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Short title: Palmer amaranth in the PNW

Palmer Amaranth (*Amaranthus palmeri*) in the Pacific Northwest: Glyphosate-Resistance Confirmation and Efficacy of Selected Herbicides

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Abstract

Herbicide-resistant Palmer amaranth has been problematic within the United States for the past 30 years. The recent introduction of Palmer amaranth into the Pacific Northwest (PNW) prompted extensive surveys in 2023 and 2024 to collect seed samples for herbicide-resistance screening and leaf tissue for resistance-mechanism genotyping. Greenhouse dose-response bioassays were conducted in Kimberly, ID, during the summer of 2024 to assess the response of Palmer amaranth populations to selected postemergence herbicides. Resistance to glyphosate predominated across populations, and reduced sensitivity to 2,4-D, dicamba, and mesotrione was also observed. In contrast, glufosinate and saflufenacil provided effective control of PNW Palmer amaranth populations. Based on the dose-response bioassays, the effective dose required to provide 90% control (ED_{90}) of the suspected glyphosate-resistant populations was 20 to 63-fold compared to the susceptible population. Subsequent 5-enolpyruvylshikimate-3-phosphate synthase (*EPSPS*) gene duplication analysis was conducted to confirm glyphosate resistance in the Palmer amaranth populations. About 74% (17 of 23) of the Palmer amaranth tissue samples showed gene duplication, with up to 150 copies of the *EPSPS* gene. The *EPSPS* gene amplification analysis of plants that survived 2X rate of glyphosate (2,520 g ae ha^{-1}) showed up to 150 *EPSPS* genes in glyphosate-resistant populations. The widespread glyphosate resistance in the collected samples suggests that Palmer amaranth populations are being introduced into the PNW from locations where resistance to herbicide sites of action has previously evolved.

Nomenclature: Dicamba; glyphosate; glufosinate; mesotrione; saflufenacil; 2,4-D; Palmer amaranth, *Amaranthus palmeri* S. Watson; alfalfa, *Medicago sativa* L.; barley, *Hordeum vulgare* L.; corn, *Zea mays* L.; dry bean, *Phaseolus vulgaris* L.; potato, *Solanum tuberosum* L.; sugar beet, *Beta vulgaris* L.; wheat, *Triticum aestivum* L.

Keywords: 2,4-D, dicamba, glufosinate, glyphosate-resistance, mesotrione, pigweed

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Introduction

Palmer amaranth is one of the most troublesome and economically damaging agronomic weeds in crop production systems across the United States (US). The rapid growth rate, high fecundity, and propensity to evolve resistance to multiple herbicide sites of action (SOA) make it one of the most economically important agricultural pests (Tehranchian et al. 2017). Originally native to the southwestern United States, Palmer amaranth has spread to other regions of North America and has reached other continents, such as South America and Europe (Küpper et al. 2017; Torra et al. 2020).

The widespread adoption of glyphosate-resistant crops and the concomitant overreliance on glyphosate as the primary weed-control practice have led to the evolution of glyphosate-resistant Palmer amaranth populations across different states (Heap 2025). Since the confirmation of the first case of glyphosate resistance (GR) in Palmer amaranth in 2006 (Culpepper et al. 2006), the number of glyphosate-resistance cases across the US has increased geometrically (Araujo et al. 2024; Heap 2025). Glyphosate resistance in Palmer amaranth is primarily conferred by increased *EPSPS* gene copy number, although other mechanisms may also contribute (Gaines et al. 2010; 2011; Küpper et al. 2017; Ward et al. 2013). To control glyphosate-resistant Palmer amaranth and diversify herbicide options, growers have turned to alternatives such as dicamba, 2,4-D, glufosinate, and mesotrione. However, the repeated use of these herbicides has also raised concerns about the potential for widespread resistance of Palmer amaranth to them (Kumar et al. 2019; Shyam et al. 2022; Tehranchian et al. 2017).

Herbicide resistance surveys have revealed alarming trends in Palmer amaranth populations. A state-level survey in Texas found that 62% of Palmer amaranth populations in the High Plains region were resistant to glyphosate, while resistance to atrazine and ALS inhibitors was also common (Garetson et al. 2019). In Kansas, a population with multiple resistance to 2,4-D, glyphosate, chlorsulfuron, atrazine, and mesotrione was identified, highlighting the ability of Palmer amaranth to evolve resistance to multiple herbicide sites of action (Kumar et al. 2019). A baseline survey in Colorado and Nebraska indicated up to a 20% incidence of dicamba resistance in Palmer amaranth populations collected from Colorado (Araujo et al. 2024). Additionally, the first cases of glufosinate resistance in Palmer amaranth were reported in Arkansas, with gene amplification and increased expression of chloroplastic glutamine synthetase identified as the mechanisms of resistance (Carvalho-Moore et al. 2022).

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The rapid spread of Palmer amaranth across the US and the rapid evolution of herbicide resistance in this weed underscore the need for regular surveys and resistance screenings to monitor its spread. These efforts can help identify new infestations and emerging resistance issues, guiding the development of effective management strategies. The first case of Palmer amaranth in the Pacific Northwest (PNW) in 2022 (Adjesiwor and Felix, personal observation). This Palmer amaranth population was introduced into Idaho through contaminated bird feed. Since then, a coordinated extension and outreach effort among land-grant universities (University of Idaho and Oregon State University), Amalgamated Sugar Company, other commodity commissions, and industry was launched to track Palmer amaranth in the PNW, with the following objectives: 1) map the presence and spread of Palmer amaranth in the PNW, 2) confirm glyphosate resistance in PNW Palmer amaranth populations through dose-response bioassays and measurement of 5-enolpyruvylshikimate-3-phosphate synthase (*EPSPS*) gene copy number, and 3) evaluate the response of PNW Palmer amaranth populations to selected herbicides.

Materials and Methods

Palmer amaranth surveys and sampling

In the summer of 2023, agronomists and crop advisors were trained for Palmer amaranth identification, and during the months of July and August, they visited GPS coordinates of suspected Palmer amaranth locations throughout Idaho and Oregon to confirm whether the suspected pigweeds were Palmer amaranth (Figure 1). If present, mature seeds were collected for future dose-response assays. In addition to visual identification, tissue samples were collected from suspected Palmer amaranth plants and sent to Colorado State University for Kompetitive Allele Specific PCR (KASP) genotyping test to confirm if the plants were Palmer amaranth (Brusa et al. 2021). The KASP test confirmed that the suspected pigweeds were Palmer amaranth and waterhemp. Two populations appeared to be hybrids. This study is focused only on Palmer amaranth populations.

Dose response bioassay

Seeds collected from Palmer amaranth populations in Idaho and Oregon in 2023 were cleaned and stored at 4 C until the experiments were initiated. Greenhouse studies were conducted in 2024 at the University of Idaho Research and Extension Center in Kimberly ID to evaluate the efficacy of selected herbicides on Palmer amaranth. The experiment was laid out as

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a 9 by 4 factorial completely randomized design with four replications. Factor A was nine different doses of glyphosate (0×, 0.125×, 0.25×, 0.5×, 1×, 2×, 4×, 8×, and 16×; where 1× is the field-use rate of glyphosate (1260 g ae ha^{-1}). Factor B was four populations of Palmer amaranth, which were comprised of three suspected glyphosate-resistant populations from Idaho and Oregon (ID3, ID7, OR1) and one glyphosate-susceptible population from Scottsbluff, Nebraska (Sus) (Table 1). The ID3 and ID7 populations were selected because they had the most seeds and were representative of where most of the Palmer amaranth were detected.

On July 8, 2024 (experiment 1) and August 23, 2024 (experiment 2), approximately 5 Palmer amaranth seeds were planted in 0.9-L individual pots containing potting soil mix made up of 75% sphagnum peat moss, perlite, dolomitic limestone, gypsum, and a wetting agent (Sunshine Mix, LC1, Sun Gro Horticulture Inc., Bellevue, WA). The average nutrient composition of the soil potting mix was 52% organic matter, 50 mg kg^{-1} N, 97 mg kg^{-1} P, and 255 mg kg^{-1} K. No additional fertilizer was applied. The experiment was conducted under 14/10 h day/night photoperiod with 25±2 C day and 22±2 C night temperatures. Plants were uniformly irrigated twice daily for two minutes using an automated overhead sprinkler system. A supplemental light (Sun System; Sunlight Supply Inc., Vancouver, WA, United States) was used to maintain a constant photoperiod (14 hours light and 10 hours dark) throughout the experiments. Plants were thinned to one seedling per pot shortly after emergence. Plants were sprayed with glyphosate when plants were approximately 7.6 cm tall using a Generation 4 Research Track Sprayer (DeVries Manufacturing Corp., Hollandale, MN, USA) fitted with a single 8002E nozzle (TeeJet, Spraying Systems Co., Wheaton, IL, USA) calibrated to deliver 188 L ha^{-1} of carrier volume at 207 kPa.

Visible Palmer amaranth control was evaluated 21 days after spraying on a scale of 0 to 100%, with 0% being no weed control or 100% being complete weed control or no aboveground green growth. At 21 days after herbicide application, aboveground biomass was clipped and oven-dried to a constant weight at 60 C for about 72 hours to measure dry biomass. Aboveground biomass was reported in terms of percent reduction compared to the nontreated check (0 g ae ha^{-1} glyphosate).

EPSPS gene amplification

The *EPSPS* gene copy number was evaluated in eight suspected glyphosate-resistant Palmer amaranth populations from Idaho and Oregon (ID1, ID2, ID3, ID4, ID5, ID6, ID7, ID8,

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and OR1) and the Sus population (Table 1). The experiments were laid out as a completely randomized design with six replications. On July 8, 2024 (experiment 1) and August 23, 2024 (experiment 2), approximately 3 Palmer amaranth seeds were planted in 0.9-liter individual pots as described in the dose-response experiments. Plants were sprayed with 2X rate of glyphosate (2,520 g ae ha^{-1}) when plants were approximately 7.6 cm tall using the same methods described in the dose-response experiments. Surviving plants were harvested 21 days after herbicide application, and tissue samples were frozen in liquid nitrogen and stored at –80 C until genomic DNA extraction. Genomic DNA was extracted from leaf tissue using the manufacturer's protocol for the MagMAX Plant DNA Isolation Kit (Thermo Fisher Scientific, Waltham, MA), which describes plant tissue homogenization and DNA purification steps. Plant tissue homogenization steps were performed using 2.0 mL tubes containing two 5 mm stainless steel beads and an Omni Bead Ruptor 96 (Omni International, Kennesaw, Georgia) set to 15 Hz for 2.00 minutes. Subsequent DNA purification steps were performed with a KingFisher Apex (Thermo Fisher Scientific, Waltham, MA,) equipped with a 96 deep-well head. Quantification of DNA was performed with a BioTek Synergy HTX multimode microplate reader (BioTek Instruments, Winooski, VT) and BioTek Gen5 software (version 3.08.01).

Quantitative real-time PCR (qPCR) was utilized to determine *EPSPS* gene copy number with *A36* serving as the reference gene. Primers for *EPSPS* and *A36* were designed by Gaines et al. (2010) and Singh et al. (2018), respectively. Names of the primers and their respective sequences are listed as the following: A36_F244 (5′TTGGAACTGTCAGAGCAACC3′) and A36_R363 (5′GAACCCACTT CCACCAAAAC3′) amplified *A36* and EPSF1 (5′ATGTTGGACGCT CTCAGAACTCTTGGT3′) and EPSR8 (5′TGAATTTCCTCCAGCAACGGCAA3′) amplified *EPSPS*.

All qPCR reactions were performed on a QuantStudio 7 Flex (Applied Biosystems, Foster City, CA) using the PowerUp SYBR Green Master Mix manufacturer's protocol (Applied Biosystems, Foster City, CA). Each 20 µL qPCR reaction consisted of 10 µL of PowerUp SYBR Green Master Mix, 2 µL of forward and reverse primer (5 µM), 4 µL of nuclease-free water, and 10 ng of gDNA. The thermoprofile consisted of the following: 2 min at 50 C for UDG activation, 2 min at 95 C for activation of the Dual-Lock DNA polymerase, and 40 cycles of 15 s at 95 C and 1 min at 60 C. A melt curve analysis was performed by subjecting samples to the following thermoprofile: 95 C for 15 s at a ramp rate of 1.6 C/s, 60 C for 1 min at a ramp rate of 1.6 C/s,

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and 95 C for 15 s at a ramp rate of 0.15 C/s. All populations had four biological replicates except for ID5, which had three biological replicates because of a damaged sample. All samples were performed in triplicate. Non-template controls were included for each set of genes, and amplification was not observed in these reactions. Increase in genomic copy number of *EPSPS* relative to *A36* was expressed as $2^{\Delta Ct}$ where ΔCt = (Ct *A36* – Ct *EPSPS*) (Gaines et al. 2011; Singh et al. 2018b).

Efficacy of selected postemergence herbicides

The efficacy of 2,4-D, dicamba, mesotrione, glufosinate-ammonium, and saflufenacil was evaluated on all populations listed in Table 1. The postemergence herbicide efficacy experiments were laid out as a 9 by 6 factorial completely randomized design with 4 replications. Factor A was eight suspected glyphosate-resistant Palmer amaranth populations from Idaho and Oregon (ID1, ID2, ID3, ID4, ID5, ID7, ID8, and OR1) and the Sus population. Factor B was field use rates of 2,4-D, dicamba, mesotrione, glufosinate-ammonium, and saflufenacil (Table 2), in addition to a nontreated check.

On July 8, 2024 (experiment 1) and August 23, 2024 (experiment 2), approximately 3 Palmer amaranth seeds were planted in 0.9-L individual pots as described in the dose-response experiments and thinned to one seed per pot after emergence. In these experiments, the postemergence herbicides (Table 2) were applied to 7.6 cm tall Palmer amaranth. Each herbicide was applied with the label-recommended adjuvant (Table 2). Visible Palmer amaranth control was evaluated 21 days after spraying on a scale of 0 to 100%, with 0% being no weed control or 100% being complete weed control or no aboveground green growth. No biomass was collected from these experiments in order to allow surviving plants to produce seeds for future experiments.

Data analysis

Nonlinear regression analysis was used to quantify the relationship between glyphosate rate and Palmer amaranth visible control and biomass reduction. For both visible control and biomass reduction, a three-parameter log-logistic model was used (Eq. [[1]]Equation 1), where Y is response variable (visible control or biomass reduction in %), d is the upper limit asymptote, X is the rate of the herbicide applied in grams acid equivalent per hectare (g ae ha^{-1}), e is the value of X at the inflection point of the curve, and b is the slope of the curve at e (Seefeldt et al. 1995).

$$Y = d / (1 + (X / e)b) \quad ([Eq. [1]]$$

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The model parameters in Eq. [1] have practical interpretations: d is an estimate of visible Palmer amaranth control or biomass reduction when no herbicide is applied (similar to the y-intercept in linear regression), and e is an estimate of the herbicide rate required to cause a 50% visible control or biomass reduction (GR50). Nonlinear regression analysis was conducted in the R statistical language using the drc package (R Core Team 2024; Ritz and Streibig 2005). The level of resistance (R/S) was calculated by dividing the GR50 value (amount of glyphosate needed for 50% visible control) of the resistant biotype (ID3, ID7, OR1) by that of the susceptible biotype (Sus).

Data from the selected postemergence herbicide efficacy and the EPSPS gene amplification experiments were analyzed using a linear mixed-effects ANOVA in R statistical language version 4.3.2 using the *lme4* and *lmerTest* packages (Kuznetsova et al. 2017). The Palmer amaranth population was considered a fixed effect, and the experimental run was a random effect. Estimated marginal means were separated with pairwise comparisons using *multcomp* and *emmeans* packages when the analysis of variance indicated significant treatment effects (alpha ≤ 0.05) (Hothorn et al. 2008; Lenth 2017), which were separated at $\alpha = 0.05$ using Fisher's protected LSD.

Results and Discussion

Palmer amaranth detections in the Pacific Northwest

As of September 2025, 154 Palmer amaranth detections had been reported in southern Idaho, and more than 70% of sampled populations were resistant to glyphosate (Figure 1). These detections affected more than 3,642 ha of cropland in several counties in the PNW and in multiple crops (alfalfa, barley, corn, dry bean, potato, sugar beet, and wheat), as well as right-of-way and private property. While only 3,642 ha are currently affected, previous research indicates that Palmer amaranth infestations can spread rapidly. For example, Norsworthy et al. (2014) found that just 20,000 Palmer amaranth seeds introduced into a 1 m^2 area spread to all corners of fields ranging in size from 0.53 to 077 ha within 3 years after introduction. Thus, the 3,642-ha infestation could increase in thousand folds in the coming years, if left unchecked. There were six detections of Palmer amaranth in Oregon, with the majority being in Malheur County (Figure 2). Although Palmer amaranth was detected in Spokane and Walla Walla counties of Washington in 2023,

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there were no additional detections in the 2024 surveys. In both Idaho and Oregon, it was suspected that Palmer amaranth was introduced into the states through contaminated livestock feed, farm machinery, and contaminated bird feed. This is consistent with previous findings on Palmer amaranth introduction into different states where contaminated seeds sources and farm machinery were identified as the major sources of introduction (Briscoe Runquist et al. 2019; Ward et al. 2013).

Dose response bioassay and EPSPS gene amplification

A representative sample of 23 tissue samples collected in 2023 and analyzed for *EPSPS* gene amplification showed that 74% (17 out of 23) of sampled populations were resistant to glyphosate (Figure 1). The dose-response bioassay confirmed glyphosate-resistance in Palmer amaranth collected from the PNW (Figures 3 & 4; Table 3). Compared to the susceptible biotype, it would require 20 to 63 times more glyphosate to provide 50% control of the glyphosate-resistant Palmer amaranth biotypes (Table 1). The *EPSPS* gene amplification analysis of plants that survived 2X rate of glyphosate (2,520 g ae ha^{-1}) showed up to 150 *EPSPS* genes in glyphosate-resistant populations (Figure 5). There were statistically significant differences in average *EPSPS* gene copy numbers among the Palmer amaranth populations. All populations from the PNW had more *EPSPS* genes than Sus (Figure 5). Previous studies have identified *EPSPS* gene amplification as the primary mechanism of glyphosate resistance (Araujo et al. 2024; Molin et al. 2018; Patterson et al. 2018), which can also exist as a rearranged extrachromosomal circular DNA that confers both glyphosate and glufosinate resistance in Palmer amaranth (Carvalho-Moore et al. 2025). The susceptible population used in this study has an *EPSPS* gene copy number of 1, which is consistent with previous findings (Gaines et al. 2011; Molin et al. 2020). The resistant populations had 15 or more *EPSPS* gene copies. In a study of the relationship between glyphosate resistance and *EPSPS* gene copies, a threshold of ~15 EPSPS gene copies was identified as the average number of genes conferring glyphosate resistance in Palmer amaranth (Yakimowski et al. 2021).

Efficacy of selected postemergence herbicides

Some Palmer amaranth populations from the PNW showed reduced sensitivity to the field use rates of 2,4-D, dicamba, and mesotrione, compared to the susceptible population (Figure 6). While this is not indicative of Palmer amaranth resistance to these herbicides in the PNW,

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reduced sensitivity to these herbicides would greatly affect the ability of farmers to effectively control Palmer amaranth and limit spread in the PNW region. In the US, Palmer amaranth resistance to 2,4-D has already been documented in Kansas (Kumar et al. 2019; Shyam et al. 2022). Similarly, reduced sensitivity and resistance of Palmer amaranth to dicamba has been reported in at least two states in the US (Araujo et al. 2024; Foster and Steckel 2022). Weed resistance to auxinic herbicides like 2,4-D is reported to be conferred by either target site mutation or non-target site mechanism, including reduced absorption, enhanced metabolism or reduced translocation (Dang et al. 2018; Figueiredo et al. 2028; Kohler et al. 2004; LeClere et al. 2018). Resistance to 2,4-D in a Palmer amaranth population was confirmed to be mediated by enhanced metabolism (Shyam et al. 2022). Continued monitoring and molecular characterization are necessary to determine whether the reduced sensitivity to 2,4-D and dicamba in the PNW populations is due to target-site mutations or non-target-site mechanisms. Mesotrione resistance or reduced sensitivity has also been documented in multiple US states (Hamberg et al. 2024; Kumar et al. 2020; Nakka et al. 2017; Singh et al. 2018a).

Although there were statistical differences between the PNW populations and Sus, all PNW Palmer amaranth populations were effectively controlled with field-applied rates of glufosinate-ammonium and saflufenacil (Figure 6). This means glufosinate-ammonium and saflufenacil would provide additional options for managing Palmer amaranth in PNW cropping systems. However, Palmer amaranth has already evolved resistance to protoporphyrinogen oxidase-inhibiting and glutamine synthetase-inhibiting herbicides in other states, indicating that the utility of the herbicides in the PNW may not last long (Heap 2025; Priess et al. 2022; Salas et al. 2016). Even with thoughtful herbicide rotations, there is a high potential for any Palmer amaranth introduced to the PNW to be resistant to one or more commonly applied herbicides, highlighting the importance of incorporating non-chemical weed management practices in the future to prevent the movement of Palmer amaranth into other counties.

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Practical Implications

Palmer has been problematic weed for farmers within the United States for decades and the recent introduction of Palmer amaranth into the PNW will substantially change weed management practices in PNW cropping systems. The results of this study provide baseline data to track the expansion of Palmer amaranth in the PNW over the next several years. The PNW states must coordinate efforts to reduce new introductions of Palmer amaranth and limit state-to-state dispersal of this weed. Since a number of detections were along rights-of-way, departments of transportation in the PNW states need to intensify weed management along roadways to limit the spread of Palmer amaranth. The widespread glyphosate resistance in PNW Palmer amaranth populations suggest that farmers relying exclusively on glyphosate for weed control in glyphosate-resistant crops such as sugar beet would need to incorporate effective residual herbicides into their herbicide programs. Although this study did not confirm resistance to any additional herbicides aside from glyphosate, it is imperative for growers to diversify their weed control programs to include non-chemical options (e.g., crop rotation, electrical weeding to control weed escapes, etc.) to reduce the selection for resistance to other herbicide sites of action).

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Table 1. Description of Palmer amaranth populations collected from Idaho and Oregon in 2023 as well as a susceptible population from Nebraska, to characterize glyphosate resistance and postemergence herbicide sensitivity.

Population	Description
Sus	Susceptible check from Scotts Bluff County, Nebraska
ID1	Small grains (barley and wheat), Owyhee County, Idaho
ID2	Potatoes, Owyhee County, Idaho
ID3	Sugar beet, Minidoka County, Idaho
ID4	Sugar beet, Minidoka County, Idaho
ID5	Small grains, Owyhee County, Idaho
ID6	Sugar beet, Elmore County, Idaho
ID7	Right of way in Canyon County, Idaho
OR1	Right of way in Malheur County, Oregon

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Table 2. Postemergence herbicides used in greenhouse herbicide efficacy experiments in 2024 at Kimberly Idaho USA, to characterize the Palmer amaranth populations.

Herbicide	Rate (g ai or ae ha^{-1})	Commercial product	Manufacturer	Adjuvant[a]
2,4-D	560	Low Vol 4 Ester	Loveland Products, Inc.	AMS (2.5% v/v+ NIS (0.25 %v/v)
dicamba	280	DiFlexx®	Bayer CropScience	COC (1 %v/v)
mesotrione	105	Callisto®	Syngenta Crop Protection, LLC	AMS (2.5% v/v+ NIS (0.25 %v/v)
glufosinate-ammonium	594	Liberty 280 SL®	BASF Ag Products	AMS (2.5% v/v)
saflufenacil	50	Sharpen®	BASF Ag Products	AMS (2.5% v/v) + MSO (1%v/v)

[a]Abbreviations: AMS, ammonium sulfate (Ultra Pro®, Loveland Products, Inc.); NIS, non-ionic surfactant (Preference®, WinField Solutions); COC, crop oil concentrate (Maximizer®, Loveland Products, Inc.); MSO, methylated seed oil (MSO® Concentrate with Leci-Tech®, Loveland Products, Inc.).

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Table 3. Parameter estimates for Palmer amaranth visible control and biomass reduction following a three-parameter log-logistic model for greenhouse experiments in 2024, Kimberly ID, USA, to characterize glyphosate resistance in the Palmer amaranth populations.

Parameter estimate[a](±se)					
Population	*d*	*b*	GR_{50}	GR_{90}	*R/S*
	----------visible control ----------				
Sus	99 (4.1)	-1.3 (0.7)	81 (41)	462	-
ID3	92 (7.6)	-2.3 (0.7)	3679 (530)	9465	20
ID7	81 (14.6)	-1.2 (0.4)	3365 (1403)	20779	45
OR1	100 (18.6)	-0.6 (0.2)	747 (516)	29218	63
	----------biomass reduction ----------				
Sus	97 (56)	-0.7 (0.4)	78 (57)	1624 (2360)	
ID3	74 (10)	-2.7 (1.4)	3923 (892)	8770 (4822)	
ID7	35 (133)	-0.9 (8.6)	161580 (83090)	1654400 (98758)	
OR1	91 (29)	-0.6 (0.3)	1227 (1542)	44993 (13519)	

[a]$y=d/(1+(x/e)^b)$, parameter estimates are described in equation 1.

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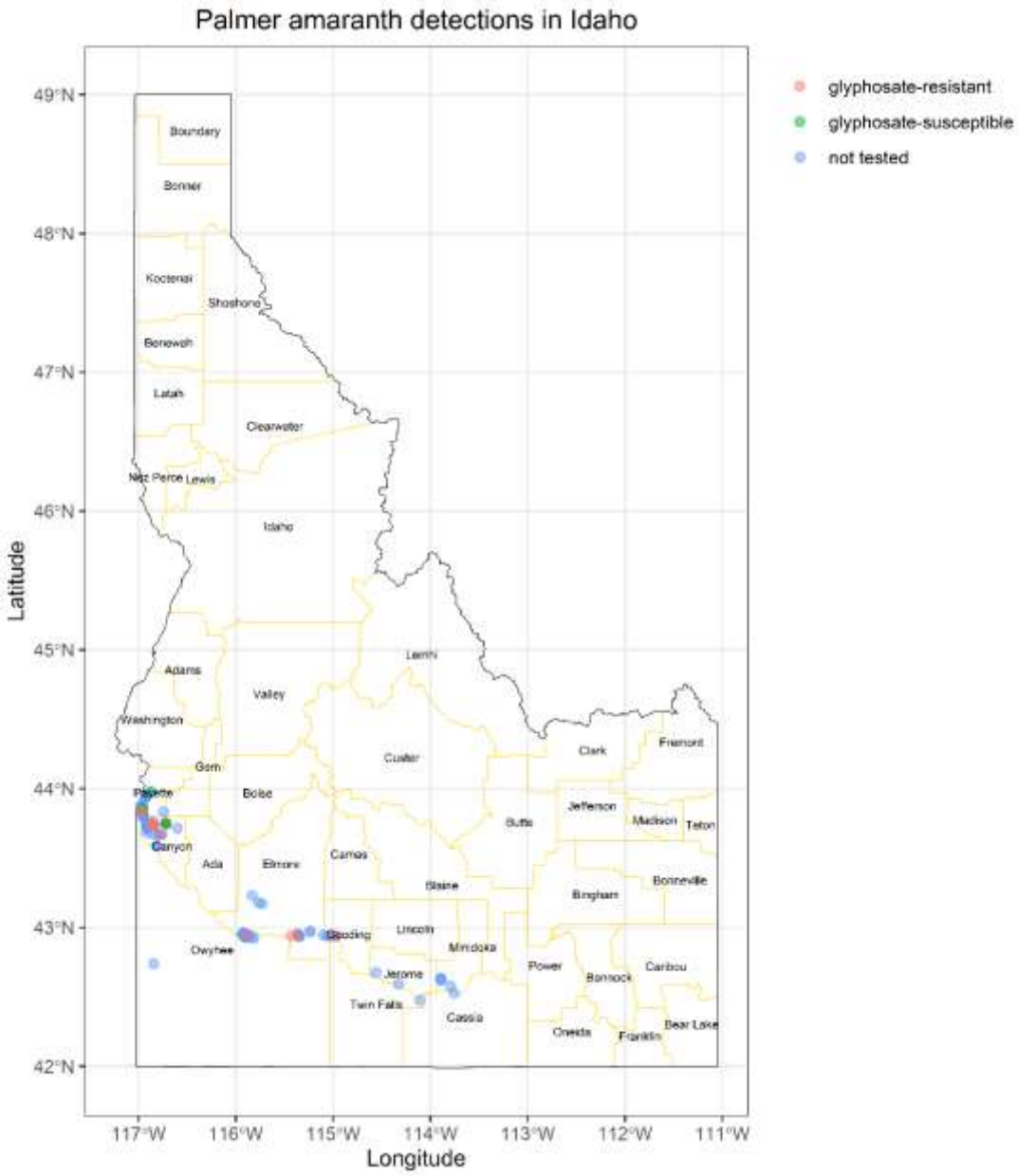

Figure 1. Distribution of Palmer amaranth in Idaho as of September 2025. There were 154 detections, affecting more than 3,642 ha of cropland, and more than 70% of sampled populations were resistant to glyphosate.

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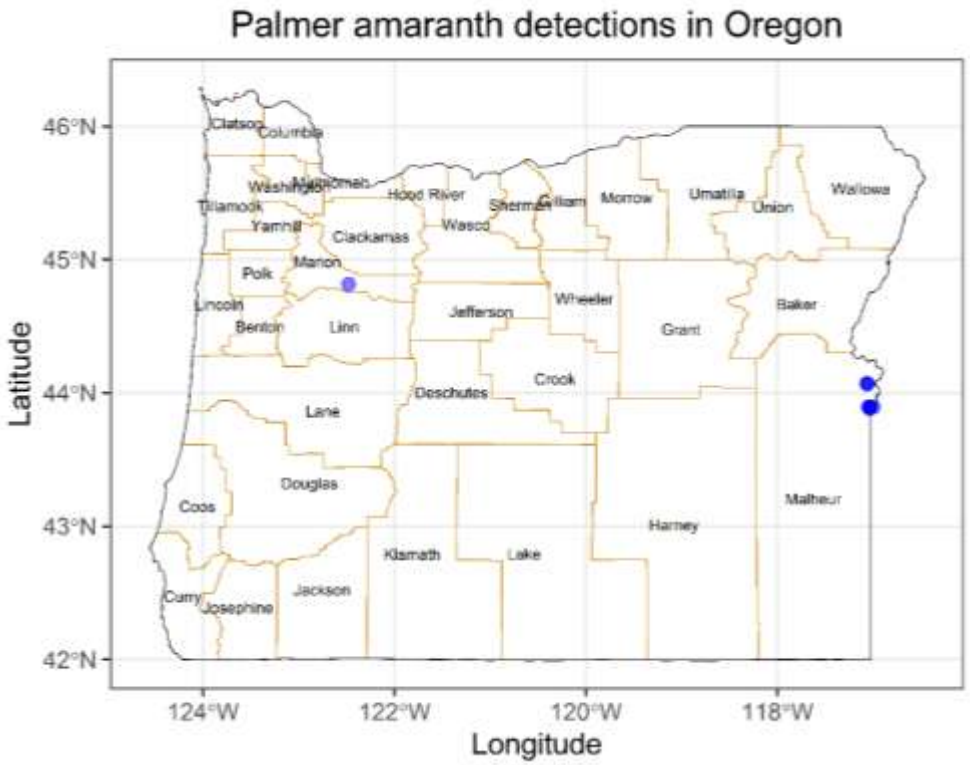

Figure 2. Counties in Oregon where Palmer amaranth was detected. Palmer amaranth presence was confirmed in Malheur County, OR as of September 2025.

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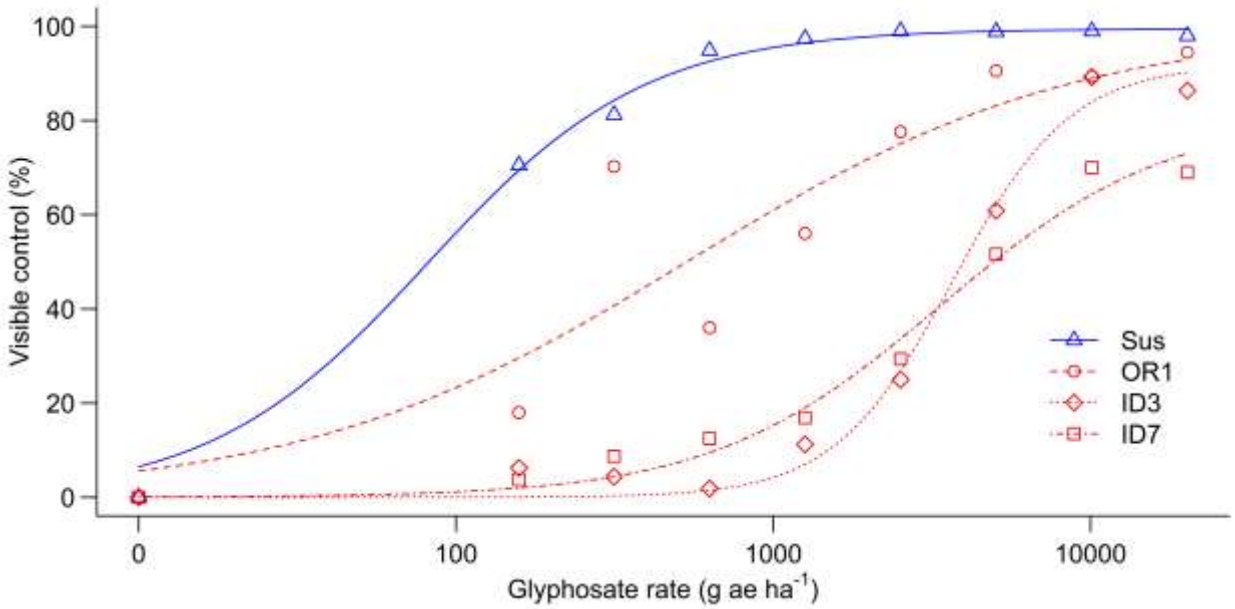

Figure 3. Dose-response curves of visible Palmer amaranth control for three suspected glyphosate-resistant Palmer amaranth populations from Idaho and Oregon (ID3, ID7, and OR1) and a susceptible population from Nebraska (Sus).

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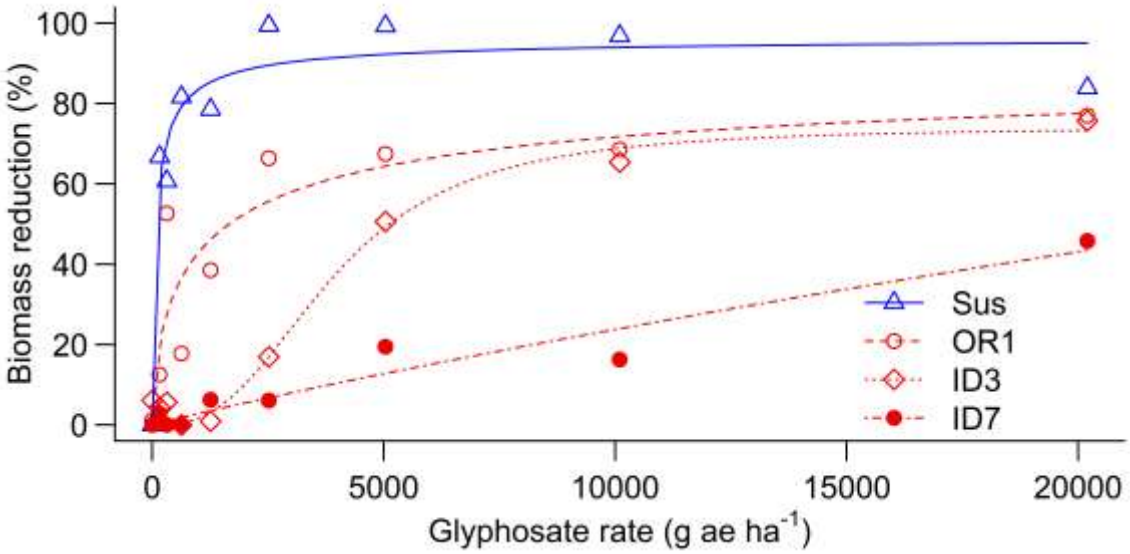

Figure 4. Dose-response curves of Palmer amaranth biomass reduction for three suspected glyphosate-resistant Palmer amaranth populations from Idaho and Oregon (ID3, ID7, and OR1) and a susceptible population from Nebraska (Sus).

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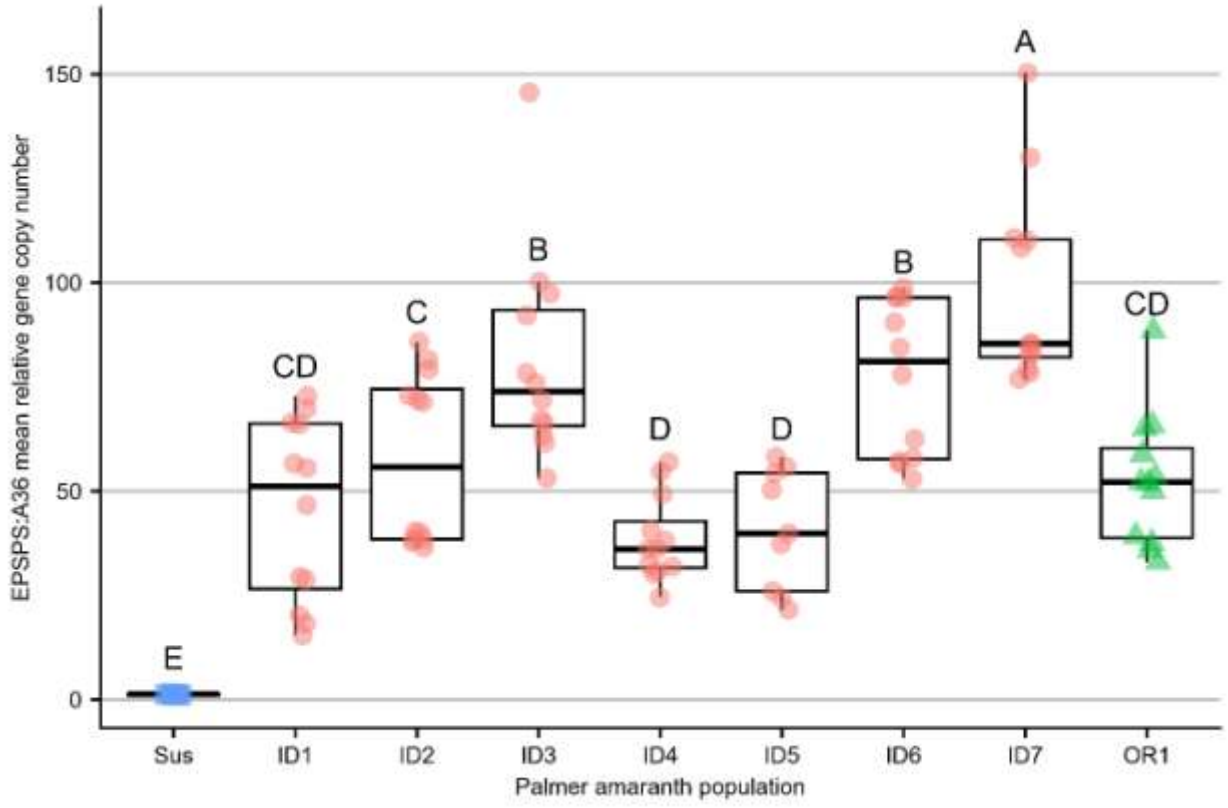

Figure 5. *EPSPS* gene copy numbers of eight suspected glyphosate-resistant Palmer amaranth populations from Idaho and Oregon (ID1, ID2, ID3, ID4, ID5, ID6, ID7, and OR1) and a susceptible population from Nebraska (Sus). The ID1, ID2, ID3, ID4, ID5, ID6, ID7, and OR1 populations all survived 2X rate of glyphosate (2,520 g ae ha^{-1}).

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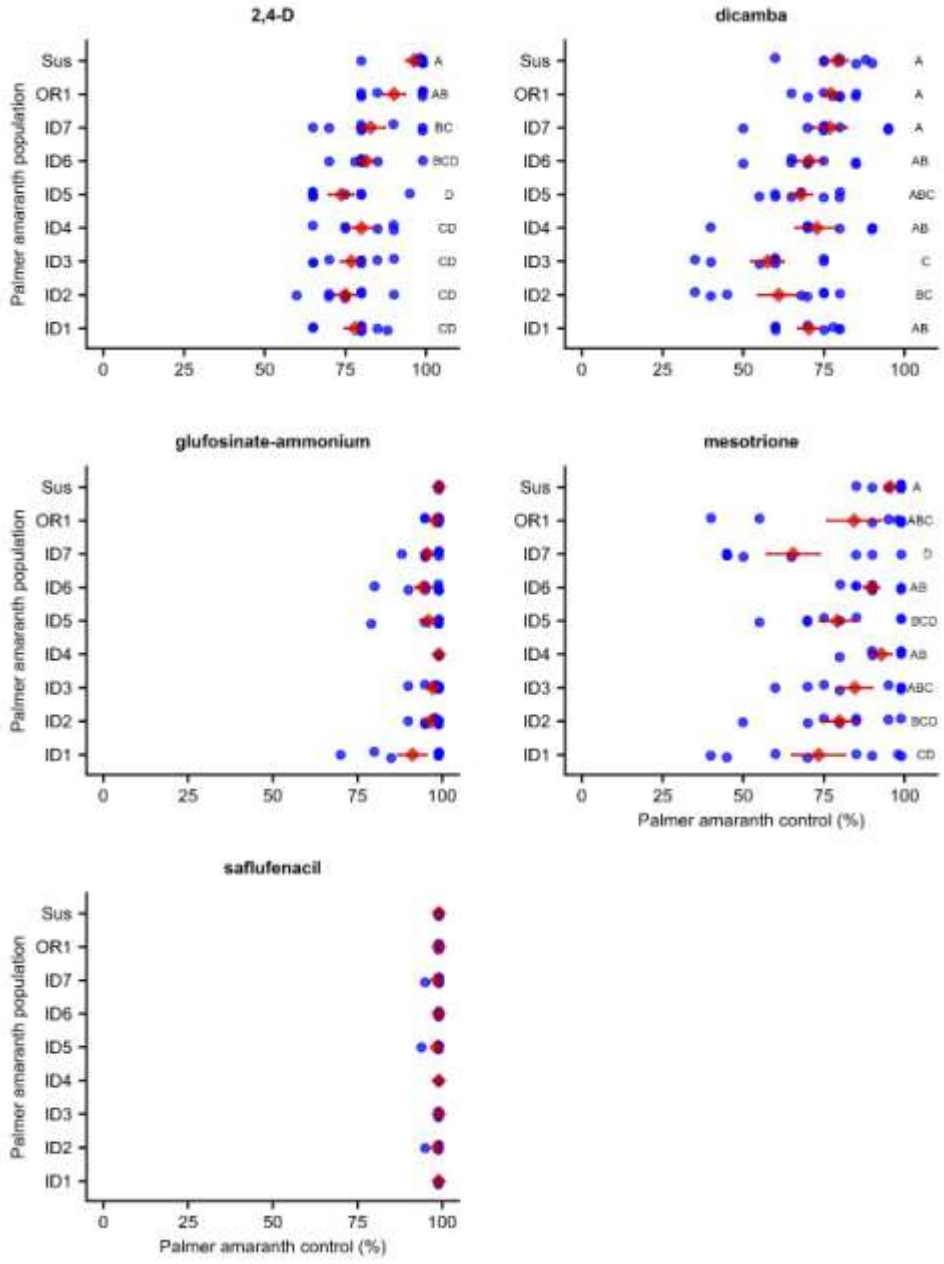

Figure 6. The efficacy of 2,4-D, dicamba, glufosinate-ammonium, mesotrione, and saflufenacil on eight suspected glyphosate-resistant Palmer amaranth populations from Idaho and Oregon (ID1, ID2, ID3, ID4, ID5, ID7, ID8, and OR1) and a susceptible population from Nebraska (Sus) as described in Table 1. Blue solid circles represent each replicate and red solid diamonds represent the means. Red error bars represent the standard error of the mean. Means followed by the same letters are not statistically different at 0.05 probability level according to Fisher's protected LSD.

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