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Abstract

Palmer amaranth, a dioecious summer annual species, is one of the most troublesome weeds in the agronomic crop production systems in the United States. In the last two decades, continuous reliance on herbicide(s) with the same mode of action as the sole weed management strategy has resulted in the evolution of herbicide-resistant (HR) weeds, including Palmer amaranth. By 2015, Palmer amaranth biotypes had been confirmed resistant to acetolactate synthase (ALS)-inhibitors, dinitroanilines, glyphosate, hydroxyphenylpyruvate dioxygenase (HPPD)-inhibitors, and triazine herbicides in some parts of the United States along with multiple HR biotypes. Mechanisms of herbicide-resistance in Palmer amaranth are discussed in this chapter. Preplant herbicide options including glufosinate, 2,4-D, and dicamba provide excellent Palmer amaranth control; however, their application is limited before planting crops, which is often not possible due to unfavorable weather conditions. Agricultural biotechnology companies are developing new multiple HR crops that will allow the post-emergence application of respective herbicides for management of HR weeds, including Palmer amaranth. For the effective in-crop management of Palmer amaranth, and to reduce the potential for the evolution of other HR weeds, growers should apply herbicides with different modes of action in tank-mixture and should also incorporate cultural practices including inversion tillage and cover crops along with herbicide programs.

Keywords: Biology, evolution, germination, genetics, integrated management, mechanisms of resistance, physiology, resistance management
1. Introduction

Palmer amaranth (*Amaranthus palmeri* S. Wats.), also known as careless weed, is native to the southwestern United States [1], and is a summer annual belonging to the family Amaranthaceae, which includes around 75 species worldwide [2]. Palmer amaranth leaves and seeds were an important food source in many Native American tribes [3]; when roasted and milled, the seeds of *Amaranthus* spp. are edible and rich in protein, whereas the leaves are high in Ca, Fe, and vitamin A [4,5]. However, Palmer amaranth plants grown under dry conditions can build up nitrates at levels harmful for cattle to consume [6]. Palmer amaranth was historically located in the southern United States; however, human activities in the 20th century—including seed and equipment transportation and agriculture expansion—led to the spread of Palmer amaranth to the northern United States, and it was first reported beyond its original habitat in Virginia in 1915 [7]. In the 1989 annual survey of the Southern Weed Science Society, Palmer amaranth appeared as a troublesome weed in the southern United States [8], and though in 1995 Palmer amaranth was listed as the most troublesome cotton weed in only two southern states (North and South Carolina) [9], by 2009, it was ranked as the most troublesome cotton weed in nine southern states and the second most troublesome weed in soybean [10]. By 2014, Palmer amaranth had become one of the most troublesome and economically important weed species in corn, cotton, and soybean in the United States [10–12].

Palmer amaranth and common waterhemp (*Amaranthus rudis* Sauer) are the only dioecious species (separate male and female plants) of all the pigweeds, whereas redroot pigweed (*Amaranthus retroflexus* L.), smooth pigweed (*Amaranthus hybridus* L.), spiny amaranth (*Amaranthus spinosus* L.), and tumble pigweed (*Amaranthus albus* L.) are monoecious (male and female flowers on the same plant). Palmer amaranth is characterized as a tall (around 2 m long), erect, broadleaf weed with lateral branching. The leaves are hairless, alternate, and lanceolate shaped in young plants and become ovate as plants mature [13]. The upper side of the leaves is often marked with a darker, V-shaped chevron. Palmer amaranth’s leaf petiole is as long as or longer than its leaf blade, whereas common waterhemp has a leaf petiole smaller than its leaf blade. The flowers of Palmer amaranth cluster together to form a terminal cylindrical inflorescence, and while the male and female inflorescences look identical, they can be distinguished by touch: the male inflorescence is softer, whereas the female inflorescence is rougher and pricklier. Chromosome number $2n = 34$ as well as chromosome number $2n = 32$ has been reported in Palmer amaranth [14–16].

2. Reproduction biology

As a dioecious species, Palmer amaranth is an obligate outcrosser [17], with pollination occurring by wind. Male plants produce large numbers of pollen seeds with a mean diameter range of 21–38 µm and a mean density of about 1,435 kg m$^{-3}$ [18]. This allows Palmer amaranth pollen to move long distances from the source plant; however, the viability of the pollen is reduced within 30 minutes of anthesis [19]. Previous research has reported the pollen-
mediated transfer of glyphosate-resistant traits from glyphosate-resistant male Palmer amaranth plants to glyphosate-susceptible female plants up to a distance of 300 m [20]. Apparent agamospermy (asexual reproduction or seed production from an unfertilized ovule) was also reported in female Palmer amaranth plants isolated from a pollen source, or in those that had been pollinated by common waterhemp [21,22].

Palmer amaranth plants normally flower during September and October [23]; however, decreasing day lengths hasten the flowering process [24]. Its seeds are smooth, round- or disc shaped, 1–2 mm in diameter [13], and are usually dispersed by gravity. In addition, seed dispersal via irrigation, birds, mammals, plowing, mowing, and harvesting has also been reported in Palmer amaranth [25,26]. Female Palmer amaranth plants are prolific seed producers even under conditions of higher competition in agronomic cropping systems; for example, in North Carolina, Palmer amaranth at densities of 5.2 plants m$^{-1}$ of peanut row produced about 124,000 seeds m$^{-2}$ [27]. In Kansas, Palmer amaranth at densities of 0.5 and 8 plants m$^{-1}$ of corn row produced around 140,000 and 514,000 seeds m$^{-2}$, respectively [28] (Figure 1). In California, Palmer amaranth plants that emerged between March and June produced more seeds (200,000–600,000 seeds plant$^{-1}$) compared with plants that emerged between July and October (≤ 80,000 seeds plant$^{-1}$) [24] (Figure 2).

Figure 1. A female Palmer amaranth plant in a cornfield in Nebraska, USA. This plant has the capacity to produce more than half a million seeds.
Late-emerging Palmer amaranth plants can also produce seeds later in the season that will add to the soil seed bank.

3. Seed germination

3.1. Temperature

One of the most important environmental factors required for seed germination is the range of temperatures to which seeds are exposed. The optimum temperature range for Palmer amaranth seed germination extends from 25 to 35°C [29]. However, Palmer amaranth seeds showed higher germination at alternating day/night temperatures of 25/20, 30/15, 35/20, 35/0, and 35/25°C compared with constant temperatures of 15, 20, 25, 30, and 35°C, respectively, with no germination at 15/10°C [29,30]. The estimated base temperature (minimum temperature below which phenological development ceases) for Palmer amaranth is 16.6°C, which is higher than other summer annual weeds, including barnyard grass (*Echinochloa crus-galli*), common purslane (*Portulaca oleracea*), large crabgrass (*Digitaria sanguinalis*), and tumble pigweed [31]. Palmer amaranth seeds showed less dormancy and germinated in a wider range of temperatures when followed by winter after-ripening compared with freshly matured seeds [32]. The induction of secondary dormancy was also reported in Palmer amaranth seeds exposed to high temperatures in summer [32].
3.2. Light

Light plays an important role in breaking dormancy and promoting germination in most of the *Amaranthus* species [33,34]. The quantity of light received by the mother plant has a profound effect on Palmer amaranth seed germination; for instance, the seeds of the female plant grown under full sunlight showed higher germination (25%) in darkness compared with plants that experienced low quantities of light (12%) [32]. Palmer amaranth germination response is partially mediated by phytochrome when followed by after-ripening in the field [32].

3.3. Plant growth hormones

The ratio of abscisic acid (ABA) and gibberellic acid (GA) regulates the physiological dormancy of seeds, with ABA promoting seed dormancy and GA preventing seed dormancy [35–37]. The levels of ABA and GA in seeds are affected by the environmental conditions experienced by the maternal plant during seed development [38,39], and in Palmer amaranth, increased exposure of the mother plants to shade increased the ABA content in seeds, which in turn reduced germination levels in dark conditions and promoted dormancy [40].

3.4. Seed location

The location of the inflorescence on the mother plant as well as the location of the seed within an inflorescence can affect seed germination [36,41]. In Palmer amaranth, 67–78% greater germination was reported from seeds matured in the middle and top third of a female plant than from seeds matured in the bottom third of the plant [40].

3.5. Seed burial depth and duration

The depth and duration of seed burial determine the seed germination, viability, and longevity in the soil. The viability of Palmer amaranth seeds buried at different soil depths (1–40 cm) reduced to < 40% within 3 years of burial; however, more deeply buried seeds showed higher viability than seeds buried at shallow depths [42]. In another study, Palmer amaranth seedlings showed ≥ 35% emergence at burial depths less than 3 cm compared with ≤ 7.2% emergence from seeds buried at depths greater than 5 cm [24].

4. Competitive abilities

4.1. Photosynthesis and growth rate

Growth rate is the chief index of plant competitiveness [43], and Palmer amaranth’s aggressive growth habit and prolific seed production make it a serious and problematic weed in agro-nomic cropping systems [44,45]. Palmer amaranth has the highest plant dry weight, leaf area, height, growth rate (0.10–0.21 cm per growing degree day), and water-use efficiency compared with other pigweeds, including common waterhemp, redroot pigweed, and tumble pigweed.
Palmer amaranth plants also respond more positively to higher temperatures and develop more root and shoot biomasses compared with common waterhemp and redroot pigweed [29,47,48]. Different physiological and morphological characteristics contribute to the greater growth of Palmer amaranth even under water stress conditions. Under high soil water availability, Palmer amaranth has a high photosynthetic capacity (80 µmol CO₂ m⁻² s⁻¹) and its photosynthesis is not light saturated (except at a lower leaf water potential of −2.9 MPa) at an irradiance level of 400–700 nm [49]. In addition, Palmer amaranth leaves can orient themselves perpendicular to incoming sunlight (diaheliotropism), allowing the plant to take advantage of its high photosynthetic capacity [50]. High photosynthetic rates along with diaheliotropic movement allow the plant to fix carbon at faster rates, promoting more aggressive growth. Although conditions of prolonged drought can decrease leaf water potentials in most plants, resulting in closure of the stomata during the day, Palmer amaranth can respond to lower leaf water potentials by increasing its leaf solute concentration, allowing the stomata to remain open longer during periods of drought [51].

Shading causes a detrimental effect on plant growth by reducing the quality, quantity, and intensity of photosynthetic active radiations (PAR) [52–54]. C₃ weed species can adapt better to lower light conditions [55,56] compared with C₄ species that are better adapted to higher irradiance levels [54,57]. However, Palmer amaranth plants have also reported adapting to reduced irradiance levels by lowering their light compensation and leaf and main-stem branch appearance rates, or by increasing their total leaf chlorophyll, leaf dark respiration, and specific leaf area [58].

### 4.2. Root morphology

Palmer amaranth has a deep and fibrous root system with a root to shoot biomass ratio of 0.16 ± 0.02 [59]. Palmer amaranth roots are finer, longer, and greater in number compared with soybean with a similar root fresh weight [48]. This root morphology enables Palmer amaranth to occupy a much larger soil volume and gain a competitive advantage over other crops in the acquisition of nutrients, especially during conditions of drought and low fertility. Palmer amaranth roots can penetrate highly compact soils and are more efficient in their nitrogen uptake compared with soybean genotypes [60]. In addition, Palmer amaranth can maintain stable overall shoot and root growth when downward root growth becomes restricted due to the compact soil or hard pans commonly found in Piedmont and coastal plain soils [60].

### 4.3. Allelopathy

Palmer amaranth, along with other *Amaranthus* species, exerts allelopathic effects on several crops and weeds [61–63]. Menges [61] reported that soil-incorporated residues of Palmer amaranth inhibited carrot (*Daucus carota* L.) and onion (*Allium cepa* L.) growth by 49% and 68%, respectively. Among the soil-incorporated residues, thyrsus (inflorescence) and leaf tissues caused more seedling damage in carrot, onion, cabbage (*Brassica oleracea* var. *capitata* L.), and sorghum (*Sorghum bicolor* L. Moench.) compared with the stems and roots of Palmer amaranth [62].
4.4. Host to plant bugs and nematodes

Palmer amaranth is an important host for tarnished plant bugs, a major pest of cotton in the midsouthern United States, and is also a host for nematodes in tobacco [64]. Crop rotation from susceptible host crop to non-host crop is an effective nematode management strategy; however, Palmer amaranth’s presence during the non-host crop season reduced the benefits of crop rotation.

4.5. Seed herbivory

Palmer amaranth seeds make up a food source for various animals, birds, and insects. Sosnoskie et al. [42] reported Palmer amaranth seed removal by rodents from seed traps, though the percentage of seed consumption is unknown. Palmer amaranth seeds are also consumed by birds such as killdeer (Charadrius vociferus) and mallard ducks (Anas platyrhynch), remaining viable even after passing through their intestinal tracts. This viability allows these birds to serve as a vector for Palmer amaranth seed dispersal across long distances [65].

5. Crop yield losses

The widespread adoption of no-tillage systems, reduced reliance on soil-applied residual herbicides, and increased herbicide resistance have contributed toward the increased infestation of Palmer amaranth in different cropping systems [45,66,67].

5.1. Cotton

Palmer amaranth’s season-long interference in cotton at densities of 10 plants per 9.1 m$^2$ area reduced cotton canopy volume, biomass, and yield by 45%, 50–54%, and 54%, respectively; however, cotton canopy height and lint properties were not affected [68]. In another study, 6–11.5% cotton yield reduction was reported with each Palmer amaranth plant in 9.1 m$^2$ area [69]. In addition to yield reduction, Palmer amaranth interferes during cotton harvesting operations and increases harvest time by two- to threefold compared with weed-free control [70]. The presence of Palmer amaranth during cotton harvesting often causes cotton lint contamination, thus increasing lint-cleaning requirements, adding to the cost of production [70].

5.2. Soybean

Development of a dense and early-season canopy makes soybean more competitive to weed pressure compared with cotton [71]. However, soybean yield reduction ranging from 17% to 68% was reported in Arkansas at Palmer amaranth densities of 0.33–10 plants m$^{-1}$ of row [72]. In Kansas, soybean yield reduction of about 78%, 56%, and 38% was reported by Palmer amaranth, common waterhemp, and redroot pigweed, respectively, at an individual density of 8 plants m$^{-2}$ [73]. Early-emerging Palmer amaranth plants also cause more soybean grain yield loss compared with later-emerging plants [73].
5.3. Corn

Palmer amaranth plants that emerged along with corn caused higher corn grain yield losses (11–91%) compared with plants emerging later than corn (7–35%) at the same densities of 0.5–8 plants m$^{-1}$ of row, respectively [28]. Palmer amaranth at densities of 0.5–8 plants m$^{-1}$ of cornrow caused 1–44% corn forage yield loss, respectively [74], and under dryland production systems, Palmer amaranth at 1–6 plants m$^{-1}$ of cornrow also resulted in 18–38% corn grain yield loss [75].

5.4. Peanut

Palmer amaranth plants outgrow peanut (*Arachis hypogaea* L.) and interfere with growth and harvesting operations. Palmer amaranth decreased the canopy diameter of peanut and caused between 28% and 68% yield loss at densities of 1 and 5 plants m$^{-1}$ of peanut row, respectively [27].

5.5. Sorghum

Palmer amaranth at 1.58 plants m$^{-2}$ was reported to cause 38–63% sorghum grain yield loss [76], and in addition, Palmer amaranth infestation increased sorghum grain moisture content, thus delaying harvesting operations.


One of the inevitable consequences of the repeated use of single mode-of-action herbicides as a primary weed control strategy is the selection of weeds resistant to that particular herbicide. To date, a total of 245 species are confirmed resistant to 22 of the 25 known mode-of-action herbicides [77]. In addition, weeds can evolve resistance to multiple herbicides through sequential selection [78]. Prolonged and repeated use of herbicides with different modes of action resulted in the evolution of multiple herbicide-resistant Palmer amaranth populations in several parts of the United States [79]. Palmer amaranth is one of the few weeds in the United States that have evolved resistance to multiple modes of action herbicides (e.g. microtubule-, photosystem (PS) II-, acetolactate synthase (ALS)-, 5-enol-pyruvylshikimate-3-phosphate synthase (EPSPS)-, and hydroxyphenylpyruvate dioxygenase (HPPD) inhibitors) [77]. Consequently, the infestation of multiple herbicide-resistant Palmer amaranth populations remains a serious threat to agriculture throughout the United States as no herbicides with new sites- or modes of action have been commercialized in the past two decades [80].

Weed resistance against herbicides can be conferred either by (a) target site resistance (TSR) and/or (b) non-target site resistance (NTSR) mechanisms. TSR mechanisms largely involve mutation(s) in the target site of action of an herbicide, resulting in an insensitive or less sensitive target protein for the herbicide [78]. In such cases, the TSR is determined by monogenic traits [81]. In addition, weeds can evolve TSR as a result of overexpression or amplification of the target gene [82]. On the other hand, NTSR mechanisms include re-
duced herbicide uptake/translocation, increased herbicide detoxification, decreased herbicide activation rates, and/or herbicide sequestration [83]. Metabolism-based NTSR involves increasing the activity of enzyme complexes such as esterases, cytochrome P450s, glutathione S-transferases (GSTs), and/or UDP-glucosyl transferases [78]. NTSR—especially if it involves herbicide detoxification by these enzymes—is usually governed by many genes (polygenic) and may confer resistance to herbicides with completely different modes of action [81,84]. Evolution of NTSR via means of herbicide detoxification is a serious threat to weed management, as it can bestow resistance to multiple herbicides, leaving growers with limited herbicide options for weed control as well as granting weeds with potential resistance to herbicides not yet commercially available [85]. Furthermore, it has been proposed that low herbicide doses result in the evolution of polygenic traits, whereas high herbicide doses may favor monogenic target site-based resistances [86–88].


As indicated above, Palmer amaranth populations have evolved resistance to at least five modes of action of herbicides: microtubule-, PSII-, ALS-, EPSPS-, and HPPD-inhibitors. Some Palmer amaranth populations across the United States have also been found resistant to more than two mode-of-action herbicides [77].

7.1. Resistance to microtubule assembly-inhibitors in Palmer amaranth

Microtubules are important components during cell division that help chromosome movement during the anaphase of the cell cycle. Microtubules are long cylindrical molecules made up of protein, and tubulin. Some herbicides, such as trifluralin, inhibit microtubule formation during cell division, resulting in cessation of growth in meristematic regions. Palmer amaranth resistant to trifluralin was reported as early as 1989 and 1998 in the United States in South Carolina and Tennessee, respectively; however, the mechanism of resistance is unknown. The Palmer amaranth population from South Carolina is also cross-resistant to other dinitroaniline herbicides such as benefin, isopropalin, pendimethalin, and ethalfluralin [89].

7.2. Resistance to PSII inhibitors in Palmer amaranth

PSII inhibitors (e.g. atrazine, simazine, bromoxynil, etc.) are among the most popular and widely used herbicides, and have been in use in corn and grain sorghum for several decades. PSII inhibitors compete for the plastoquinone-binding site in the electron transport chain in PSII; this inhibits electron flow in the light reaction of photosynthesis, resulting in depletion of reducing power (NADPH) and ATP synthesis, which are required for the Calvin cycle. Atrazine resistance in the majority of weeds was reported to be due to nucleotide substitution in the psbA gene that encodes D1 protein in PSII (the target site of atrazine in the chloroplast) and hence is maternally inherited. PSII inhibitor resistance in Palmer amaranth was first documented in 1993 in Texas and thereafter reported in other states, namely, Kansas (1995), Georgia (2008), and Nebraska (2011) [77]. Although the mechanism of resistance to atrazine in
Amaranthus species such as waterhemp [90] and Amaranthus powelli [91] is reported as Ser-264 to Gly substitution in the psbA gene, the mechanism of resistance to PSII inhibitors in Palmer amaranth is not known.

7.3. ALS inhibitor resistance in Palmer amaranth

ALS-inhibitors inhibit the ALS enzyme, which is required for the biosynthesis of branched-chain amino acids valine, leucine, and isoleucine. ALS-inhibitor resistance in Palmer amaranth is widespread across the United States [77], and many Palmer amaranth populations also exhibit cross-resistance to several ALS-inhibiting herbicides. For example, imazethapyr-resistant Palmer amaranth from Kansas was found to be approximately 2,800 times more resistant to imazethapyr compared with the sensitive biotype, with the population also cross-resistant to the sulfonyleurea herbicides such as thifensulfuron and chlorimuron. ALS enzyme inhibition assays suggest the presence of an insensitive ALS enzyme, possibly because of a target-site mutation [92] in this population. Similarly, imazaquin-resistant Palmer amaranth from Arkansas is also cross-resistant to chlorimuron, diclosulam, and pyrithiobac [93]. Although the specific mutation/s contributing to the resistance of imidazolinone/sulfonyleurea herbicides in Palmer amaranth is unknown, in other Amaranthus species such as waterhemp and smooth pigweed, mutations in the ALS gene at amino acid positions W574 (tryptophan) or S653 (serine) [94] and A122 (alanine), A205 (alanine), D376 (aspartate), W574 (tryptophan), or S653 (serine) [95], respectively, are known to confer ALS-inhibitor resistance.

7.4. Glyphosate-resistance in Palmer amaranth

Glyphosate is the most widely used agricultural pesticide globally and it is used extensively in Roundup Ready corn, cotton, and soybean crops in the Midwestern United States. Glyphosate inhibits EPSPS enzyme synthesis in plants, thus preventing the biosynthesis of the aromatic amino acids phenylalanine, tyrosine, and tryptophan, and resulting in the death of glyphosate-sensitive plants. Glyphosate resistance in Palmer amaranth was first documented in 2004 in Georgia [96], and since then Palmer amaranth populations resistant to glyphosate have been documented in several U.S. states [77]. Glyphosate resistance in Palmer amaranth is conferred by two mechanisms: TSR due to EPSPS gene amplification, and NTSR through reduced absorption and translocation of glyphosate. These resistance mechanisms evolved independently at two locations due to intense glyphosate use in Roundup Ready cropping systems. The mechanism of resistance in Georgia Palmer amaranth populations was investigated, and for the first time it was found that resistant plants have increased EPSPS gene copies (> 100 copies), which are distributed throughout the genome. The EPSPS copies are also functionally correlated to the increase in enzyme expression to resist high rates of glyphosate [97]. EPSPS gene amplification as the mechanism of glyphosate resistance was also reported in Palmer amaranth populations from North Carolina [98,99], Mississippi [100], and New Mexico [101], whereas low levels of resistance to glyphosate because of reduced uptake and translocation were reported in Palmer amaranth populations from Tennessee [102] and Mississippi [103].
7.5. HPPD inhibitor-resistance in Palmer amaranth

Both PSII- and HPPD-inhibitor (e.g. mesotrione, tembotrione, pyrasulfotole) herbicides are used mostly as premixes, although it is not uncommon to use them alone. HPPD-inhibitors primarily inhibit carotenoid and tocopherol biosynthesis in plants [104]. In photosynthesis, carotenoids protect chlorophyll from photo-oxidation that occurs due to the formation of triplet chlorophyll and singlet oxygen [104]. Ultimately, sensitive species will die because of lipid peroxidation, leading to membrane destruction. HPPD-inhibiting herbicides are effective management tools for controlling glyphosate-, triazine-, and ALS-inhibitor-resistant Palmer amaranth populations in corn and grain sorghum; however, some Palmer amaranth biotypes were not controlled following treatment with pyrasulfotole and bromoxynil (formulated in 1:8 ratio in Huskie®) in central Kansas [105]. Paradoxically, this particular field had no previous history of HPPD-inhibitor herbicide use. This Palmer amaranth biotype was 7–11 times more resistant to premixes of pyroxsulfotole and bromoxynil than susceptible biotypes were [105]. Furthermore, inheritance studies suggested that the HPPD-resistant trait in this population is transmitted via pollen. To date, HPPD-inhibitor resistance has been reported in only two weed species; *viz.*, Palmer amaranth and waterhemp, although from several Midwestern states (Kansas, Nebraska, Illinois, and Iowa) [77]. It is highly likely that HPPD-inhibitor resistance may spread via pollen or seed to other regions and possibly into other related species through pollen-mediated interspecific hybridization [17,106]. Experiments are in progress to determine the mechanism of HPPD-inhibitor resistance in this Palmer amaranth biotype.

8. Management of Palmer amaranth

8.1. Chemical control

Herbicide-resistant Palmer amaranth management with herbicides should involve herbicides with different modes of action. An ideal management program should start with a preplant burndown treatment followed by a PRE residual herbicide, and one or two POST herbicide applications. It is also recommended to tank-mix POST treatments with a residual herbicide to prevent Palmer amaranth emergence later in the growing season.

8.1.1. Corn

Several PRE corn herbicides containing single or multiple active ingredients including acetochlor, alachlor, atrazine, dimethenamid-P, flumioxazin, fluthiacet-methyl, isoxaflutole, mesotrione, pyroxasulfone, S-metolachlor, and saflufenacil can effectively control emerging Palmer amaranth resistant to both ALS-inhibitors and glyphosate [107,108]. Palmer amaranth 16–20 plants m$^{-2}$ was controlled 95%, 78%, and 44% with acetochlor, atrazine, and flufenacet plus isoxaflutole, respectively, at 10–12 weeks after planting [109]. Palmer amaranth at moderate densities (8–10 plants m$^{-2}$) was controlled at least 97% with acetochlor, atrazine, and flufenacet plus isoxaflutole [109].
There are multiple POST herbicide options for controlling Palmer amaranth resistant to both ALS-inhibitors and glyphosate in glyphosate-tolerant corn. Commonly used POST herbicides may contain one or more active ingredients from growth regulators (2, 4-D, dicamba, and difluenzopyr), HPPD inhibitors (mesotrione, tembotrione, and topramezone), and PS II inhibitors (atrazine) [107]. Of these POST herbicides, 2, 4-D and dicamba provide only POST activity, whereas atrazine, mesotrione, tembotrione, and topramezone can provide both PRE and POST control of Palmer amaranth. If the herbicide label allows tank mixing, a residual herbicide such as acetochlor or S-metolachlor should be combined with a POST herbicide to prevent Palmer amaranth emergence later in the season. Another viable alternative is planting a glufosinate-tolerant (LibertyLink® trait) variety of corn and using glufosinate with labeled POST tank-mix partners.

8.1.2. Cotton

An ideal herbicide program for controlling herbicide-resistant Palmer amaranth in cotton should begin with tillage or a preplant burndown treatment containing a residual herbicide to produce a clean start [110,111]. For small (≤ 10 cm) emerged Palmer amaranth seedlings, glufosinate or paraquat can be used in tank-mixture with a residual herbicide such as flumioxazin or diuron at or prior to planting [7,112–115]. When flumioxazin is used in a burn-down program, depending upon the flumioxazin rate used, cotton planting should be delayed 15–21 days after burndown treatment in a no-till system and 30 days in a conventional tillage system to prevent cotton injury.

The preplant burndown treatment should be followed by a residual PRE treatment. Several residual PRE herbicides including diuron, flumeturon, fomesafen, pendimethalin, and prometryn can be used to achieve early-season control of Palmer amaranth resistant to ALS-inhibitors, glyphosate, or both [115–118,115]. Pyrithiobac, an ALS-inhibitor herbicide, may be used PRE for controlling glyphosate-resistant Palmer amaranth, though it will not control ALS-resistant populations. Research conducted in Alabama reported 90% Palmer amaranth control at 6 weeks after treatment with PRE application of pendimethalin plus fomesafen [119]. In North Carolina, flumeturon or pendimethalin PRE-alone programs failed to provide adequate (< 60%) control of Palmer amaranth late in the season [120]. Flumeturon was reported less effective on Palmer amaranth than diuron or fomesafen in studies conducted in Georgia and North Carolina [121], and in another study, pyrithiobac (71 g ha⁻¹) applied as preplant incorporation (PPI) or PRE controlled Palmer amaranth ≥ 97% at 6 weeks after treatment [122]. Research conducted at the University of Georgia recommended a PRE program consisting of tank mixing two herbicides out of acetochlor, diuron, and fomesafen [110]. However, the effectiveness of these PRE herbicides is quite variable and depends upon timely activation by rain or irrigation.

Unfortunately, limited POST herbicide options are available for controlling glyphosate-resistant Palmer amaranth in glyphosate-resistant cotton. Pyrithiobac and trifloxysulfuron applied POST can control small (≤ 10 cm) Palmer amaranth [122–124]; however, Palmer amaranth control with pyrithiobac applied POST may vary depending upon environmental
conditions [122]. In previous studies, pyrithiobac and trifloxysulfuron caused significant cotton injury (35%); however, the injury was transitory and did not affect the yield [124–128]. POST herbicides such as diuron, fluometuron, and prometryn can be tank-mixed with MSMA (monosodium methyl arsenate) for Palmer amaranth control in both transgenic and non-transgenic cotton. These herbicides can control small and newly emerged weeds, and provide residual control [124,129,130]. However, growers often miss the height differential period necessary for POST application because of Palmer amaranth’s rapid growth rate [45]. In addition, POST herbicides may injure cotton and often adversely affect cotton maturity and yield [131,132].

Research conducted in the southern United States recommended one to two follow-up tank-mixed applications of a residual herbicide such as pyrithiobac or S-metolachlor, and a POST herbicide such as glyphosate in glyphosate-tolerant cotton or glufosinate-tolerant cotton for Palmer amaranth control [110,117,119]. However, glyphosate and pyrithiobac tank-mixed applications will not be a viable option for controlling Palmer amaranth resistant to both ALS-inhibitor and glyphosate. Therefore, for controlling Palmer amaranth resistant to both ALS-inhibitors and glyphosate, planting glufosinate-tolerant cotton and using glufosinate tank-mixed with pyrithiobac and/or S-metolachlor as POST application will be an effective POST management strategy. Finally, a POST-directed lay-by application of diuron or prometryn plus MSMA made no later than first bloom cotton stage would control the late-emerged Palmer amaranth and also ensure a clean field later in the season [110,117].

8.1.3. Soybean

Before the evolution of glyphosate-resistance, Palmer amaranth populations—including those resistant to ALS-Inhibitor herbicides—were effectively managed by glyphosate in glyphosate-tolerant crops [123,133,134]. Currently, glyphosate-resistant Palmer amaranth interferes with soybean production in more than 22 states in the United States [77]. To manage Palmer amaranth resistant to ALS-inhibitors and glyphosate, it is necessary to start clean with a preplant burndown treatment using an herbicide such as 2,4-D, carfentrazone, dicamba, glufosinate, or paraquat [118,135]. To prevent early season Palmer amaranth emergence, a residual herbicide such as flumioxazin or saflufenacil can be tank-mixed with 2, 4-D or dicamba burndown treatment [114]. Soybean planting intervals of at least 21 days must be maintained after burndown application of 2, 4-D (0.56 kg ae ha⁻¹) or dicamba (0.28 kg ae ha⁻¹) to avoid significant soybean injury [136].

Herbicides labeled for PRE control of glyphosate-resistant Palmer amaranth may contain one or more of the five different site-of-action groups: ALS-inhibitors (chlorimuron, imazaquin, and imazethapyr), long-chain fatty acid inhibitors (acetochlor, alachlor, dimethenamid-P, pyroxasulfone, and S-metolachlor), microtubule inhibitors (pendimethalin and trifluralin), PPO-inhibitors (flumioxazin, fomesafen, saflufenacil, and sulfentrazone), and photosystem II (PS II)-inhibitors (linuron and metribuzin), all of which can effectively prevent glyphosate-resistant Palmer amaranth emergence [116, 117,137, 138, 107, 118, 108]. When properly activated by timely rainfall or irrigation, residual PRE herbicides can provide 2–3 weeks of Palmer amaranth control depending on soil moisture and weed pressure. However, in fields
where Palmer amaranth is resistant to both ALS-inhibitors and glyphosate, herbicides with an ALS-inhibitor site of action will not be effective in controlling it. Research conducted in North Carolina reported that S-metolachlor was more effective than pendimethalin, and that flumioxazin and fomesafen were more effective than metribuzin plus chlorimuron in controlling glyphosate-resistant Palmer amaranth [139].

Contemporary POST herbicides labeled for glyphosate-resistant Palmer amaranth control in glyphosate-resistant soybean belong to two sites of action groups: ALS-inhibitors (thifensulfuron, imazamox, imazaquin, and imazethapyr) and PPO-inhibitors (acifluorfen, fomesafen, and lactofen) [135,107]. In non-sulfonylurea-tolerant (ST) soybean varieties, a premix of chlorimuron plus thifensulfuron at 26.5 g ha\(^{-1}\) can control ≤ 10-cm-tall Palmer amaranth; however, in ST soybean varieties, higher rates (80 g ha\(^{-1}\)) can be used to control 20-cm-tall plants. In a North Carolina study, > 80% late-season control of glyphosate-resistant Palmer amaranth was achieved with pendimethalin/S-metolachlor plus flumioxazin; fomesafen/metribuzin plus chlorimuron applied PRE followed by a POST application of fomesafen [139].

Palmer amaranth populations resistant to ALS-inhibitors can be effectively managed with POST applications of glyphosate in glyphosate-tolerant production systems. Early-POST application of PPO-inhibitors is the only POST option for control of Palmer amaranth resistant to both ALS-inhibitors and glyphosate in glyphosate-tolerant soybean. However, Palmer amaranth control with PPO-inhibitors is highly variable depending on weed size and environmental conditions. PPO-inhibitors are contact herbicides and would not adequately control Palmer amaranth > 10 cm tall. It is strongly recommended to tank-mix the POST treatment with a residual herbicide such as acetochlor, dimethenamid-P, pyroxasulfone, or S-metolachlor to prevent Palmer amaranth emergence later in the season. As of 2015, no Palmer amaranth population resistant to PPO-inhibitors has been reported [77]; however, sole reliance on PPO-herbicides as a POST-only option will likely result in selection for Palmer amaranth biotypes resistant to PPO-inhibitor herbicides. Ideally, PPO-inhibitor herbicides should be used once per growing season as a PRE or POST treatment along with a residual herbicide containing a different mode of action to ensure long-term sustainability.

An alternate option for POST control of glyphosate-resistant Palmer amaranth is planting glufosinate-tolerant soybean and using glufosinate POST tank-mixed with acetochlor, pyroxasulfone, or S-metolachlor to control the Palmer amaranth that has already emerged while further preventing late-season emergence [137]. Additionally, glufosinate will not control Palmer amaranth > 10 cm in size. In a Nebraska study, excellent season-long control of common waterhemp was achieved with sulfentrazone plus metribuzin applied PRE followed by early POST application of glufosinate tank-mixed with acetochlor, pyroxasulfone, or S-metolachlor [137].

8.2. Non-chemical control

The principal non-chemical options for Palmer amaranth management involve the use of tillage and cover crops. Tillage can alter weed seedling emergence patterns by modifying seed burial depth, dormancy, predation, and mortality. Furthermore, tillage modifies the environmental factors crucial for germination, such as temperature, moisture, and oxygen [140–142].
In conservation agriculture, weed seed germination is often higher because most of the weed seeds lie on the soil surface where germination conditions are more favorable [143–146]. Therefore, small-seeded weed species such as Palmer amaranth have become highly prevalent in reduced tillage production systems [147]. As Palmer amaranth seedlings cannot emerge from depths ≥ 5 cm, tillage systems that bury seeds deeper than 5 cm can reduce Palmer amaranth densities to levels easily controlled by a PRE or POST herbicide [117,119]. A moldboard plow can bury Palmer amaranth seeds at least 10 cm deep and provides around 50% control of Palmer amaranth [148]. Another study reported ≥ 75% reduction in Palmer amaranth densities with the use of inversion tillage in cotton [119], while sweep cultivators further improved Palmer amaranth control with PRE herbicides in cotton [149]. Spring tillage that included two passes of a disk cultivator or one pass of a disk cultivator followed by one pass of a field cultivator/chisel plow reduced Palmer amaranth densities by 40% compared with no-tillage [119]. In the same study, following inversion tillage, spring tillage did not improve Palmer amaranth control.

Cover crops control weeds by reducing early-season weed density as a result of direct competition from cover crop biomass [150–156] or allelopathy [157–160]. Palmer amaranth was controlled 78–90% by various cover crops when evaluated at the four-node stage of cotton in Arkansas [161]. In the same study, a cereal rye cover crop that produced 846 g biomass m⁻² controlled Palmer amaranth by 90%. In Alabama, cereal rye cover crop reduced early-season Palmer amaranth density more than 60% compared with conventional tillage and winter fallow systems [162]. Similarly, crimson clover and cereal rye cover reduced Palmer amaranth density by > 50% in cotton [117,119], whereas Price et al. [163,164] and Saini et al. [165] reported similar reductions in Palmer amaranth and other weed densities by cover crop residues. When cover crops were combined with fall inversion tillage, Palmer amaranth density was reduced by > 85% [117,166]. Cereal rye cover following deep tillage in the fall increased control by 18% when used in conjunction with a glufosinate-based cotton herbicide program [167]. In addition, allelochemicals produced by cereal rye can inhibit Palmer amaranth germination and seedling growth [159].

Evidently, tillage systems and cover crops can significantly reduce Palmer amaranth emergence, but considering Palmer amaranth’s high seed production potential (600,000 seeds plant⁻¹), reduction in densities as high as 99% may not warrant the long-term validity of a control tactic. The ideal approach for Palmer amaranth management must embrace a zero-tolerance strategy (100% control) for year-round management of Palmer amaranth on a long-term basis. Nevertheless, the potential for reducing weed emergence should encourage the use of appropriate tillage systems and cover crops in an integrated weed management approach for early-season suppression of Palmer amaranth.

9. Multiple herbicide-resistant crop technologies

Multiple herbicide-resistant corn, cotton, and soybean cultivars have recently been developed using molecular techniques for addressing the growing need to control glyphosate-resistant...
weeds. The major developments in herbicide-resistant technologies include the Enlist™ weed control system and the Roundup Ready Plus Xtend system in corn, cotton, and soybean.

9.1. Enlist Duo™ weed management system

The Enlist Duo™ weed control system will be applicable in all Enlist™ crops (corn, cotton, and soybean) containing traits that make them tolerant to 2,4-D as well as glyphosate. Enlist™ corn will also be tolerant to the grass herbicides belonging to the aryloxyphenoxy propionate family that contain quizalofop, fluazifop, etc. In addition, Enlist™ corn, cotton, and soybean will also be tolerant to glufosinate.

The Enlist Duo™ herbicide contains glyphosate and 2,4-D choline, a low-volatile formulation of 2,4-D manufactured using Colex-D™ technology. The spectrum of weed control with the Enlist™ system will be similar to glyphosate plus 2,4-D. In corn, the new system will provide flexibility in applying this tank-mixture up to the V8 growth stage or 76 cm height. In cotton and soybean, the Enlist Duo™ system will enable POST application of 2,4-D choline to manage glyphosate-resistant broadleaf and other difficult-to-control weeds. Enlist™ soybean can receive POST applications of Enlist Duo™ herbicide up to the R2 or full-flower stage of soybean. The new seed traits (Enlist™ Corn and Soybean) and the new herbicide premix (Enlist Duo™) have recently been deregulated by the United States Department of Agriculture (USDA) and the United States Environmental Protection Agency (USEPA), respectively.

9.2. Roundup Ready® 2 Xtend weed management system

The Roundup Ready® 2 Xtend weed management system is being developed in corn, cotton, and soybean based on Roundup Ready 2 Xtend™ seed traits that make them tolerant to both glyphosate and dicamba. The Roundup® Xtend herbicide, a premix of glyphosate and dicamba, will provide an additional tool for controlling troublesome weeds, including those resistant to glyphosate. The new formulation of dicamba integrated into the Roundup® Xtend herbicide has been claimed to be significantly less volatile than existing formulations of dicamba based on VaporGrip™ Technology. The dicamba component amenable with the Roundup® Xtend weed management system will also be available separately as Xtendimax to allow growers to apply it with labeled tank-mix partners in addition to glyphosate.

The new traits will be marketed as Roundup Ready 2 Xtend soybeans and Bollgard II Xtend Flex cotton. The Roundup® Xtend herbicide may be applied up to 7 days before harvest in cotton and up to the R1 or flower-initiation stage of soybean. Previous researchers have reported excellent control of glyphosate-resistant weeds when dicamba was used alone or combined with glyphosate [168–171].

These technologies will offer growers the flexibility to control weeds, allow for the continued use and adoption of reduced tillage practices, and will help reduce the risk of selecting glyphosate-resistant weeds. The main concern about these technologies is off-target movement via particle drift or volatility that can severely damage sensitive crops such as tomato, grape, melons, and nursery plants as well as the agronomic crops that are not tolerant to 2,4-D or dicamba [172]. Although the new formulations of both 2, 4-D and dicamba are claimed to be
far less volatile than traditional chemistries, the manufacturers of these traits are also developing application technologies to minimize the drift potential.

Other herbicide-resistant technologies include MGI soybean™, which will be tolerant to mesotrione, glufosinate, and Isoxaflutole, and Balance Bean tolerant to Isoxaflutole [173]. The latest version of Balance GT™ soybeans carries traits for tolerance to either glyphosate and isoxaflutole, or the latter plus glufosinate. It is widely believed that the HPPD component will probably be applied PRE followed by glyphosate/glufosinate or other POST herbicides. Both MGI soybean and Balance Bean traits will broaden the herbicide options for soybean growers for resistant weed management; however, their commercial cultivation is pending approval by federal government agencies.

Author details

Parminder S. Chahal1, Jatinder S. Aulakh1, M. Jugulam2 and Amit J. Jhala*

*Address all correspondence to: Amit.Jhala@unl.edu

1 Department of Agronomy and Horticulture, University of Nebraska–Lincoln Lincoln, NE, USA

2 Department of Agronomy, Kansas State University, Manhattan, KS, USA

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