, WS was characterized by plaques of necrotic, light brown cells that were attached to the surface of the green fruit. As fruit matured, these areas, usually on the side of the fruit, became distinctly rough and light brown to beige (Fig. 2A) and caused premature fruit shrinkage (Fig. 2B). On mature fruit, WS was easily distinguished from RS by the rougher appearance of affected areas. After dehydration, affected areas became beige, dry, and scabby and lacked flexibility and shininess in contrast to dehydrated healthy fruit (Fig. 1B, category 0).

Cross sections of areas expressing WS showed several layers of cell walls when stained black with Sudan IV (Fig. 3A). The epidermal cells had been killed and

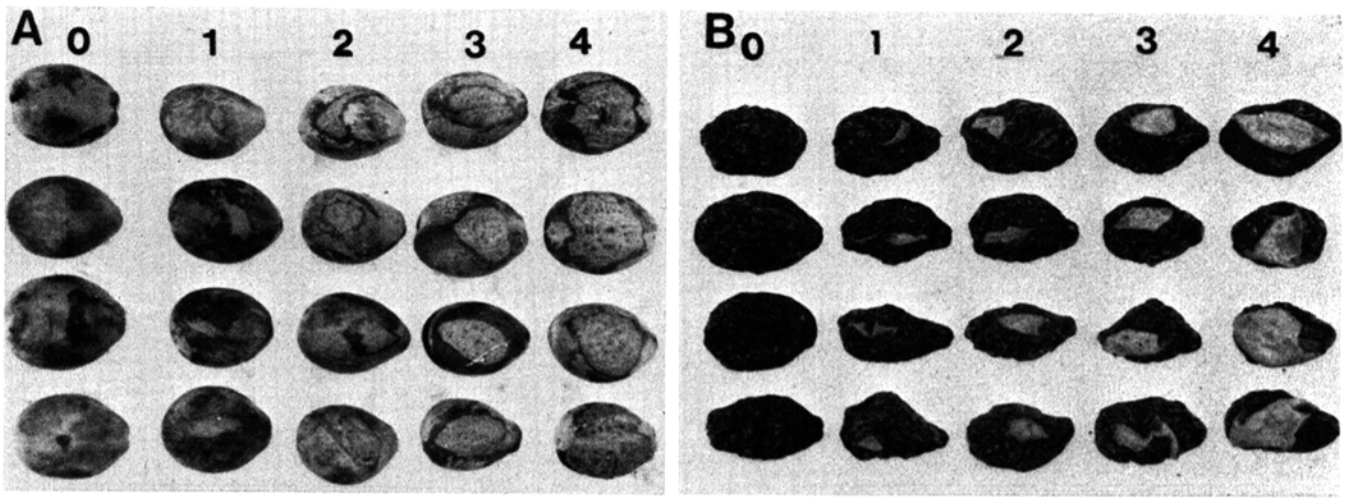

Fig. 1. Wind scab severity index on fresh ripe and dehydrated fruit **(A)** before and **(B)** after dehydration. 0 = Healthy (no abrasions), 1 = abrasions 2–4 mm long, 2 = abrasions 5–8 mm long, 3 = abrasions 9–12 mm long, and 4 = abrasions ≥13 mm long.

could not be distinguished easily. Layers of heavily stained cells (periderm) formed beneath the affected area in the fruit tissue (Fig. 3A). The thickness of these layers was not uniform, and extensive (Fig. 3B) or restricted (Fig. 3C) discontinuities of the cutinized cells were frequently observed.

Scanning electron microscopy. Examination of sections of young fruit with WS indicated that epicuticular strands were still present on the surface of the fruit but were not uniform in density (Fig. 4A). As fruit matured (July–August), cracks and scabbing of the fruit surface became obvious even though epicuticular wax was still accumulated on some areas of the fruit surface (Fig. 4B). WS resulted in exposed, fractured outer layers of mature fruit (August–September) (Fig. 4C). Budding yeast cells (Fig. 4B) and germinated fungal spores (probably of a *Cladosporium* sp.) (Fig. 4C) were found on the surfaces of these cracks.

Relationship of WS before and after dehydration. The incidence (8–72%) of WS after dehydration was directly related to the incidence (10–88%) of WS of mature fruit in both experiments in 1988. The regression line for the first experiment (orchard 1) was $Y = 4.0 + 0.69\ X$ (adjusted $R^2 = 0.72$, $P < 0.001$) and that for the second experiment (orchard 2) was $Y = -8.84 + 0.96\ X$ (adjusted $R^2 = 0.91$, $P < 0.001$), where Y and X represent the incidence of WS expressed as percentage of fruit after and before dehydration of fruit, respectively. In addition, WS severities after (0.02–2.14) and before (0.20–2.18) dehydration of the fruit were also strongly related. The regression equation was $Y = -0.08 + 1.14\ X$ (adjusted $R^2 = 0.94$, $P < 0.001$), where Y and X represent the severity index of WS after and before dehydration of fruit, respectively.

Effects of winds on WS and decay of prune fruit. Speeds of the three strongest winds within 3 wk after full bloom were higher in 1984, 1988, and 1991 than in other years (Table 1). Correspondingly, the levels of WS were high only in 1984, 1988, and 1991; in all other years, the incidence of WS was sporadic. The overall mean levels of off-graded prunes (evaluated by commercial inspectors in a Sunsweet Dehydrator facility at Yuba City, California, located 96 km from Davis) were high for 1982, 1983, 1984, 1988, and 1991 (Table 1). However, the high levels of off-graded fruit in 1982 and 1983 were primarily caused by severe RS (8).

In all three orchards, the incidence and severity of WS before and after dehydration were significantly higher on fruit samples collected from the north side of the trees than on those collected from the south side (Table 2). In addition, fruit harvested from the north side of the trees had a significantly higher incidence of decay of WS areas than fruit harvested from the south side (Table 2).

Fungi isolated from decay lesions associated with WS were primarily *P. cinerascens* and two other *Phomopsis* spp. (84–96% of all isolates); *A. melleus*, characterized by pale yellow conidial heads and abundant golden sclerotia (0–4%); and various yeasts (0–10%). Isolations from decay associated with stylar-end or side cracking revealed a larger diversity of microorganisms, including species of *Penicillium* (25–28%), *Phomopsis* (24%), *Cladosporium* (<10%), *A. melleus* (2–16%), yeasts (0–26%), and

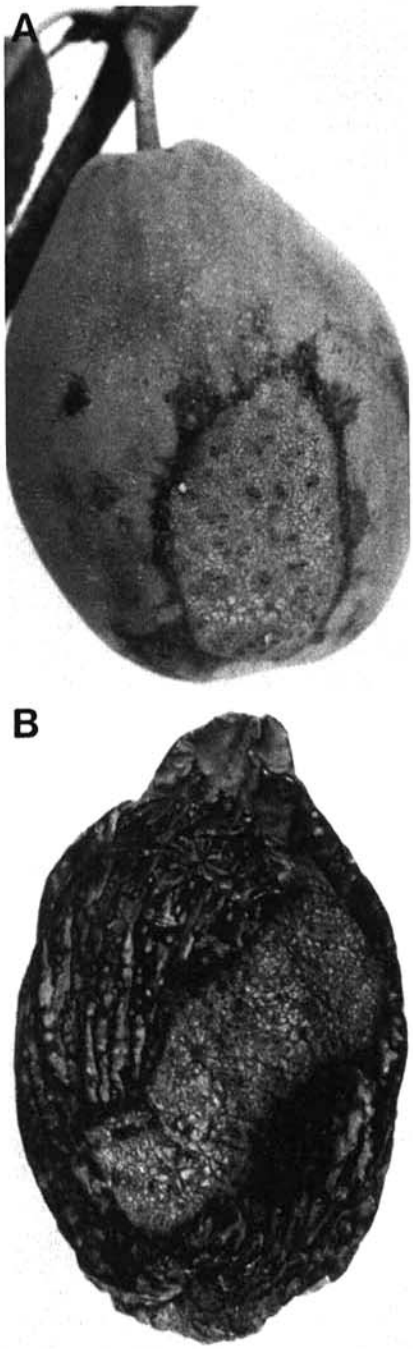

Fig. 2. Symptoms of wind scab on **(A)** ripe fruit (rough and light brown to beige) and **(B)** overmature fruit (dehydrated in the field). (×2)

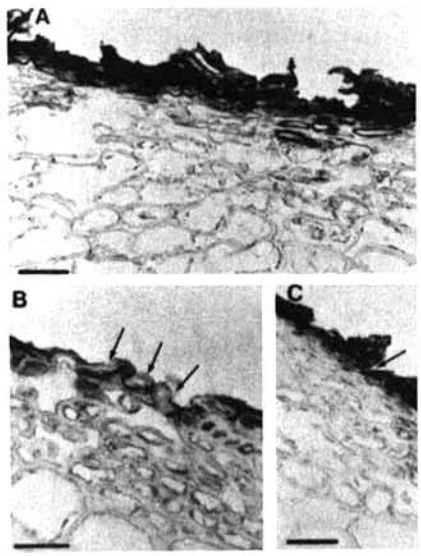

Fig. 3. Cross sections of prunes with wind scab: **(A)** Layers of periderm cells on the outer surface of fruit stained black with Sudan IV, and areas of **(B)** extensive and **(C)** restricted discontinuity of the cutinized layers of cells. Scale bars = 50 μm.

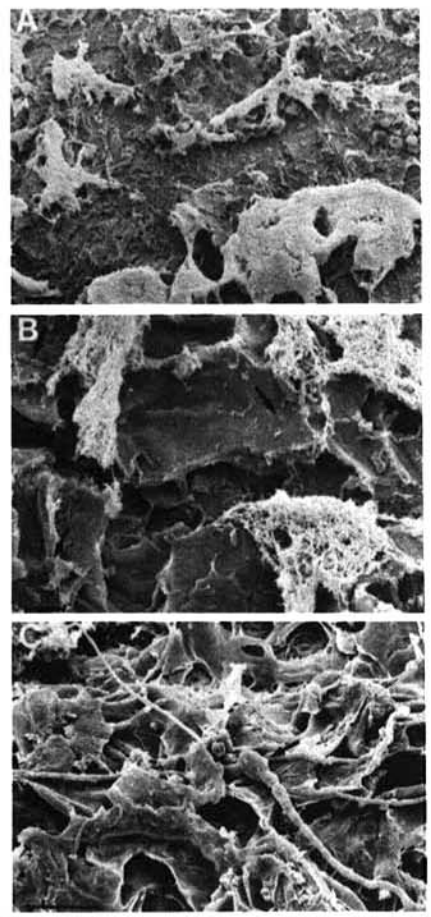

Fig. 4. Scanning electron micrographs of prune fruit with wind scab: **(A)** Dense but not uniform epicuticular wax in young fruit. Scale bar = 100 μm. **(B)** Cracking in young fruit, showing plaques of necrotic cells despite accumulations of epicuticular wax; arrows indicate budding yeast cells. Scale bar = 10 μm. **(C)** Flaking, cracking, and lack of epicuticular wax in ripe fruit; arrow indicates germinated spore of a fungus resembling *Cladosporium* sp. Scale bar = 25 μm.

M. fructicola/*M. laxa* (Aderhold & Ruhland) Honey and *Alternaria alternata* (Fr.:Fr.) Keissl. (each <10%). *Phomopsis* spp. and *A. melleus* were also isolated (66 and 10%, respectively) from decay lesions not associated with any obvious wounds.

Mycoflora associated with wind-scabbed and healthy prunes. The levels of mycoflora recovered from washings of fruit with WS were significantly ($P < 0.01$) greater than those recovered from healthy fruit in all experiments. For instance, 2.14×10^4 propagules of filamentous fungi and yeasts per healthy fruit were recovered contrasted with 1.25×10^5 propagules per WS fruit.

When fruit skin disks were washed, healthy fruit had 52 propagules of mycoflora per square centimeter and wind-scabbed tissue had 4.6×10^3. More than 95% of the propagules were *Cladosporium* spp. (including *C. herbarum*). Other fungi recovered sporadically in dishes containing APDA were *A. alternata*, *Epicoccum purpurascens* Ehrenb., *Penicillium* and *Fusarium* spp., *Mucor genevensis* Lendner, *Aureobasidium pullulans* (de Bary) G. Arnaud, *Aspergillus niger* Tiegh., and *Rhizopus stolonifer* (Ehrenb.:Fr.) Vuill.

After surface disinfestation, 26 and 88% of plated tissues from healthy fruit and fruit with WS, respectively, yielded filamentous fungi and yeasts. Fungi isolated included (percentages of all isolations from healthy fruit/from wind-scabbed fruit): *A. alternata* (17/55%), *E. purpurascens* (2/2%), *Fusarium* spp. (2/9%), *Cladosporium* spp. (5/16%), *A. pullulans* (0/1%), *P. expansum* and other *Penicillium* spp. (0/1%), *A. niger* (0/5%), *R. stolonifer* (0/1%), and *Aspergillus ochraceous* Wilh. (0/1%).

Phomopsis spp. were isolated from 75% of overripe fruit with stylar-end or side-crack decay collected in November 1986 (Table 3). Three morphologically different isolates of *Phomopsis* were recovered at a higher frequency than *P. cinerascens*. *P. cinerascens* was identified after comparison with a known isolate of *P. cinerascens* that causes shoot blight on figs (3). The same *Phomopsis* isolates, including *P. cinerascens*, were also isolated from 55–90% of ripe or overripe prune fruit showing decay in wind-scabbed areas during September 1988. The incidence of *Phomopsis* decay was directly related to the length of time that fruit with WS or stylar-end and side cracks were retained in the field (Table 3). For instance, on 16 September 1988, 55% of the collected ripe prunes had Phomopsis decay, whereas on 19 and 29 September 1988, when fruit were ripe or overripe, 78 and 90%, respectively, contained decay caused by *Phomopsis* spp. (Table 3).

Relationship of WS to fungal decay. Without artificially wounding of the wind-scabbed fruit, the decay by *P.*

Table 1. Effect of north or northwest winds on incidence of wind scab in three French prune orchards at the University of California, Davis, and regional incidence of off-graded prunes from 1982 to 1991[x]

Year	Date of full bloom in March	No. of days with north or northwest winds within 3 wk after full bloom	Speeds of three strongest wind gusts within 3 wk after full bloom (km/hr)	Average wind speed within 3 wk after full bloom (km/hr)	Mean wind scab incidence[y] (%)	Incidence of off-graded fruit[z]
1982	19	5	4.8, 8.0, 22.5	8.4	NE	13.66 (RS)
1983	11	3	4.8, 6.4, 11.3	7.6	NE	10.85 (RS)
1984	14	12	20.9, 22.5, 27.4	14.5	14	10.92 (WS)
1985	19	11	14.5, 17.7, 27.4	10.5	NE	9.94 ...
1986	5	5	11.2, 17.7, 19.3	11.9	NE	9.49 (RS)
1987	28	9	12.9, 17.7, 17.7	9.7	NE	9.40 ...
1988	13	10	24.1, 30.6, 30.6	15.3	33	13.55 (WS)
1989	12	6	8.0, 9.7, 12.9	8.0	NE	8.78 ...
1990	15	11	11.3, 11.2, 12.9	8.4	NE	9.83 ...
1991	22	12	23.7, 26.7, 27.8	16.6	12	10.00 (WS,CR)

[x] Wind data were obtained from a weather station adjacent to experimental plots at the University of California, Davis.
[y] Determined by collecting three to six 100-fruit samples from the experimental orchards. NE = negligible (1–3%).
[z] Results recorded by commercial inspectors including all defects. Data were obtained from Sunsweet Growers, Inc., Yuba City, California, and represent the yearly average off-graded fruit from all prunes processed and evaluated in that plant, located about 96 km north from the experimental plots. Predominant defect for the year in parentheses: RS = russet scab, WS = wind scab, CR = side and stylar-end cracks.

Table 2. Incidence and severity of wind scab on French prune before and after dehydration and percent decay initiated from wind scab in three experimental orchards

Year	Orchard	Side of tree[w]	Wind scab (%)		Wind scab index[x]		Decayed fruit[y] (%)
			Before dehydration	After dehydration	Before dehydration	After dehydration	
1984	1	North	21.0 a[z]	21.3 a	0.36 a	0.41 a	ND
		South	7.7 b	7.3 b	0.14 b	0.09 b	ND
1988	1	North	63.8 a	50.1 a	1.87 a	1.42 a	17.5 a
		South	19.2 b	12.3 b	0.35 b	0.27 b	4.3 b
1988	2	North	66.8 a	56.2 a	1.44 a	1.55 a	ND
		South	26.0 b	14.7 b	0.36 b	0.30 b	ND
1991	3	North	30.7 a	22.3 a	0.59 a	0.42 a	6.1 a
		South	5.7 b	1.7 b	0.09 b	0.02 b	1.1 b

[w] On 6–20 September, 100 prunes each were collected from the north and south sides of three to six replicate trees in two rows.
[x] Rated on a scale of 0–4, where 0 = fruit without wind scab and 4 = the most severe wind scab.
[y] Only decay initiated from wind scab is presented; *Phomopsis cinerascens* was isolated from 84–96% of decayed fruit. ND = not determined.
[z] Numbers in each column for each orchard in each year followed by a different letter are significantly different according to a pairwise *t* test ($P = 0.05$).

Table 3. Fungi isolated from fruit with decay lesions associated with wind scab and stylar-end and side cracks in a French prune orchard

Plant source	Sampling date	Fungi isolated	Incidence[y] (%)
Overripe fruit, decay in stylar-end or side cracks	13 November 1986	*Phomopsis* spp.	75 a
		Yeasts	11 b
		Alternaria alternata	7 b
		Aspergillus niger	2 b
Ripe fruit, decay in wind-scabbed areas	16 September 1988	*Phomopsis* spp.	55 a
		Cladosporium spp.	32 b
		Rhizopus stolonifer	13 c
Ripe fruit, decay in wind-scabbed areas	19 September 1988	*Phomopsis* spp.	78 a
		Rhizopus stolonifer	11 b
		Aspergillus melleus	17 b[z]
Overripe fruit, decay in wind-scabbed areas	29 September 1988	*Phomopsis* spp.	90 a
		Cladosporium spp.	10 b

[y] Average of two collections of three replicates each of 10-fruit samples. For each date of sampling, numbers followed by different letters are significantly different according to LSD test ($P < 0.05$).
[z] Several fruit had both *Phomopsis* spp. and *A. melleus*.

cinerascens was 41%. After artificial inoculation, 81% of the wind-scabbed fruit were infected by *P. cinerascens* but only 42% (ripe) to 69% (overripe) of the inoculated unwounded healthy fruit were infected. When ripe and overripe healthy fruit were wounded and inoculated with *P. cinerascens*, 98–100% decayed, whereas all wounded uninoculated control fruit remained healthy. For unwounded inoculated fruit, significantly more ($P < 0.01$) overripe than ripe fruit were decayed from *P. cinerascens*.

Scanning electron microscopy indicated that germ tubes of conidia of *P. cinerascens* formed appressoria on the surface of healthy ripe fruit. Some germ tubes of *P. cinerascens* spores appeared to enter through the exposed, fractured epidermal and subepidermal layers of wind-scabbed fruit.

Pathogenicity tests with other fungi commonly associated with WS. Although infection levels were always high for artificially wounded fruit, infection levels depended on ripeness for unwounded fruit (Table 4). With the exception of inoculations with *Fusarium* sp. (89% incidence), 98–100% of artificially wounded and inoculated fruit became infected. Mature (but not ripe) unwounded fruit did not become infected after inoculation with all four different fungi tested (Table 4). However, 29 and 19% of ripe unwounded fruit inoculated with *P. cinerascens* or *M. fructicola*, respectively, became infected. In addition, for overripe unwounded fruit inoculated with *P. cinerascens* or *M. fructicola*, 89 and 81% became infected, respectively (Table 4). In most cases, the mean lesion expansion rate was significantly greater on wounded than on unwounded fruit (Table 4).

DISCUSSION

Wind early in the season has been reported to cause severe scarring on Friar plums (5). Nevertheless, to the best of our knowledge, this is the first report of prune fruit being severely affected by WS and of this type of scarring predisposing the fruit to increased infection by decay fungi. Affected prune fruit with wind-scabbed areas 10 mm long or longer were unmarketable as fresh or dehydrated fruit and could be utilized for prune juice. Fruit with wind-scabbed areas of any size were unmarketable in the fresh market.

Wind scab may be confused with RS in prunes because both disorders affect the normal development of the fruit cuticle and epidermal layers. However, while RS usually begins as shiny areas lacking epicuticular wax and surrounding the lower part of the fruit (8), WS almost invariably occurs on the side of fruit as longitudinal, scabby areas, representing areas of friction between affected fruit and other surfaces. Another indication that a fruit is damaged by WS is the development of gum on the affected areas (*unpublished*).

The results presented in Table 2 support the hypothesis that wind was the cause of WS in 1984, 1988, and 1991. Because strong winds prevailed in Davis, California, in the spring (Table 1), fruit on the north side of the trees were subjected to the force of prevailing north winds while fruit on the south side, protected by the tree canopy, received less force and developed less WS. More WS developed in years with 10–12 days of north or northwest winds, at least three of which reached speeds of >20.9 km/hr during the 3-wk period after full bloom (Table 1). Similarly, young citrus fruit are easily damaged by winds during the first 3 wk after petal fall, with little scarring occurring after the fruit are 12 wk old (14). Although the frequency of north or northwest winds within 3 wk after full bloom in prune orchards was high in 1985 and 1990, the speeds of the strongest winds and the average wind speed during the same time period apparently were not high enough to cause WS (Table 1).

Wind scab predisposes fruit to decay in three ways. First, because of their rough surfaces, wind-scabbed areas apparently act as traps for collecting propagules of fungal pathogens. Second, wind-scabbed areas provide avenues for fungal infection. Thin sections of these areas indicated discontinuities of the cutinized cells, which could be invaded by fungi. In fact, all six fungi commonly causing decay in prune fruit (9), some of which have been associated with WS, are known to require wounds on the prune skin to invade mature fruit. Third, fungal infection through wind-scabbed areas could be increased because such areas retain water from rain or dew, whereas water runs off more readily from the hydrophobic, waxy surfaces of fruit without WS. Such moisture could enhance the risks of spore germination, germ tube elongation by decay fungi, and yeast multiplication. Although the symptoms of decay caused by *Phomopsis* spp. or *A. melleus* are obvious on fresh prunes, it is unknown whether such decay affects the appearance of dehydrated prunes. However, decay of prunes before dehydration, particularly from *Rhizopus* spp. or *Aspergillus japonicus* Saito, can result in a slip-skin maceration disorder ("box rot") of dehydrated fruit (12). In addition, several molds and sporogenous and asporogenous yeasts cause spoilage of dehydrated prunes (13).

P. cinerascens and two other strains of a *Phomopsis* sp. were consistently isolated from decaying fruit in the field, suggesting that they could be major causes of decay of French prune that are harvested late. Because rains occurred in November 1986, it is possible that fruit on the tree became infected when spores splashed from pycnidia on small leaf lesions or twig cankers. *P. cinerascens* was consistently isolated from small necrotic lesions on leaves and cankers on 1- and 2-yr-old twigs of prunes (*unpublished*). Once spores land on the surface of fruit with WS, they can germinate and penetrate through the fractures and discontinuities in the epidermal layers. In contrast, spores landing on healthy areas may require the formation of appressoria to invade

Table 4. Incidence and severity of infection on wounded and unwounded healthy French prune fruit inoculated with various fungi commonly associated with wind scab and incubated at 23 ± 1 C for 3–8 days

Pathogen	Condition of fruit[x]	Wounded (+) or unwounded (−)[y]	Fruit infected (%)	Mean decay lesion expansion rate (mm/day)
Phomopsis cinerascens	Mature	+	100*[z]	4.3
		−	0	...
	Ripe	+	98*	2.5
		−	29	2.2
	Overripe	+	100*	2.3*
		−	89	1.7
Monilinia fructicola	Ripe	+	100*	9.3*
		−	19	6.0
	Overripe	+	100*	3.2*
		−	81	2.5
Penicillium expansum	Overripe	+	100*	2.2*
		−	76	1.6
Aspergillus melleus	Mature	+	100*	2.3
		−	0	...
Cladosporium herbarum	Mature	+	100*	1.2
		−	0	...
Fusarium sp.	Mature	+	89*	1.1
		−	0	...

[x]Fruit were harvested on 1 September (mature), 16 September (ripe), and 2 October 1991 (overripe) from an experimental orchard of the Department of Pomology at the University of California, Davis.

[y]Fruit were inoculated with 30–40 µl of a 2×10^4 spores per milliliter suspension of *M. fructicola* or a $1–2 \times 10^5$ spores per milliliter suspension of the other fungi.

[z]* = Significant difference between wounded and unwounded fruit according to a pairwise *t* test ($P = 0.05$).

successfully. *Phomopsis mali* can infect intact apples through the epidermis via penetration hyphae (10), and *Phomopsis* spp. can infect unwounded kiwifruit (7). *P. cinerascens* infected unwounded prune fruit only when they were ripe or overripe, but infection might occur earlier if there is WS (Table 4).

Although protection of prunes with special pruning or with windbreaks is not at present justified because of the cost, in certain years WS can cause significant damage either by scarring the fruit or by making it more prone to fungal infection. In 1988, 13.5% of prunes at a major processing plant (5.5% over the commercial limit) were rated as off-grades primarily because of wind scabbing. The severity of damage on prunes caused by WS in some years almost equaled that caused by RS in others. Effects of WS on yields of prune have not yet been quantified, but winds are known to significantly decrease yields of Friar plums (5).

Many of the fungi that cause decay on prunes are soilborne (6,9). Fungi brought to the fruit by soil dust can increase decay. Therefore, creation of dust by mowing weeds in the orchard during maturation of fruit and/or before harvest may result in additional decay and should be avoided, particularly if there is much WS.

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