Response of Young, Fruiting Sour Cherry Trees to One-time Trunk Injury at Harvest Date

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Abstract. The influence of increasing levels of trunk damage on vegetative and reproductive capacity of 3- to 5-year-old 'Montmorency' sour cherry (Prunus cerasus L.) trees was determined for three seasons. Removal of or damage to bark up to halfway around the trunk circumference minimally affected growth and productivity. The total wound callus produced per tree was related to wound size. Wound repair was variable depending on the type or extent of injury. Removal of damaged bark greatly reduced wound repair. Girdling 75% or 100% of the trunk circumference resulted in no tree mortality at one site and 17% and 50% mortality, respectively, at another. Differentiated phloem in wound callus of trees with 100% bark removal and survival 4 years following injury indicated that vascular reconnection occurred across wounds.

Trunk injury to young, fruiting sour cherry trees from mechanical harvest, careless orchard implement use, freezing temperatures, animal feeding, insect boring, and disease invasion can reduce tree vigor, productivity, and orchard life. Brown et al. (1984) indicated that trunk or bark injury resulting from mechanical harvest is an important factor contributing to the rapid decline of sour cherry orchards. Damage that results from tree shaking can be attributed to operator or mechanical error, high cambial activity (low bark strength) at time of harvest (Fridley et al., 1970), or use of excessive force to remove immature fruit (Cargill et al., 1982). Bark may be ruptured without disturbing the periderm and go unnoticed (Diener et al., 1968). In either case, vascular sap flow may be reduced. Trees with damaged bark are more susceptible to pathogens, since wounds provide a favorable environment for pathogen development (Cargill et al., 1982; Fridley et al., 1970).

Repair of wounds depends on wound size and severity, tree health and vigor, and the time of year at which injury occurs. Wounds incurred in the spring will heal more rapidly than wounds occurring in the late summer or fall (Crowdy, 1953; McQuilkin, 1950; Neeley, 1970; Wensley, 1966). Wounds heal most rapidly on vigorous trees (Chadwick and Nank, 1949; McQuilkin, 1950; Wensley, 1966).

Callus production in the wound zone is necessary for wound repair. This callus proliferation originates in the vascular rays in close proximity to the cut surface (Noel, 1968; Sharples and Gunner, 1933; See, 1959). In some cases, vascular continuity can be reestablished as a result of vascular differentiation within the new callus tissue (Noel, 1968).

Since young sour cherry tree trunks are often damaged, the purpose of this study was to: 1) evaluate the responses of sour cherry trees to one-time trunk damage at commercial harvest and 2) determine threshold levels for one-time trunk injury that would be detrimental to vegetative and reproductive capacity and result in tree mortality of young, fruiting sour cherry trees.

Materials and Methods

All trunk damage experiments were conducted on young, fruiting sour cherry trees ('Montmorency' on Mahaleb rootstock). Two sites were used: 1) the Horticultural Research Center, Michigan State Univ., East Lansing (HRC). Trees were planted in 1983 in a single east–west-oriented row at a 1-m spacing in Miami loam soil (pH = 5.5-6.0) and were not pruned or supplementedly irrigated. 2) Clarksville Horticultural Experiment Station, Clarksville, Mich. (CHES). Trees were planted in 1982 in north–south-oriented rows at spacings of 3.0 × 6.0 m and 3.0 × 4.5 m on a Bixby sandy loam soil (pH = 5.9-6.1). Trees at this site were trickle-irrigated, and pruning, fertilization, and other cultural practices were performed according to local standards. These trees were more than twice the size [based on trunk cross-sectional area (TCA) and tree height] and had a much higher fruit density than those at HRC in the season of injury. The trees at HRC were smaller than expected in a commercial orchard at the same age, while those at CHES were more typical of young, fruiting, commercial sour cherry trees in Michigan. Pesticides were applied according to commercial recommendations (Hewitt et al., 1987). All trunk injury treatments at both sites in 1986 and 1987 were imposed in the 2nd week of July, which is during the period of commercial harvest in Michigan.

Two preliminary experiments were conducted in 1986: 1) a bark removal experiment at HRC; and 2) a bark compression experiment at CHES. At HRC, 50 trees were selected based on uniformity. Trunk injury was imposed by removing a 5-cm-wide strip of bark encircling 0, 25%, 50%, 75%, or 100% of the trunk circumference (percent bark removal). There were 10 replicate trees used per treatment. Bark was cut to the xylem (between 15 and 20 cm above the graft union) with a sharp grafting knife and immediately peeled off. The exposed underlying tissue was left undisturbed. At CHES, mechanical harvest injury was simulated by bark compression using an instrument developed by the Agricultural Engineering Dept., Michigan State Univ. Clamping pressure and shear stress were imposed on a 14.3-mm-diameter circular section of bark (Brown et al., 1984) at a constant pressure of 0.38 MPa. Twenty-eight trees at the 3.0 × 6.0-m spacing were selected based on uniformity, and wound zones 0, 10%, 20%, or 40% around the trunk circumference were marked on the trunk. There were seven replicate trees used per treatment. Bark compressions were made using this instrument until the entire wound zone was filled in (bark was left intact). All trunk injury treatments in 1986 and 1987 would be detrimental to vegetative and reproductive capacity.
at CHES were imposed between 30 and 35 cm above the graft union, which is a normal position for mechanical harvester clamp attachment.

In 1987, the bark removal experiment (as described above at HRC in 1986) was repeated at CHES using 30 uniform trees at the 3.0 × 4.5-m spacing with six replicate trees per treatment. Eighteen additional trees at this spacing were selected for another experiment in which 50% of the bark was damaged as follows: 1) bark was removed using a grafting knife; 2) bark was compressed as described above and left intact; and 3) bark was compressed and then immediately removed using a grafting knife. There were six replicate trees per treatment.

Statistical calculations. Unless otherwise indicated, a randomized complete-block design was used for all studies and trees were blocked by TCA (Westwood and Roberts, 1970) before treatment imposition. Data were subjected to regression analysis or analysis of variance, as appropriate.

Nonstructural carbohydrates. Bark tissue was collected on 17 Nov. 1987 from trees at HRC that had 100% or 0% (control) of the strip removed. Five 16-mm-diameter disks were collected above and below the wound on trees that had 100% of the strip removed and five at the approximate wound height on the control trees. Five replicate trees representing each of the TCA size categories were sampled for each treatment. The protocol of Gucci (1988) was modified for bark tissue. Samples were freeze-dried and ground in a Wiley mill to pass through a 40-mesh screen. Four 100-mg subsamples of each sample were extracted four times each for 20 min with 2 ml of 80% ethanol. The homogenates were centrifuged at 1500×g for 5 min after each extraction.

For determination of soluble sugars (sorbitol, fructose, glucose, inositol, and sucrose), the supernatants were transferred into 100-ml round-bottom flasks and evaporated to dryness using a rotary vacuum evaporator in a water bath at 40°C. The samples were converted into oximes (Roper et al., 1988) and derivatized to tri-methylsilyl ethers (Sweeney et al., 1963). Analyses were performed using a dual-column, temperature-programmed Varian 3700 gas chromatography (Varian Associates, Sunnyvale, Calif.) with a flame ionization detector and 3% OV-17 on 80/100-mesh Chromosorb WHP in a 2 mm × 2-m glass column. Temperature was programmed from 150 to 250°C at 5°C/min. Quantity was calculated by peak area with a Spectra Physics SP4100 integrator (Spectra Physics, San Jose, Calif.) using internal standards. Two separate 1-μl injections were made for each sample and results were averaged.

Starch in the pellet was measured using the method of Roper et al. (1988) modified as follows: Samples incubated at 55°C for 16 h with amyloglucosidase were assayed calorimetrically using glucose oxidase (Sigma Chemical Co., 1988). Absorbance at 440 nm was read with a Shimadzu UV-Vis 260 spectrophotometer (Shimadzu Corp., Kyoto, Japan).

Cold hardness. Deep winter hardness was evaluated for current-season shoots collected on 8 Feb. 1987 from trees at HRC according to methods of Bittenbender and Howell (1974). Four shoots were randomly selected from each of 20 trees representing all five treatments. Shoots were cut into 5-cm-long sections and then subjected to a controlled temperature reduction in a freezing chamber. Samples were exposed to temperatures ranging from –20 to –40°C and then visually evaluated for xylem browning. T₅₀ values, i.e., the temperature (ºC) required to kill half of the samples, were calculated for each treatment.

Anatomical examination. Five trees at HRC were sampled where callus tissue had completely filled in the wound zone during the season of injury. On 24 Mar. 1987, bark strips 7.0 cm long and 0.5 cm wide beginning just above the wound, then passing completely through the callus, and finally into the bark below were excised and fixed in formalin : acetic acid (FAA). Tissue was imbedded in paraffin, serial-sectioned at 13 μm, and stained with safranin and fast green. Sections were examined and photographed with a Zeiss (Oberkochen, Germany) Photomicroscope II at × 250 magnification.

Reproductive and vegetative growth measurements. Wound repair was rated in Fall 1986 through 1989 when terminal growth had ceased. The amount of callus tissue present was visually rated as a percentage of the total wound surface.

TCA was calculated from the average of two perpendicular trunk diameters immediately above and below the wound, measured with a vernier caliper for all trees wounded in 1986 at HRC and CHES at the time of injury (11 July 1986) and in the late fall (15 Nov. 1986). Change in TCA, or trunk growth, was then calculated.

Current-season shoot growth was measured for eight and 10 terminal shoots per tree after terminal bud set for trees at HRC and CHES, respectively. Shoots were selected at a height of 2 m on the east side of trees.

Fruit density in the season following injury was determined as the number of fruit per square centimeter of limb cross-sectional area at the base of two branches on opposite sides of the tree for all experimental trees at HRC and CHES on 2 June 1987. On 6 July 1987, fresh weights and percent soluble solids concentration (SSC) were determined for 25 fruits per tree for the 0% and 100% bark removal treatments at HRC. Fruit from all trees wounded in 1987 at CHES were harvested in 1988 and 1989, and yield was expressed as fruit weight (in kilograms) per tree.

Results

Bark removal experiment (HRC, 1986). In the season of injury, no relationship was observed between TCA or bark removal level and percentage of the total wound zone filled in with callus tissue (r² = 0.16 and 0.06, respectively). However, a linear relationship was observed between the amount of bark removed and the absolute amount of wound callus produced by the tree (r² = 0.61). On average, trees filled in 58% of the wound zone with callus tissue. Shoot growth during the season of injury was not reduced by bark removal (mean value = 24 cm). Progression of healing for one tree with 100% bark removal is illustrated in Fig. 1. Trees with bark removed completely around the trunk had swollen bark above the wound, and this bark tissue accumulated more starch and sorbitol than bark from a similar position on a noninjured tree (Table 1). Fructose, glucose, inositol, and sucrose content of these tissues was not affected (mean values were 1.09, 1.25, 0.016, and 2.65 mg/100 mg tissue dry weight, respectively).

Deep winter cold hardness at HRC was reduced from –29.0 to -25.6°C (significant at P = 0.05) in shoots from trees with 100% bark removal, but fruit density (0.75 vs. 0.93/cm²) and fresh weight (3.37 vs. 3.61 g) were not affected by bark removal. Fruit SSC at harvest was reduced, but not significantly (16.0% vs. 14.7%), in trees with 100% bark removal. Phloem sieve tube cells were found in callus tissue that closely resembled those in bark tissue (Fig. 2). Four years after treatment, no tree death resulted from any of these degrees of bark removal.

Bark compression experiment (CHES, 1986). No significant bark
Swelling was observed above the wound in trees at the end of the season of injury. Fruit density the season following injury was not affected by the bark damage treatments (mean value = 0.9 fruits/cm\(^2\)). Periderm was peeled back from wounds at the end of Summer 1987 and all wounds had been completely repaired. Photographs of one tree with 40% compression, taken at different dates following injury, are presented in Fig. 3 b-d.

### Table 1. The effect of 100% bark removal (July 1986) on trunk growth and bark carbohydrate content of 4-year-old 'Montmorency'/Mahaleb sour cherry trees at HRC.*

| Bark removal (%) | Trunk growth July–Nov. 1986 (mm\(^2\)) | Sorbitol Nov. 1987 (mg-100 mg\(^{-1}\)) | Starch Nov. 1987 (mg-100 mg\(^{-1}\)) |
|------------------|----------------------------------------|----------------------------------------|--------------------------------------|
| 0, Control       | 462 b                                  | 0.74 b                                 | 1.03 ab                              |
| 100, Above wound | 761 a                                  | 1.18 a                                 | 1.21 a                               |
| 100, Below wound | 383 b                                  | 0.84 b                                 | 0.77 b                               |

*Mean separation in columns by Duncan's multiple range test, \(P = 0.05\).

Annual shoot growth the season following injury (1988) was reduced at all levels of bark removal (Table 2). In 1988, fruit yield was highest in trees with 75% bark removal and lowest in trees with 100% bark removal. Differences in shoot growth and fruit yield between treated and control trees were negligible by 1989. In 1988, two of six trees with 100% bark removal were dead. By 1989, one tree with 75% and a total of three trees with 100% bark removal were dead. Dead trees were treated as missing plots for all data, except tree mortality, in the analysis.

**Type of injury experiment (CHES, 1987).** Wound repair was significantly affected by the type of bark injury imposed at harvest date. When crushed bark was left intact, more wound repair resulted than when bark was removed or crushed followed by bark removal (Table 3). Essentially no wound callus was produced in trees where crushed bark was removed. One such tree, typical for this experiment, is shown immediately following compression/removal in Fig. 3a. Note that both exposed trunk tissue and peeled-off bark (below) were discolored, indicating injury. Annual shoot growth was highest in 1988 for trees in which crushed bark was removed. By 1989, shoot growth was...
Fig. 3. Photographs of injury from bark compression instrument (A) of one tree at CHES immediately after removal of bark of which 50% of the trunk circumference had been compressed on 7 July 1987 (note discolored exposed wood and peeled-off bark below) and for another tree at CHES (B) immediately following bark compression 40% around the trunk circumference on 22 July 1986; (C) same as (B) on 5 Aug. 1986; (D) same as (B) on 17 Sept. 1987 (note that the periderm and outer bark have fallen off the wound).

Table 2. The effect of bark removal (July 1987) on shoot growth, fruit yield, and tree death of 5-year-old ‘Montmorency’/Mahaleb sour cherry trees at CHES.

| Bark removal (%) | Annual shoot growth (cm) | Fruit yield (kg/tree) | Dead trees (%) |
|------------------|--------------------------|-----------------------|----------------|
|                  | 1988 1989                | 1988 1989             | 1988 1989      |
| 0                | 31.0 17.8                | 9.1 27.6              | 0 0            |
| 25               | 25.3 20.9                | 9.1 31.8              | 0 0            |
| 50               | 26.1 22.7                | 8.7 28.8              | 0 0            |
| 75               | 17.3 17.5                | 13.6 30.3             | 0 17           |
| 100              | 6.6 15.0                 | 5.9 25.1              | 33 50          |

Significance: L 0.29** 0.01 0.04 0.02 0.14* 0.24** 0.31** 0.07** 0.04 0.21 0.25* 0.32**

*Coefficient of determination (r²) and significance at P = 0.05 (*) and 0.01 (**) for the linear (L) and quadratic (Q) terms.

Table 3. The effect of different types of trunk injury (July 1987) on wound repair, shoot growth, and fruit yield of 5-year-old ‘Montmorency’/Mahaleb sour cherry trees at CHES.

| Trunk injury treatment | Wound repair (%) | Annual shoot growth (cm) | Fruit yield (kg/tree) |
|------------------------|------------------|--------------------------|-----------------------|
| 50% bark removal       | 45 b 16.2 b 20.7 b | 5.2 31.3                 |
| 50% of bark compressed | 78 a 14.4 b 22.2 ab | 5.0 28.1                 |
| 50% of bark compressed + removal | 6 c 19.4 a 24.3 a | 4.4 30.7                 |

Discussion

One objective of this study was to determine threshold levels for one-time trunk injury that would be detrimental to vegetative and reproductive capacity and kill young, fruiting sour cherry trees. This objective was not met for trees at HRC, since wounds were largely repaired in the season of injury and vegetative growth and yield were only marginally affected by bark removal. However, at CHES, removal of bark 75% or 100% around the trunk circumference did result in tree death (Table 2), but not in the season of injury.

Although the younger trees at HRC were grown under more limiting conditions, wound repair was much greater in the season of injury than that observed for older trees at CHES. The older, higher-yielding trees at CHES possibly were more susceptible to stress, or tree age or initial size may have interacted with the healing response. None of the trees injured at the 25% to 50% level died, and they were at least as productive as non-injured trees and only slightly less vigorous in the first year after treatment (Table 2). These results suggest that trees at CHES were able to compensate for bark damage up to 50% around the trunk circumference.

The other objective of this study was to evaluate responses of young, fruiting sour cherry trees to one-time trunk damage at commercial harvest. Since removal of nondamaged bark resulted in substantial wound callus production in trees at HRC, we hypothesized that removal of damaged bark might facilitate wound repair. The complete inhibition of wound callus production (Table 3) of trees at CHES implied, in fact, that removal of damaged bark was detrimental to wound repair. The discoloration of the underlying wood and the underside of the bark (Fig. 3a) indicated that cambial/xylem desiccation probably occurred, leading to reduced callus production because of vascular ray death (Noel, 1968; Shariples and Gunerry, 1933; See, 1959).

Although some authors have indicated that wounds incurred in the spring (or early summer) heal more rapidly than wounds occurring in the late summer or fall (Crowdy, 1953; McQuilkin, 1950; Nee-
Because the vascular cambium was active at commercial harvest and since no insect or disease infestation was observed in any wounds at either site, wound repair conditions were probably ideal. Although all of the trees with 100% bark removal at CHES were of similar vigor (indicated by shoot growth and TCA) at the time of injury, half of them died over the next 2 years. The trees that died produced little or no wound callus, whereas those that survived produced a moderate amount. The absence of wound callus prevented vascular tissue regeneration, which may have resulted in root carbohydrate deprivation, as indicated by the significantly reduced foliation and shoot extension the following season (Table 2). At both HRC and CHES, trees in which vascular continuity was reestablished were only marginally affected in terms of growth and productivity.

This study indicates that the amount of wound callus produced is related to wound size in young, fruiting sour cherry trees. Trees at HRC that had bark removed completely around the trunk filled 71% of the wound zone with callus tissue during the season of injury. Remarkably, some trees had completely filled this wound zone by the end of the summer. Trees within each site differed minimally in vigor, as estimated by trunk girth or vegetative shoot growth. Had there been large differences in tree vigor at a given site, a relationship between vigor and wound repair might have been observed, as has been noted by others (Chadwick and Nank, 1949; McQuilkin, 1950; Wensley, 1966). Martin and Sydnor (1987), however, noted poor relationships between wound closure rates and trunk caliper increases or twig extension in 12 tree species.

This study did not consider repeated injury occurring to the same tree as might occur in a mechanically harvested commercial orchard. The threshold for one-time trunk injury at harvest in young, fruiting sour cherry trees is quite high. Bark damage encircling up to 50% of the trunk circumference was not detrimental to trees in this study. Further experiments are needed to document the response of mature trees to annual damage as it relates to orchard decline.

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