Effect of Soil Fumigation and Pruning Date on the Indoleacetic Acid Content of Peach Trees in a Short Life Site

George E. Carter, Jr.
Department of Plant Pathology and Physiology, Clemson University, Clemson, SC 29631

Abstract. Peach tree (Prunus persica (L.) Batsch) short life, a disease complex resulting in premature death of peach trees, appears to be caused ultimately by cold injury to the vascular cambium. Indoleacetic acid (IAA) concentrations from the vascular cambium region of peach trees grown under various cultural practices were quantitated by thin-layer densitometry. Fall-pruned trees and trees in nonfumigated soil showed an elevated IAA level with respect to controls. It is suggested that early breaking of dormancy of the vascular cambium of peach trees, caused by an altered growth hormone balance, is responsible for the predisposition of certain trees to the cold injury death of peach tree short life.

Peach tree short life (PTSL) is a term that is used to describe a disease or disease complex that causes premature peach tree death, often in the first 4 years after the trees are set. Most trees affected by PTSL die during bloom or just after the foliage buds open. The young foliage fails to mature, withers, and abscises. The vascular cambium area exhibits a brown discoloration indicative of cold injury. A sour fermentation odor is very characteristic of the sap of affected trees (7).

Prince suggested that tree death in PTSL sites results from cold injury to the vascular cambium (15). Fall pruning, a detrimental cultural practice (8, 10, 16), reduces cold hardiness of trees. Soil fumigation, a beneficial practice in orchards where ring nematodes are present (6, 9, 11), promotes increased cold hardiness while controlling nematodes. Nesmith and Dowler (14) concluded that cold hardiness and cold injury were associated with tree survival on a PTSL site and that the observed PTSL symptoms could logically be expected from cold injury in the late winter after the chilling requirement has been met.

The fact that tree death from cold injury is scattered in PTSL sites suggests that certain trees are protected from cold injury. In many biological systems the primary adaptive protection mechanism is the dormant condition (2, 3, 18, 19, 23). That this condition of dormancy is under endogenous hormonal control is widely accepted and has been demonstrated in seeds (1, 2, 3). The auxin theory of cambial growth foliages that terminates cambial activity in spring is initiated by auxin produced in the expanding portion of the crown and transported to the stem (13, 22). A loss in cold hardiness has been reported to accompany this termination of dormancy (14). The study reported herein was initiated to examine the effects of fall pruning and fumigation on the IAA content of the vascular cambium of peach trees.

In 1973 'Blake' peach trees on Lovell rootstock were planted at the Sandhill Experiment Station near Columbia, SC, in Lakeland fine sand that was infested with Criconemoides xenoplax Raski. Peaches had been grown on the site twice previously. Half of the trees were planted in soil that was fumigated with 1,2-dibromo-3-chloropropane (Fumazone 85, 47 liters/ha treated). The remaining trees were planted in nonfumigated soil in a randomized block design. On March 31, 1975, 23 randomly selected tree sites were sampled and soil nematode populations were established by the sugar flotation centrifugation method (12). When the present experiment was initiated 20 trees were surviving in nonfumigated soil and 24 in fumigated soil. Trees were randomly numbered and were divided into 3 treatments as follows: (a) all trees in nonfumigated soil were fall-pruned (Nov., 1974) (NF-FP); (b) 12 randomly selected trees in fumigated soil were fall-pruned (F-FP); (c) the remaining 12 trees in fumigated soil were winter pruned (Feb., 1975) (F-WP). Duplicate samples consisting of 10 g fresh wt of terminal woody tissue from the 1974 growing season from 5 trees in each treatment were collected monthly from Nov., 1974, through May, 1975. Trees were pruned immediately after the Nov. or Feb. samples were collected.

Twig samples were immediately taken to the laboratory where all buds were cut off to prevent hormone translocation and were discarded. IAA from the twig samples was extracted by the procedure of Sengupta et al. (20) modified as follows:

Ten g fresh wt woody tissue were macerated in a Waring blender containing 100 ml 80% ice cold methanol. The blend was rinsed with 200 ml 80% methanol and the suspension was maintained at 4°C for 72 hr. After 72 hr the suspension was filtered and the residue washed with an additional 100 ml 80% methanol. The filtrate was rotary evaporated under vacuum at 35°C to remove the organic phase. The pH of the aqueous phase was adjusted to 3.0 with 2 N HCl. The acidic fraction containing IAA was collected by shaking the aqueous phase 3 times with equal volumes of methylene chloride and combining the organic phases. The methylene chloride was evaporated, and the extract redissolved in 100% methanol. This solution was further purified by stirring it with activated charcoal and then filtering it.

Aliquots (0.5 and 1 ml) of each extract were spotted with a Kontes Chromaflex spotter on activated 5 x 20 cm silica gel 6254 thin-layer plates (E. Merck). Chromatograms were developed to a height of 17 cm in a solvent system consisting of 75 toluene:45 ethyl acetate:6 acetic acid (v/v) and air dried about 10 min. Plates were examined on a Kontes scanning thin-layer densitometer coupled to a Houston Instrument recorder. Peaks on the recorder paper resulting from absorption of the ultraviolet light by IAA were measured with a compensating polar planimeter. Quantitative determinations were made from previously prepared standard curves. Evidence for authenticity of IAA in the extracts was developed by chromatography with known standards in a number of different solvent systems. The nonfumigated sites contained an average of 204 ring nematodes (Criconemoides sp.) per 100 cc of soil and, in some sites, a very few Helicotylenchus sp. and Tylenchorhynchus sp., while the fumigated sites contained an average of 20 individuals per 100 cc of soil (only one fumigated site contained any nematodes). Samples prior to this date showed similar counts.

Eight of 20 fall-pruned trees in nonfumigated soil died during the course
of the experiment while only one of 12 died in fumigated soil. No winter-pruned trees in fumigated soil died. On other rootstocks (Elberta and Halford), tree loss of winter-pruned trees in nonfumigated soil was 37%. The NF-FP treatment resulted in significantly more tree death than the F-FP or F-WP treatments (probability >f = 0.01%). Symptomatology associated with all tree death was identical to that previously described (7).

In Nov., trees in the nonfumigated treatment had significantly higher (5%) IAA concn than the plants in the fumigated treatments (Fig. 1). In Dec., after the fall pruning, the F-WP plants had significantly less (5%) IAA than the NF-FP plants. However, the NF-FP and the F-FP plants were not different. No significant differences occurred at the remainder of the sampling dates. Cultural practices have been shown to influence cold hardiness of peach on a PTSL site (14). The results of our study show that certain cultural practices also affect the IAA concn of peach trees. The high correlation between treatment and tree death, and the significant demonstrated between treatment and IAA levels strongly suggest a relationship between cold injury and IAA levels.

Various factors predispose peach trees to the cold injury type death seen in PTSL. Since tree death is neither uniform nor predictable, the susceptible trees seem to have lost the protected quality of the noninjured trees. Nematode infection and fall pruning, factors known to be injurious to peach trees (6, 8), affected the peach trees used in this study. Soil fumigation may have affected other factors associated with peach trees in this experiment, but nematode control has important effects in reducing losses to PTSL (6). Both nematodes and pruning constitute a wound stress on the tree. Classically a wound stress response is seen as an elevated cambial IAA level (5, 17, 21). It appears from this study that fall pruning affects IAA levels more strongly than does winter pruning. In fact, winter pruning does not appear to affect IAA levels. Trees in the fumigated treatment showed an immediate IAA increase after fall pruning, but not after winter pruning. In the nonfumigated treatment, however, IAA levels were high initially, and subsequent early pruning did not further stimulate IAA levels in these trees. The shift toward more growth promoter may have resulted in later entry of the vascular cambium into dormancy, earlier release from dormancy, or both. Either situation appeared to predispose trees to cold injury.

Endogenous hormonal control of dormancy is a complicated and many-faceted phenomenon (4). However, dormancy can be simplified if one envisions a biological system that contains dynamic levels of both growth-promoting and growth-inhibiting hormones. In such a system both environmental and physical parameters can effect changes in the concn of either or both hormones (4). It is suggested that in certain trees some event predisposes the tree to cold injury by altering the growth hormone balance to release the tree from dormancy. If the rest period of nondormant trees is then broken by the fluctuating cold-warm-cold temp common to the Southeast in late winter, cold injury to the active vascular cambium may result.

**Fig. 1.** The effect of fumigation (F) or nonfumigation (NF) and fall pruning (FP) or winter pruning (WP) on the endogenous IAA content of peach trees (g IAA per 10 g fresh weight bark).

### Literature Cited


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