equivalent maturity. Fruit from infected trees are slightly better in color than normal fruit (7), and commercial picking is based on color. Previous pickings may have left only the small, less colored, firm fruit on infected trees. Variations in fruit firmness between phony and normal trees were compensated for by regressing fruit weight against firmness.

The regression equations showed that fruit weight for phony-infected trees increased significantly less than normal as firmness decreased (Fig. 1). Fruit from normal trees in Orchard 1 increased in weight an average of 2.90 g per newton decrease in firmness, whereas fruit from phony-infected trees increased only 1.03 g (Fig. 1). The corresponding values for phony and normal trees in Orchard 2 were increases of 1.69 g and 0.95 g, respectively, per newton decrease in firmness (Fig. 1). The low R^2 values for the regression equations reflected, in part, the wide variation in fruit weight that occurred within each tree at harvest (Fig. 1). Higher order regression coefficients were not significant (data not shown).

The differences in weight were greatest for the softest fruit at harvest. The differences probably developed as fruit firmness decreased during final swell. The regression lines for fruit from phony-infected trees and normal trees in Orchard 1 intersected at a fruit firmness of 56.5 N and a weight of 52.1 g. Only mature fruit were harvested in Orchard 1, and these regression lines may not apply to very firm, immature fruit harvested before final swell. The regression lines for Orchard 2, where fruit with a wide range of firmness were harvested, intersected at a firmness of 90.3 N and a weight of 30.3 g, which was outside the range of fruit firmness and weight sampled. A flesh firmness of more than 80 N generally indicates the fruit will not mature properly after harvest (12).

Several theories have been advanced to explain the symptoms of phony disease. These theories involve toxins, growth regulator imbalances, and internal moisture stress caused by xylem blockage. Growth regulator imbalances could be a direct or an indirect cause of the symptoms, because xylem blockage also could interfere with the movement of growth regulators and nutrients from roots to shoots.

Internal moisture stress caused by xylem blockage seems to explain most symptoms of phony disease. These symptoms include limited increases in fruit weight during final swell, drier leaves (4), incubation times of 18 months or more before disease symptoms develop (7), reduced shoot growth developing over the entire tree (7), a complete absence of leaf scald or twig-dieback (8, 13), differences in leaf elemental concentrations after symptoms develop (3), and blocked root xylem (2). Additional research is needed to confirm this theory and to determine where blockage occurs.

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Cold Resistance in Peach, Apricot, and Cherry as Influenced by Soil-applied Paclobutrazol

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Additional index words. *Prunus persica, Prunus armeniaca, Prunus avium*, wood, flower buds

Abstract. **Low temperature injury to flower buds of peach [*Prunus persica* (L.) Batsch.] and sweet cherry [*Prunus avium* L.] and to one-year-old shoots of peach and apricot (*Prunus armeniaca* L.) during fall and winter was more severe on trees treated with paclobutrazol (PP 333) than on those not treated. Paclobutrazol had no measurable effect on cold resistance of apricot buds or cherry shoots. The average date of 1st bloom was advanced by one to 2 days in all 3 species by paclobutrazol.**

Paclobutrazol, believed to be an inhibitor of gibberellin biosynthesis (4), is a plant growth inhibitor, reported to increase cold resistance of sweet cherry flower buds (5). The 1st requirement for low temperature acclimation in woody plants is that they must not be growing actively (6). Gibberellin applications can reduce vegetative hardiness in *Prunus* (2, 3). Therefore, the hypothesis that paclobutrazol hastens cold acclimation in woody plants by inhibiting gibberellin biosynthesis and terminal growth seems reasonable.

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Paclobutrazol (50% wettable powder) was applied to the soil under 20-year-old 'Bing' and 'Chinook' sweet cherries (6 replicates) at 15 g per tree on 26 Aug. 1982, and at 2½ and 7½ g a.i. per tree to 12-year-old 'Goldrich', 'Rival', and 'Wenatchee' apricots (3 replicates), 'Roza' and 'Redhaven' peaches (8 replicates), and 'Independence' nectarines (8 replicates) on 20 Oct. 1982. All 3 experiments were split plot designs with cultivar as the main plot. All treatments were applied in the 2 irrigation furrows nearest the tree (25 to 100 cm from the trunk) then incorporated into the soil. Trunk diameters of the cherry, apricot, and peach trees were about 35, 20, and 17 cm, respectively.

The cold resistance of one-year-old apricot shoots was evaluated in Oct. 1983 and Jan. 1984, by measuring the electrical conductivity of a water extract of the tissues following freezing (1). Peach and cherry were evaluated in Jan. 1984. The shoots were cut into 2.5 cm segments and divided into 40 g samples before freezing. The temperature was

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Table 1. The effect of field applications of paclobutrazol on cold injury to flower buds of apricot and peach by −24°C on 23 Dec. 1983.

Species	Temp. (°C)	Cultivar	Paclobutrazol (g a.i./tree) 0 (% killed)	2½ (% killed)	7½ (% killed)	LSD 5%[z]
Prunus armeniaca	−24	Goldrich	12	10	13	NS
		Rival	14	14	8	NS
		Wenatchee	42	50	49	NS
		Mean	23	25	23	NS
Prunus persica	−24	Roza	40	43	48	NS
		Redhaven	21	32	31	9
		Independence	35	52	53	9
		Mean	32	42	44	5

[z]NS = nonsignificant.

Table 2. The effect of field application of paclobutrazol at 15 g a.i./tree on cold injury to flower buds of sweet cherry (*Prunus avium*) in laboratory tests on 2 dates.

Date	Temp. (°C)	Cultivar	% killed Check	Treated	LSD 5%[z]
15 Dec. 1983	−22	Bing	67	87	9
17 Jan. 1984	−25	Bing	55	80	NS
		Chinook	20	40	NS
		Mean	38	60	21

[z]NS = nonsignificant.

Table 3. The effect of field applications of paclobutrazol on cold injury to one-year-old shoots of peach and apricot in laboratory evaluations.

Species	Temp. (°C)	Cultivars	Paclobutrazol (g a.i./tree) 0	2½	7½	LSD 5%[z]
			micromhos			
Prunus armeniaca	−26	Goldrich	129	160	159	NS
	−28	Rival	104	127	125	NS
	−27	Wenatchee	152	239	263	41
		Mean	128	175	182	24
Prunus persica	−25	Redhaven	108	117	126	12

[z]NS = nonsignificant.

Table 4. The effect of field application of paclobutrazol on the average date of first bloom in apricots, peaches, and cherries in 1984.

Species	Cultivars	Paclobutrazol (g a.i./tree) 0	2½	7½	15	LSD 5%[z]
		Date of bloom				
Prunus armeniaca	Goldrich	23 Mar.	22 Mar.	22 Mar.	---	1.0
	Rival	23 Mar.	22 Mar.	21 Mar.	---	1.0
	Wenatchee	26 Mar.	24 Mar.	24 Mar.	---	1.0
Prunus persica	Roza	7 Apr.	6 Apr.	5 Apr.	---	1.0
	Red haven	7 Apr.	6 Apr.	6 Apr.	---	1.0
	Independence	5 Apr.	5 Apr.	5 Apr.	---	NS
Prunus avium	Bing	2 Apr.	---	31 Mar.	31 Mar.	0.9
	Chinook	1 Apr.	---	30 Mar.	30 Mar.	0.9

[z]NS = nonsignificant.

lowered at 2°C/hr to predetermined injurious levels, thawed for 1 hr, then extracted 24 hr in 200 ml water.

The cold resistance of about 100 peach and cherry flower buds per sample was determined in Dec. 1983 and Jan. 1984. The temperature was lowered at 1°C/hr to injurious levels. Lethal injury was determined as the percentage of the buds with brown floral parts.

On 23 Dec. 1983, the temperature in the orchard reached −24°C. This was low enough to injure some peach and apricot flower buds, but not cherry buds. About 100 buds from each tree in the apricot and peach experiments were examined.

During late March and early April of 1984 the date on which the 1st blossoms opened was determined for each tree, and an average date of 1st bloom was calculated.

Cherry trees treated with paclobutrazol on 26 Aug. 1982 bloomed 4 days earlier than untreated trees in 1983. Apricot and peach trees, treated 20 Oct. 1982, bloomed on the same date regardless of paclobutrazol treatment.

By mid-May of 1983, growth of the cherry trees treated in August of 1982 clearly was retarded. Most trees had strong active shoots, but there were fewer of them on treated trees as compared to those not treated. By early August, the growth of nontreated trees was rated 5 on a 1 to 5 scale. The treated trees were rated 2.5.

Reduced terminal growth of treated peach and apricot trees was apparent by mid to late June. When the 2nd flush of growth began in apricots in late July, none of the treated trees grew. Defoliation was advanced by a week or more in apricots, but not in peach or cherry.

On 10 and 25 Oct. 1983, one-year-old shoots of treated and nontreated apricot trees were evaluated for cold resistance. The nontreated shoots were still growing. The treated shoots had not grown since July and were defoliating earlier than nontreated shoots. The temperature required to induce a conductivity value of 200 micromhos was increased by 4°C on 10 Oct. and 2° on 25 Oct. where the trees had been treated with paclobutrazol, indicating that paclobutrazol had reduced cold resistance. This surprising observation prompted further measurement of the effect of paclobutrazol on cold resistance in *Prunus*.

Paclobutrazol did not affect the severity of injury to apricot flower buds in the December freeze but significantly increased injury to peach and nectarine flower buds (Table 1). A subsequent laboratory test on 'Redhaven' peach tended to confirm the field observation, although the differences were not significant at the 5% level. Sweet cherries were not injured by the December freeze because the temperature was not low enough, but, in laboratory tests in mid-January, paclobutrazol increased the amount of low temperature injury (Table 2).

Paclobutrazol increased the severity of low temperature injury to one-year-old shoots of peach and apricot in January laboratory tests (Table 3). There was no difference in injury to cherries.

First bloom in 1984 was one or 2 days earlier on trees treated with PP333 than on nontreated trees (Table 4). Resistance of flower buds to spring frost decreases as they develop to anthesis and beyond. The plots were not exposed to damaging temperatures in the field during anthesis, and laboratory tests during that period did not show significant differences in low temperature injury between treated and nontreated samples.

These preliminary data are sufficient to show that we must not assume increased cold resistance simply because terminal growth can be inhibited. Apricot shoots from paclobutrazol-treated trees had ended terminal growth in July and defoliated a few days earlier than nontreated trees, which still had active meristems in October. Nevertheless, the treated trees were less cold resistant during dormancy rather than more resistant as their growth status might have led one to believe.

Paclobutrazol has been reported elsewhere to increase cold resistance in sweet cherry

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flower buds (5) in contrast with reduced resistance observed in this experiment. Therefore, there is an interaction of the paclobutrazol effect on cold resistance with other factors related to location or weather. Almost certainly there will be an interaction with species and maybe cultivar. Nevertheless, the risk of reduced cold resistance resulting from the use of paclobutrazol in the managment of peach, apricot, and sweet cherry must be considered in evaluating the potential benefits.

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Wholesale, Retail, and Consumer Level Losses of Nectarines in Metropolitan New York

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Additional index words. parasitic diseases, disorders, injuries

Abstract. **Quantitative and qualitative losses incurred in the marketing of nectarines (*Prunus persica* L. 'Nectarina' Ait) in metropolitan New York were studied at wholesale, retail, and consumer levels from 1981 to 1983. Examination of nectarines at the wholesale level indicated that losses to pathological, physiological, and physical deterioration were 3.3%. Losses of 6.6% were observed at retail store and consumer levels. Decay, mainly due to brown rot, was the leading cause of loss in wholesale and consumer level sampling, whereas mechanical damage caused most of the loss in retail.**

The volume of fresh nectarines delivered to metropolitan New York has been increasing steadily in recent years, and it presently averages about 11,000 MT annually (10). Aside from a small volume of winter imports from Chile (2–3%), California supplies practically all of the nectarines to this market. A study to determine the nature and extent of losses in wholesaling, retailing, and consumption of nectarines could provide guidelines for developing commercially feasible measures to reduce these losses. This report is one in a series of studies on marketing losses of fresh produce crops that began in 1966 (1, 2, 3, 4, 5, 6, 7, 12).

The evaluation of the wholesale condition of nectarines was conducted during the period from 1981 to 1983, beginning in June and ending in September of each year. Cartons containing 11.35 kg of fruit were obtained weekly from food-chain distribution centers and from wholesalers in New York City's Terminal Market at Hunts Point. The samples were brought to the Postharvest Research Center in New Brunswick, N. J., where they were examined. In 1983, the nectarines also were examined after they had ripened at 21°C for 3 days.

The criteria used for sorting nectarines at wholesale reflected the normal culling practice of retailers. The culls were placed into categories which included parasitic diseases, injuries from mechanical handling, and nonparasitic disorders such as internal breakdown, desiccation, overripeness and freezing.

Retail loss data for a 1- or 2-day sales period were obtained from 8 or 9 metropolitan New York supermarkets for each week California nectarines were in season. At least 2 stores each in representative low, middle, and high income locations were visited 1–3 days after the wholesale examinations. The loss data were derived from the number of culls removed from cartons of fruit sold or displayed in each store, and once weekly a consumer-size store sample (10–15 fruits) also was purchased. When the nature of the retail loss could not be determined immediately, the affected specimens were brought to the laboratory for a thorough examination. The consumer samples also were held in the laboratory at 21°C until the nectarines were eating ripe, usually in 1–3 days. Losses recorded for retail and consumer levels were based on the weight of the fruit. Retail losses consisted of whole fruits, whereas trimmings made up the bulk of losses in consumer samples.

The culls in wholesale cartons examined ranged from 3.2% to 3.3% per year (Table 1). Parasitic diseases accounted for approximately two-thirds of the culls. Brown rot [*Monilinia fructicola* (Wint.) Honey and *M. laxa* (Aderh. & Ruhl) Honey] was the most prevalent fruit rot observed, affecting about 60% of the diseased culls. An unidentified yeast rot that was usually localized at the distal end of the nectarine was the next most common rot. It was associated with a softening and advanced ripening of the tissue at that end. Other rots in wholesale samples were gray mold rot (*Botrytis cinerea* Pers. ex Fr.), cladosporium rot (*Cladosporium herbarum* Lk. ex Fr.), alternaria rot (*Alternaria* sp.), and sour rot (*Geotrichum candidum* Lk. ex Pers.).

Physical injuries accounted for approximately 25% of the fruit losses in wholesale samples. Mechanical damage, manifested as severe bruises, prominent cuts, and punctures, was chiefly responsible for the loss. Nonparasitic disorders affected only a few fruit that were culled because of excessive softness or a badly split pit. Another 2.4% of the fruit had defects such as split pits, cuts, punctures, and bruises that were not serious enough to warrant a cull designation and for which data are not presented.

When the 1983 wholesale samples were ripened by holding at 21°C for 3 days, a sharp increase in cullage occurred. Brown rot (4.8%), gray mold rot (0.5%), yeast rots (0.7%), and sour rot (0.1%) were responsible for spoilage of an additional 6.1% of the fruits, and another 0.3% was overly soft or overripe. While some rots, especially those caused by yeasts, developed in bruised tissue, brown and gray mold rots apparently developed from incipient and/or latent infections. These infections became active when the fruits were removed from cold storage or when they ripened. Ripening produces physiological changes in host tissue and cell walls that could activate latent infections (11).

Annual retail store losses ranged from 6.1% to 7.1% and averaged 6.6% of the 156,850 nectarines (17,100 kg) retailed during the 3-year study (Table 1). Mechanical injury was the leading cause of loss in retail stores and, with freezing and insect damage, wasted 4.2%. Parasitic diseases were responsible for spoilage of 1.9%. Shrivelling, internal breakdown and overripeness caused the remaining 0.5%. No significant differences in wholesale or retail losses were found between years of sampling, nor was there any significant yearly difference in parasitic disease loss.

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