Control of Mistletoe in Pecan Trees

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Abstract. Semi-parasitic evergreen mistletoe (Phoradendron flavescens Nutt.) is an increasingly serious weed causing loss of nut yield and tree vigor in pecan [Carya illinoiensis (Wangen.,) K. Koch] orchards of the southeastern United States. Several herbicides and growth regulators were evaluated for efficacy against mistletoe. The dimethylamine salt of 2,4-D proved to be an effective control agent. Ethephon, glyphosate, paraquat dichloride, and polyborate exhibited little or no long-term efficacy. The dimethylamine salt of dicamba also killed mistletoe, but exhibited potential for harming host trees. Dormant season treatment of mistletoe clusters with 2,4-D reduced photosynthesis by about one-third soon after treatment, and by ≥90% from 6 to 16 weeks posttreatment, but clusters did not die until ≥4 months posttreatment. Host limbs, less than ≥3 cm in diameter at the site of mistletoe attachment, usually died within 12 months of 2,4-D treatment of the associated mistletoe cluster. Treatment of entire host trees with 2,4-D did not harm trees if applied prior to ≥1 week of budbreak. Spot treatment of mistletoe clusters, with 2,4-D at ≥1 to 2.4 g·L⁻¹ a.i. (plus 2% crop oil), ≥2 to 3 weeks before budbreak, gave effective long-term control of mistletoe. The inclusion of a crop-oil in the 2,4-D spray greatly increased efficacy. Chemical names used: (2-chloroethyl) phosphonic acid (ethephon).

There are ≥1300 species of mistletoe worldwide (Milius, 2000); many of which parasitize horticultural and silvicultural crops. Of this group, the leafy mistletoes (Phoradendron sp.) are obligate semi-parasites of several hardwood tree species (Scharp and Hawksworth, 1974). Eastern mistletoe (Phoradendron flavescens Nutt.), a semi-parasitic evergreen, infects several species of North America’s mixed mesophytic hardwood forests (Harris, 1979). Parasitized trees act as infection sources for pecan trees and orchards throughout large sectors of the southeastern pecan belt of the United States. Canopies of affected pecan trees commonly support ≥500 mistletoe clusters. The long life span of pecan trees and orchards (≥100 years) increases the relevance of mistletoe parasitism of pecan.

Parasitized trees suffer from water and nutrient pirating (Stewart and Press, 1990), plus competitive shading of large sectors of canopy; consequently, trees are injured and often die (Haller, 1978; Sala et al., 2001; Stewart and Press, 1990; Tattar, 1978; Tinnin, 2001; Torngren et al., 1980). Colonization is usually most severe on relatively old trees (≥50 years old), but can also be severe on young trees (<20 years old). Thus, mistletoe is an arboreal weed possessing the ability to inflict major financial losses on pecan farming operations.

There are several possible approaches to mistletoe control. These include: 1) pruning, 2) biological, and 3) chemical. While pruning can be effective, it is often impractical because clusters in tall trees are inaccessible, or ineffective due to resprouting from the root-like haustorium that grows a meter or more down the host limb. Pruning is also undesirable due to wounds being potential entry points of pests (Haidfield and Flanagan, 2000). Biological control options offer potential, in that a stem-boring weevil (Myremex sp.) has been noted to kill clusters of Phoradendron serotinum (Solomon et al., 1984). Addition ally, Colletotrichum gloeosporiodes and Nectria neomacrosora are two fungi that have been reported to exhibit potential for control of certain species of mistletoe by attacking the crown and haustoria, respectively (Milius, 2000). Unfortunately, practical biological control and pruning options do not appear to presently exist for this increasingly invasive weed of mature commercial orchards. The most promising short-term option appears to be chemical control. Plant growth regulating substances exhibit potential for control of mistletoe (Michaides et al., 1987; Minko and Fagg, 1989; Johnson, 1992; Joyce and Rein, 1987). The objective of this study was to identify a chemical means of controlling mistletoe while not harming the pecan host. It is hypothesized that 2,4-D, and certain other growth regulators, can be used to safely control mistletoe in pecan trees and orchards.

Materials and Methods

Effectiveness of several plant growth regulators. Large (≥0.5 m diameter) clusters of mistletoe growing on pecan host limbs were treated with one of several chemical growth regulators. Treatments were applied with a pressurized hand gun sprayer until mistletoe foliage was dripping. Evaluated chemicals and concentrations were: ethephon [Ethephon-6 @ 720 g·L⁻¹ a.i.; applied at 2.5 and 22.5 mL·L⁻¹ (Micro Flo., Co., Lakeland, Fla.)]; glyphosate [Roundup Ultra @ 480 g·L⁻¹ a.i.; applied at 38 and 76 mL·L⁻¹ (Monsanto, St. Louis)]; polyborate [Borosil-10 @ 132 g·L⁻¹ a.i.; applied at 100 and 400 mL·L⁻¹ (Nortrace, Ltd., Greeley, Colo.)]; dimethylamine salt of dicamba (3,6-dichloro-0-anisic acid [Banvel (BASF Corp., Research Triangle Park, N.C.) @ 480 g·L⁻¹ a.i.; applied at 6 and 24 mL·L⁻¹]; dimethylamine salt of 2,4-D dichlorophenoxyacetic acid [Amine-4 2,4-D @ 449 g·L⁻¹ a.i.; applied at 5.2 and 22.5 mL·L⁻¹ (Plate Chemical Co., Fremont, Neb.)]; and paraquat dichloride [Gramoxone Extra @ 300 g·L⁻¹ a.i.; applied at 48 and 144 mL·L⁻¹ (Zeneca Agricultural Products, Wilmington, Del.)]. Application was 22 Mar. 1998, at an air temperature of ≥25 °C. Trees were dormant at this time, with budbreak (defined as outer bud scale split) occurring ≥1 Apr. All chemical treatments included a non-ionic surfactant (NIS) at 0.5% solution v/v (i.e., Biosurf-HF (Plate Chemical Co.)). The study consisted of seven chemical treatments (i.e., six regulators plus unsprayed control), two rates per chemical, and 10 clusters randomly selected from the lower canopy of twenty ≥80-year-old pecan trees. Clusters were evaluated for death of mistletoe clusters and host limbs 4 and 12 months posttreatment. Treatments were also usually assessed 3 years posttreatment.

Impact of dicamba and 2,4-D on host trees. Screening of the above mentioned chemicals was narrowed to dicamba and 2,4-D in 1999. The influence of these chemicals on pecan trees was assessed using dicamba (at 1.3 and 2.6 g·L⁻¹ a.i.) and 2,4-D (1.2 and 2.4 g·L⁻¹ a.i.) on 15-year-old ‘Stuart’ trees. All chemical treatments included a NIS at 0.5% v/v (i.e., Biosurf-HF). Trees were drenched with sprays for a period of 15- to 20-hrs posttreatment. Trees were evaluated ≥2 weeks after normal budbreak, and again in mid-July, for either foliar damage or budbreak suppression. Foliar damage was rated as follows: 1 = no damage; 2 = slight wavy-like pattern to leaflet margins; 3 = wavelike disorder plus reduction of leaflet size; 4 = severe distortion of leaflets; 5 = leaflet or shoot death. Budbreak suppression was rated as follows: 1 = majority of buds breaking at normal time of budbreak; 2 = most buds breaking 1 week after normal budbreak; 3 = most buds breaking 2 weeks after normal budbreak; 4 = most buds breaking 3 weeks after normal budbreak; 5 = shoot death or buds not breaking by ≥5 weeks after treatment.

Influence of 2,4-D on percentage of mistletoe clusters killed when whole trees were sprayed. The impact of 2,4-D sprays on whole trees was evaluated 22 Feb. 2000 by treating ≥15- to 50-year-old trees, possessing 10 to 50 mistletoe clusters, with 2,4-D concentrations of 0, 1.2, or 2.4 g·L⁻¹ a.i. Clusters, and the entire limb structure of dormant trees, were sprayed with a pressurized hand gun until soaked. Canopy volume of certain test trees was comprised of ≥50% mistletoe clusters. Experimental design was three 2,4-D treatments with seven single-tree replicates. Trees and clusters were evaluated for damage or death the following June, September, and a year later.
in June. The percentage of clusters killed per tree was determined in September.

Posttreatment damage to host limbs by 2,4-D. Mistletoe-infested limbs of 2,4-D (1.2 or 2.4 g · L⁻¹ a.i.) treated trees were evaluated for subsequent damage to host limbs. Host limb diameters ranged from ≈2 to 9 cm. Rated limbs came from several different trees and were rated 1 year posttreatment. The rating scale was: 1 = no visible harm; 2 = 5% to 25% of shoots dead; 3 = 26% to 50% of shoots dead; 4 = 51% to 99% of shoots dead; 5 = 100% of shoots dead.

Influence of 2,4-D on photosynthesis of mistletoe clusters. Because mistletoe clusters treated with 2,4-D took several months to die, clusters treated with 0, 1.2 or 2.4 g · L⁻¹ a.i. 2,4-D in early June (sprayed with a hand sprayer until leaf-drip) were evaluated for photosynthesis 1, 6, and 16 weeks after treatment. Net photosynthesis was measured on the apical-most leaf of the tip or the typical-most leaf of three randomly selected shoots. Measurements were taken from 1000 to 1200 hr using a LI-COR 6400 (LI-COR, Lincoln, Nebr.) portable open-system photosynthesis apparatus (Wood, 1988). Measurements were made at a photosynthetic photon flux of 1500 umol m⁻²·s⁻¹ at ≈400 ppm CO₂. The experiment consisted of three 2,4-D treatments, three dates of measurement, and three replications (i.e., a single cluster on each of three trees) as RCBS.

Influence of 2,4-D formulated with a surfactant. The efficacy of 2,4-D was further evaluated by treating large clusters of mistletoe (≥0.5-m diameter with hand-gun sprays of 2,4-D at 0.3, 0.6, 1.2, 1.8, or 2.4 g · L⁻¹ a.i.). Sprays were applied 22 Feb. 2000, with air temperatures ≈19 °C. All chemical treatments included a NIS at 0.5% v/v (i.e., Biosurf-HF). Treatments were applied to clusters on the lower canopy of five trees, resulting in six treatments with five replicates arranged in a RCB experimental design. Treatments were evaluated for damage in late June and again in mid-September. The five treated clusters per treatment were assessed for death and percentage killed regressed against 2,4-D concentration of the spray treatment.

Influence of 2,4-D + Dicyprop, oil. The efficacy of various amounts of 2,4-D applied in combination with a crop oil to aid penetration through the cuticle of mistletoe, was evaluated in 2002. The host cultivar was ‘Desirable’. Treatments were 2,4-D at 0, 0.3, 0.6, 1.2, 2.4 g · L⁻¹ a.i. applied 15 Feb., using a hand-gun sprayer to soak foliage. The crop oil used was Agri-oil (Chem Nut, Albany, Ga.) at 1% v/v (Agri-oil is 83% petroleum oil and 17% surfactant blend of polylol fatty acid esters). Treatments were evaluated monthly until August to assess damage to treated clusters. Experimental design was a RCB with five 2,4-D treatments (with a single tree possessing several clusters serving as the experimental unit) and five blocks. Treatments were evaluated in August for efficacy. A second study applied 15 Feb. 2002 to ‘Desirable’ trees compared a constant concentration of 2,4-D and surfactant, but with a variable concentration of oil. The RCB design consisted of five oil (Agri-oil) concentrations

**Results and Discussion**

Effectiveness of several plant growth regulators. Several of the evaluated growth regulators either damaged or killed mistletoe clusters (Table 1). Ethephon [2-chloroethoxy] phosphonic acid and paraquat dichloride, at both low and high rates, defoliated clusters without harming the host limb, but host shoots respouted mistletoe within 1 year posttreatment and some were once again relatively large clusters by 3 years posttreatment. Resprouting was also reported in a different species of mistletoe (i.e., *Phoradendron tomentosum*) after ethephon application (Joyce and Rein, 1987; Parks and Hoffman, 1991). Glyphosate and polyborate failed to either damage or kill clusters within 1 year after application (Table 1). Both 2,4-D or dicycamb killed mistletoe clusters within 4 months of treatment in late March. Earlier studies by French (1970) also noted that 2,4-D salts of esters exhibited efficacy against mistletoe, and Michailides et al. (1987) noted that localized treatments of severed stems of *P. tomentosum* with a mixture of 2,4-D plus dicycamb effectively controlled mistletoe up to 1.5 years posttreatment. Thus, of the chemicals evaluated, 2,4-D and dicycamb exhibited the greatest promise as potential control agents because clusters were killed and did not show signs of resprouting.

Application of the two auxin-type regulators (i.e., 2,4-D and dicycamb) during the bud swelling period did not kill branches of the pecan host, but they distorted subsequent clusters growing on 80-year-old ‘Mobile’ pecan trees.

### Table 1. Influence of a single application of growth regulators or herbicides, in mid-March, on mistletoe clusters growing on 80-year-old ‘Mobile’ pecan trees.

| Treatment | Amount of cluster killed or defoliated (%) | Resprouted (%) | Host limb killed (%) | Influence on mistletoe |
|-----------|------------------------------------------|----------------|---------------------|----------------------|
| Untreated | 0                                        | 0              | No                  | None                 |
| Ethephon  | @ 2.5 mL · L⁻¹                             | 100            | No                  | Partial defoliation  |
|           | @ 22.5 mL · L⁻¹                           | 100            | No                  | Total defoliation, respouting by July |
| Parquat   | @ 48 mL · L⁻¹                             | 80             | No                  | Defoliated, respouted from haustorium |
|           | @ 144 mL · L⁻¹                            | 90             | No                  | Defoliated, respouted from haustorium |
| Glyphosate| @ 38 mL · L⁻¹                             | 0              | No                  | Slightly chlorotic leaves |
|           | @ 76 mL · L⁻¹                             | 0              | No                  | Slightly chlorotic leaves |
| Boron     | @ 100 mL · L⁻¹                            | 0              | No                  | Necrotic leaf tips and margins |
|           | @ 400 mL · L⁻¹                            | 0              | No                  | Necrotic leaf tips and margins |
| Amines    | 4.24-D                                   |                | 100                 | Yes                  |
|           | @ 5.2 mL · L⁻¹                            | 90             | Yes                 | Chlorotic leaves, dead by July |
|           | @ 22.5 mL · L⁻¹                           | 100            | Yes                 | Chlorotic leaves, dead by July |
| Dicamba   | @ 6 mL · L⁻¹                              | 100            | Yes                 | Chlorotic leaves, dead by July |
|           | @ 24 mL · L⁻¹                             | 100            | Yes                 | Chlorotic leaves, dead by July |

1. Application was 22 Mar. 1999. Air temperature was ≈25 °C.
2. All chemical treatments included a non-ionic surfactant at concentration of 0.5% v/v (i.e., Surfel). Ethephon = Ethephon-6, ethephon (2-chloroethoxy) phosphonic acid @ 720 g · L⁻¹ a.i.
3. Glyphosate = Roundup Ultra; glyphosate @ 480 g · L⁻¹ a.i.
4. Boron = Borosol-10 comprised of polyborate @ 132 g · L⁻¹ a.i.
5. Dicamba = Banvel; dimethylamine salt of dicamba (3,6-dichloro-0-anisic acid) @ 480 g · L⁻¹ a.i.
6. Amines 4.24-D = Weed Killer; dimethylamine salt of 2,4-D dichlorophenoxyacetic acid @ 449 g · L⁻¹ a.i.
7. Parquat = Gramoxone Extra; paraquat dichloride @ 300 g · L⁻¹ a.i.
8. *Respouting by following March (i.e., 2000), after treatment in March (1999), from either the base of the mistletoe cluster or on the host limb within 1 meter of the attachment point of point of the cluster.
9. *Death of the pecan shoot beyond the attachment site for the mistletoe cluster.

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leaf development on nearby shoots of the mistletoe infected branch. Thus, these two herbicides were highly effective in killing mistletoe, but their application near the time of budbreak could harm the host. While application to trees just prior to budbreak (within 1–2 weeks) harms subsequent foliar canopy of limbs supporting the treated mistletoe cluster, this damage did not carry over into the second year after treatment.

Impact of dicamba and 2,4-D on host trees. The two most effective growth regulators from the first study were further evaluated for their deleterious impact on the pecan host. Treatment with either low or high concentrations of 2,4-D or dicamba did not suppress budbreak or distort (or kill) foliage when trees were treated 7 weeks prior to budbreak. Treatment 3 weeks prior to budbreak with the 1.2 g·L⁻¹ a.i. rate of 2,4-D did not suppress budbreak, but did induce wavelike leaflet margins. The high 2,4-D concentration (2.4 g·L⁻¹ a.i.) delayed budbreak for about 1 week and also induced leaflets with wavelike margins. Both dicamba treatments delayed budbreak 2 weeks and also induced wavelike leaflet margins, but also reduced leaflet size. Trees drenched 3 weeks before budbreak exhibited much less damage from the 2,4-D treatments than the dicamba treatments. Budbreak was delayed 1 week by the low 2,4-D concentration (1.2 g·L⁻¹ a.i.). It also distorted leaflet margins and reduced leaflet size.

No treatment killed trees (or limbs), delayed budbreak, or altered leaflet morphology the following crop year. These data indicate that: 1) 2,4-D is less damaging to the pecan tree host than dicamba; and 2) trees can safely tolerate exposure to both herbicides if applied during the dormant season.

Posttreatment damage to host limbs by 2,4-D. Death of host limb regions apical to the point of mistletoe attachment depended on the size of the host limb. Host limbs <3 cm in diameter at the mistletoe attachment point almost always died when clusters died (Fig. 2). Limb >3 cm in diameter rarely died. Thus, 2,4-D induced death of mistletoe clusters on small limbs usually also killed that portion of the host limb apical to the point of attachment. No damage was noted to regions basal to the attachment point. This death of small host limbs is evidence of a transfer of 2,4-D via the haustorium to the host limb.

Influence of 2,4-D on photosynthesis of mistletoe clusters. The foliage of 2,4-D treated mistletoe clusters gradually changed from green to yellowish-green, to greenish-yellow, to yellow, to brown within about 4 months posttreatment. Photosynthesis of 2,4-D (i.e., 1.2 or 2.4 g·L⁻¹ a.i.) treated mistletoe foliage declined one-third by 1 week posttreatment. Foliage was still green at this time and was indistinguishable from untreated foliage. By 6 and 16 weeks posttreatment, photosynthesis not shown, with no difference in efficacy between the two concentrations. Trees were undamaged by treatments during the crop year of application, but there was death to portions of host limbs apical to infection point evident the second crop season after application. Other portions of treated trees were undamaged. In this study, the spring foliage was slightly distorted and there was considerable death of small limbs apical to the attachment point of the mistletoe clusters. These host limbs were already greatly weakened by the attached clusters. The foliage of these trees was normal the second growing season after treatment.

Influence of 2,4-D on percentage of mistletoe clusters killed when whole trees were sprayed. Treatment of 15- to 50-year-old mistletoe infested trees in mid-February (>2 weeks prior to budbreak) with 2,4-D (1.2 or 2.4 g·L⁻¹ a.i.) killed >90% of clusters (data not shown), with no difference in efficacy between the two concentrations. Trees were undamaged by treatments during the crop year of application, but there was death to portions of host limbs apical to infection point evident the second crop season after application. Other portions of treated trees were undamaged. In this study, the spring foliage was slightly distorted and there was considerable death of small limbs apical to the attachment point of the mistletoe clusters. These host limbs were already greatly weakened by the attached clusters. The foliage of these trees was normal the second growing season after treatment.

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was 10% of untreated foliage (Fig. 3). Clusters were dead by 20 weeks posttreatment. Thus, photosynthesis of treated clusters declines soon after treatment, but continues at a low level for several months before they die.

**Influence of 2,4-D formulated with a surfactant.** The percentage of mistletoe clusters killed per tree with 2,4-D, applied 6 weeks before budbreak, increased hyperbolically with 2,4-D concentration (Fig. 4). It is noteworthy that while 1.2 and 2.4 g·L⁻¹ a.i. treatments effectively killed mistletoe (=85% of clusters killed), it had no apparent effect on certain adjacent clusters. This extreme variability in efficacy indicated that there was something about certain clusters that protected against 2,4-D. Thus, initiating an alternative approach involved the addition of crop oil to the spray solution to increase 2,4-D penetration.

**Influence of 2,4-D formulated with crop oil.** The addition of crop oil to the 2,4-D spray increased efficacy (Fig. 5). When the amount of 2,4-D was held constant at 1.2 g·L⁻¹ a.i. (selected because it killed well), but the amount of oil varied from 0 to 2%, the percent kill increased in a linear manner from 73% at 0% oil to 90% at 2% oil. When the amount of oil was set at 2% and the amount of 2,4-D varied from 0 to 4.8 g·L⁻¹ a.i., the amount of damage to mistletoe clusters peaked at about 95% at 1.2 g·L⁻¹ a.i. (Fig. 6). Higher concentrations of 2,4-D did not increase efficacy. When infected trees were totally drenched 4 weeks before budbreak with 2.4-D at 2.4 g·L⁻¹ a.i. + 1% oil, there was no phytotoxicity in the current or the subsequent growing season (data not shown).

The mistletoe population parasitizing these trees was reduced by >90% without harming the host tree. It should be noted that because of differences in dates of application, sites, air temperatures, and probably physiological states, a particular 2,4-D treatment varied in efficacy.

It is noteworthy that application of 2,4-D treatments ≈2 weeks before budbreak killed clusters better than in the other studies where application was several weeks before budbreak. Clusters treated 4 to 7 weeks before budbreak often appeared unharmed or displayed undamaged foliage at shoot tips. By comparison, clusters treated 2 weeks before budbreak rarely survived. Thus, 2,4-D efficacy appears to increase as time of application nears budbreak of the pecan host and may be associated with increased physiological activity as host xylem sap flow increases. This indicates that in order to protect the host tree while killing the mistletoe, the 2,4-D treatment should probably be applied about 2 weeks before budbreak.

**Conclusion.** These data indicate that 2,4-D application during dormancy of the host tree can effectively control mistletoe in pecan orchards. Ethephon ([2-chloroethyl] phosphonic acid) and paraquat dichloride effectively defoli-
Growth regulators

Fig. 6. Efficacy of different amounts of 2,4-D for killing mistletoe clusters when applied 2 weeks prior to budbreak.

Of the several regulators evaluated, the dimethylamine salt of 2,4-D at ≈1.2 to 2.4 g·L⁻¹ a.i. sprayed ≈2 weeks before budbreak possesses the greatest feasibility for mistletoe control in pecan orchards. It is currently "labeled" for weed control in pecan orchards and might potentially be specifically labeled for mistletoe control in pecan orchards. It is currently "labeled" for weed control in pecan orchards and might potentially be specifically labeled for mistletoe control. While both the NIS and crop oil options are highly effective, the 2,4-D + 2% crop oil option is most suitable for spot-spraying individual clusters or whole trees. While this treatment often kills small host limbs (i.e., <3 cm in diameter), it has no adverse effect on trees the following year. Treated clusters soon decline in photosynthesis and continue to do so until they die ≈4 months later. Treatment of host trees within ≈1 week of budbreak (defined as outer budscale split) can distort foliage and delay budbreak. The proper usage of 2,4-D appears to offer a practical means of eradicating mistletoe from pecan orchards.

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